Space-occupying cerebellar infarction: complications, treatment, and outcome

HERMANN NEUGEBAUER, M.D., M.Sc.,¹ JENS WITSCH, M.D.,² KLAUS ZWECKBERGER, M.D.,³
AND ERIC JÜTTLER, M.D., M.Sc.¹

¹Department of Neurology, Rehabilitation and University Hospital Ulm; ²Department of Neurology, Charité University Medicine Berlin, Campus Virchow Klinikum, Berlin; and ³Department of Neurosurgery, Ruprecht-Karl-University Heidelberg, Germany

Space-occupying brain edema is a frequent and one of the most dreaded complications in ischemic cerebellar stroke. Because the tight posterior fossa provides little compensating space, any space-occupying lesion can lead to life-threatening complications through brainstem compression or compression of the fourth ventricle and subsequent hydrocephalus, both of which may portend transtentorial/transforaminal herniation. Patients with large cerebellar infarcts should be treated and monitored very early on in an experienced stroke unit or (neuro)intensive care unit. The general treatment of ischemic cerebellar infarction does not differ from that of supratentorial ischemic strokes. Treatment strategies for space-occupying edema include pharmacological antiedema and intracranial pressure-lowering therapies, ventricular drainage by means of an extraventricular drain, and suboccipital decompressive surgery, with or without resection of necrotic tissue. Timely escalation of treatment is crucial and should be guided by clinical and neuroradiological rationales. Patients in a coma after hydrocephalus and/or local brainstem compression may also benefit from more aggressive surgical treatment, as long as the conditions are reversible. Contrary to the general belief that outcome in survivors of space-occupying cerebellar stroke is usually good, recent studies suggest that for many of these patients, the long-term outcome is not good. In particular, advanced age and additional brainstem infarction seem to be predictors for poor outcome. Further trials are necessary to investigate these findings systematically and provide better selection criteria to help guide decisions about surgical therapies, which should always be carried out in close cooperation among neurointensive care physicians, neurologists, and neurosurgeons. (<http://thejns.org/doi/abs/10.3171/2013.2.FOCUS12363>)

KEY WORDS • cerebellum • decompressive surgery • outcome •
space-occupying infarction • treatment

THE first case reports involving patients with space-occupying cerebellar infarcts date back to the end of the 19th century.⁸⁰ The first case reports of surgical treatment by craniectomy were published independently but almost simultaneously in 1956 by Fairburn and Olivier²⁶ and Lindgren.⁷³ As of the writing of this study, 115 original papers on space-occupying cerebellar stroke including a total of over 750 patients have been published (see Table 3).

The exact number of patients who develop space-occupying edema after cerebellar infarction is not known. Data from the literature show heterogeneous results,

ranging from 17% to 54%.^{15,19,41,47,51,58,75,84,94,104,112} The proportion of patients in which infarct swelling is potentially life-threatening also remains uncertain. In one study only 50% of patients who developed radiographic evidence of mass effect showed subsequent clinical deterioration.⁶³

The diagnosis and management of cerebellar stroke with mass effect is still a controversial issue. Because of nonspecific symptoms^{47,51,58,75,84,112} and insufficient diagnostic sensitivity of brain CT (the most widely available and commonly used imaging technique in the evaluation of patients with suspected stroke)⁷⁷ large cerebellar infarctions prone to space-occupying edema formation are often not recognized until complications due to mass effect become evident with clinical deterioration. The most feared complications in the posterior fossa are brainstem compression and occlusive hydrocephalus. In the case of extensive mass effect, conservative treatment strategies are not well validated and are often unsuccessful in clinical practice; surgical treatment is widely accepted

Abbreviations used in this paper: BAEP = brainstem auditory evoked potentials; BI = Barthel Index; EVD = external ventricular drainage; GASCIS = the German-Austrian Space-Occupying Cerebellar Infarction Study; GOS = Glasgow Outcome Scale; ICP = intracranial pressure; mRS = modified Rankin Scale; SDC = suboccipital decompressive craniectomy; SEP = somatosensory evoked potentials.

as the treatment of choice. However, the best time point for starting and the factors triggering surgical intervention remain unclear. Furthermore, the different surgical procedures have not been sufficiently tested in larger, multicenter, prospective, or randomized controlled trials. Therefore, management strategies are often based solely on personal experience or local standards and differ between neurological/neurosurgical centers. Although numerous reports suggest that surgical intervention reduces mortality in malignant cerebellar infarction, little is known about the long-term outcome in survivors.

The current work provides an overview of the available data on complications, treatment, and outcome of space-occupying cerebellar infarction.

Methods

References for this review were identified by searches of PubMed, Medline and local search machines in the library of the Charité-Berlin up to October 2012 using the terms “cerebellar,” “stroke,” “ischaemia,” “infarction,” “malignant,” “space-occupying,” “swelling,” “extraventricular,” “drainage,” “decompressive,” “surgery,” “surgical,” “decompression,” “craniectomy,” and “suboccipital” in various combinations. Articles were also identified through searches of the authors’ own files. Papers published in English or German and papers providing an abstract in either language were reviewed. All articles reporting outcome data on space-occupying cerebellar infarction were considered for the analysis. Primary data of studies that enrolled a large number of patients were given priority over case reports and reviews with respect to treatment recommendations.

Complications

Large infarcts of the cerebellar hemispheres, particularly those affecting the posterior inferior cerebellar artery and infarcts affecting multiple arterial territories may be accompanied by the development of brain swelling due to cytotoxic and vasogenic edema.^{15,19,41,47,51,56,58,75,84,91,94,104} So far it is not clear which factors promote edema formation after stroke. In addition to infarct size, other factors such as hemorrhagic transformation and inadequate collateral blood flow may be involved. Interestingly, large infarcts can be accompanied by only a small amount of edema, whereas comparatively small infarcts can result in extensive swelling.⁶³ The timing of edema formation can vary. According to data from neuroimaging studies, maximum swelling is reached in most patients between the 2nd and the 4th day but has also been reported as late as the 9th day, when patients were already regarded as being stable.^{15,47,51,58,61}

The 2 major complications of edema formation in the posterior fossa are obstructive hydrocephalus due to blockage of the fourth ventricle and direct compression of the midbrain and pons. Upward transtentorial herniation of the superior vermis cerebelli through the tentorial notch or downward herniation of the cerebellar tonsils through the foramen magnum are the most common causes of death in the acute phase.^{3,41,51,58,75,94,104,112}

Treatment

Conservative Treatment

Guidelines for conservative treatment are derived from those for acute ischemic stroke in general and include airway protection, adequate oxygenation, blood pressure management, management of blood glucose levels, body temperature, and prevention of deep venous thrombosis and pulmonary embolism.^{1,25} The level of consciousness should be monitored closely. Immediate imaging should be prompted in the case of clinical deterioration or occurrence of brainstem signs. Escalation of conservative treatment includes ventilation, deep sedation, and other intensive care measures. Medical treatment strategies for increased ICP are in accordance with those used in the management of supratentorial stroke and may include osmotic therapy, artificial coma, hyperventilation, barbiturates, buffers, and steroids.^{8,55} However, none of these treatments is supported by adequate evidence of efficacy from experimental studies or randomized clinical trials.^{1,25} In fact, several reports suggest that these measures are ineffective or even detrimental, at least in supratentorial infarction.^{8,55} No valid data are available in infratentorial stroke.

Surgical Treatment

Surgical therapy includes ventriculostomy and suboccipital decompressive craniectomy (SDC). In contrast to the controversial debate about surgical treatment of space-occupying supratentorial stroke, surgical intervention is widely accepted as the treatment of choice in cerebellar stroke with mass effect and strongly recommended in current stroke guidelines.^{1,25}

Ventricular Drainage

Ventriculostomy is most commonly performed by inserting an extraventricular drain in a lateral ventricle. This procedure represents the gold standard of treatment and offers the opportunity for both measuring the supratentorial ICP and, if necessary, draining CSF. Although few authors have described a lower risk for infections in endoscopic ventriculostomy, this technique remains an individual approach (only used by some surgeons) and entails, besides a higher risk of surgical complications, the additional risk that CSF drainage is not controllable and might potentially be insufficient.^{7,123}

Suboccipital Craniectomy

The rationale for suboccipital decompressive surgery is to create space for the swollen tissue outside the narrow posterior fossa and thus avoid life-threatening compression of the brainstem and fourth ventricle. There are various surgical approaches: unilateral or bilateral craniectomy, additional opening of the foramen magnum with or without resection of the posterior arch of the atlas, and the use of different types of duraplasty. Some neurosurgeons also remove necrotic cerebellar tissue to create extra space within the posterior fossa and reduce development of cytotoxic edema.^{51,56,91}

In cerebellar stroke, there is no general agreement about the optimal time to initiate surgery or about what

Space-occupying cerebellar infarction

factors would mandate proceeding to surgery. Local surgery protocols differ greatly, and the questions of whether and when ventriculostomy or SDC should be performed and whether surgery should be performed in a single intervention, in combination, or in a stepwise approach remain unanswered. It is important to note that none of these procedures has been tested or compared with others in larger, multicenter, and prospective studies with adequate control groups or in randomized trials.

As for SDC, the following protocol may be used: Patients are operated in a prone or semiprone (park-bench) position. After vertical midline skin incision from the protuberantia occipitalis to the upper cervical spine, skin and subcutaneous tissue are separated from the underlying fascia, which is cut through laterally to the processus spinosus of the upper cervical spine, and muscles are disconnected from the processus spinosus and the occiput. Osteoclastic bilateral craniotomy is performed beneath the transverse sinus and enlarged laterally. Great attention must be paid to opening the foramen magnum sufficiently to decompress the cerebellar tonsils. Resection of the posterior arch of the atlas is recommended in order to achieve sufficient decompression. To maximize the decompressive effect, the dura should be opened (Y-shaped) above the cerebellar hemispheres and the medulla oblongata. If the effect of decompression seems insufficient, removal of necrotic tissue might be considered as an additional option. Finally, the dura should be reconstructed by means of duraplasty with an autologous galea or fascia flap or, more commonly, an artificial dural substitute. Complications such as injury to the vertebral arteries, induction of major bleeding, or epidural and subdural hematoma and hygroma should be avoided.¹¹⁸

Outcome

The analysis of available studies on space-occupying cerebellar stroke showed that conservative treatment alone is associated with a high mortality rate of 42.9%, increasing to a rate as high as 85% in those patients who progress to coma.^{27,41}

Surgical treatment is associated with much higher survival rates: 81.6% for patients treated with EVD alone, 76.8% for those treated with SDC alone, and 77.5% for those treated with both EVD and SDC. Mortality and functional outcomes are summarized in Tables 1 and 2. There is some evidence that higher age is associated with poorer outcome.^{47,56}

The summary tables should be interpreted with caution as most of the studies investigating outcome suffer from the limitations of retrospective data collection, lack of control groups, single-center or small multicenter (oligo-center) experience, limited size of patient cohorts, and other selection biases (Table 3). Most importantly, there were marked imbalances between treatment groups; i.e., patients with pronounced mass effect on CT and impaired level of consciousness were more likely to be treated with SDC and additional EVD. Most studies do not provide information about factors predicting the course of the disease or functional outcome in survivors.

The only study with a prospective multicenter design

TABLE 1: Mortality stratified by treatment strategy in 764 patients with space-occupying cerebellar stroke*

Variable	Dead	Survived
overall outcome	213 (27.9)	551 (72.1)
conservative treatment	85 (42.9)	113 (57.1)
SDC w/o EVD	29 (23.2)	96 (76.8)
SDC w/ EVD	39 (22.5)	134 (77.5)
EVD alone	27 (18.4)	120 (81.6)
SDC w/ strokectomy†	33 (26.6)	91 (73.4)

* Values represent numbers of patients (%).

† Patients in this category are also included in the SDC w/o EVD or SDC w/ EVD categories above.

addressing clinical outcome after space-occupying cerebellar stroke is the German-Austrian Space-Occupying Cerebellar Infarction Study (GASCIS).⁵¹ In GASCIS, the level of consciousness before treatment was found to be the only predictor for poor outcome. Patients who were awake/drowsy or somnolent/stuporous had a good functional outcome (mRS score 0–2) in 86% and 76% of cases, respectively, while only 47% of comatose patients had a good outcome. GASCIS was not randomized and therefore was subject to selection bias. In addition, 22.2% of patients treated with ventriculostomy were later treated with decompressive surgery due to further deterioration.

The observation that about 50% of patients who progress to coma and are treated with craniectomy have good outcomes (mRS score ≤ 2) is seen in some of the published studies, but others found no association between the initial level of consciousness or its course and functional outcome.^{44,47,51,57,68,69,91,112,115}

Today most neurosurgeons and neurologists believe that the clinical outcome of patients with space-occupying cerebellar stroke who have undergone successful life-saving surgery is usually good irrespective of other prognostic factors. This opinion is reflected by current guidelines and recommendations.^{1,12,25,51,78,94} However, little is known about the long-term outcome in survivors. There are only

TABLE 2: Outcome stratified by treatment strategy in 299 survivors of space-occupying cerebellar stroke

Variable	Disability (%)*		
	None or Mild	Moderate	Severe
overall outcome	162 (54.4)	101 (33.9)	35 (11.7)
conservative treatment	58 (78.4)	15 (20.3)	1 (1.4)
SDC w/o EVD	13 (40.6)	13 (40.6)	6 (18.8)
SDC w/ EVD	39 (45.3)	33 (38.4)	14 (16.3)
EVD alone	36 (50.7)	25 (35.2)	10 (14.1)
SDC w/ strokectomy†	50 (47.6)	43 (41.0)	12 (11.4)

* None or mild disability was defined as mRS Score 0 or 1, GOS Score 5, or BI Score 90–100; moderate disability as mRS Score 2 or 3, GOS Score 4, or BI Score 65–85; and severe disability as mRS Score 4 or 5, GOS Score 2 or 3, or BI Score ≤ 60.

† Patients in this category are also included in the SDC w/o EVD or SDC w/ EVD categories above.

TABLE 3: Summary of available publications reporting on patients with space-occupying cerebellar infarction*

Authors & Year	No. of Pts†	Mean Age (yrs)	Sex	Conservative Treatment	SDC w/o EVD	SDC w/ EVD	EVD Alone	Mean FU in Mos (range)
Menzies, 1893	1	27	1 F	1 (dead)	0	0	0	NR
Goodhart & Davidson, 1936	1	66	1 F	1 (dead)	0	0	0	NR
Germain & Morvan, 1936	1	32	1 M	1 (dead)	0	0	0	NR
Kubik & Adams, 1946	2	75	1 F, 1 M	2 (dead)	0	0	0	NR
Lindgren, 1956	2	47.5	2 M	0	2 (1 dead, 1 surv, outc NR)	0	0	1
Fairburn & Oliver, 1956	3	58.3	3 M	2 (1 dead, 1 w/o disab)	1(+) (dead)	0	0	19 (5–33)
Ford & Clark, 1956	1	37	1 M	1 (dead)	0	0	0	NR
Pomme et al., 1959	2	59.5	2 M	0	2 (1 dead, 1 surv, outc NR)	0	0	NR
Fisher et al., 1961	2	48.5	2 F	2 (dead)	0	0	0	NR
Norris et al., 1969	3	NR	NR	0	3 (3 surv, outc NR)	0	0	NR
Wood & Murphey, 1969	5	43.8	5 M	0	5 (2 dead, 3 surv, outc NR)	0	0	NR
Lehrich et al., 1970	4	59.3	1 F, 3 M	0	3 (3+) (1 dead, 2 surv, outc NR)	0	1 (dead)	43 (1–85)
Cohadon et al., 1970	2	36	1 F	0	2 (2 surv, outcome NR)	0	0	NR
Corcos et al., 1971	1	NR	NR	0	1 (dead)	0	0	NR
Fischer et al., 1972	1	0.4	1 M	0	0	1 (surv, outc NR)	0	4.5
Graham & Adams, 1972	2	30	2 M	1 (dead)	0	0	1 (dead)	NR
Momose & Lehrich, 1973	13	52.9	6 F, 6 M, 1 NR	4 (4 dead)	9 (9+) (1 dead, 8 surv, outc NR)	0	0	NR
Sypert & Alvord, 1975	28	63	7 F, 21 M	28 (28 dead)	0	0	0	NR
Duncan et al., 1975	2	65.5	2 M	1 (dead)	1 (surv, mRS0)	0	0	2
Ferguson et al., 1976	1	NR	NR	0	0	1 (surv, outc NR)	0	NR
Savitz et al., 1977	1	60	1 M	0	0	1 (surv, mRS1)	0	12
Miki et al., 1978	2	NR	NR	0	2 (2 surv, outc NR)	0	0	NR
Levina, 1978	1	NR	NR	NR	NR	NR	NR	NR
Géraud et al., 1978	3	NR	NR	0	0	2 (1 dead, 1 surv, outc NR)	1 (1 surv w/ shunt, outc NR)	NR
Vereshchagin et al., 1978	1	NR	NR	0	1 (surv, outc NR)	0	0	NR
De Reuck & Vander Eecken, 1978	4	64.5	1 F, 3 M	3 (3 dead)	0	0	1 (surv, outc NR)	NR
George et al., 1978	4	NR	NR	0	4 (2 dead, 2 surv, outc NR)	0	0	NR
Arnow et al., 1978	1	19	1 M	1 (dead)	0	0	0	NR
Greenberg et al., 1979	2	65	2 M	0	0	0	2 (1 surv no disab, 1 dependent w/ shunt)	10
Feely, 1979	2	40.5	2 F	0	2 (2 surv, 1 no disab, 1 NR)	0	0	NR
Schmidek & Guthikonda, 1979	1	NR	NR	0	1 (surv, outc NR)	0	0	NR
Heros, 1979	1	22	1 M	0	0	1 (surv, outc NR)	0	NR
Cuneo et al., 1979	1	23	1 M	1 (dead)	0	0	0	NR

(continued)

Space-occupying cerebellar infarction

TABLE 3: Summary of available publications reporting on patients with space-occupying cerebellar infarction* (continued)

Authors & Year	No. of Pts†	Mean Age (yrs)	Sex	Conservative Treatment	SDC w/o EVD	SDC w/ EVD	EVD Alone	Mean FU in Mos (range)
Spinnler et al., 1979	6	NR	NR	1 (surv, outc NR)	5 (2 dead, 3 surv, outc NR)		0	NR
Jones et al., 1980	NR	NR	NR	NR	NR	NR	NR	NR
Scotti et al., 1980	6	NR	NR	1 (surv, outc NR)	5 (5+) (2 dead, 3 surv, outc NR)	0	0	NR
Patrick et al., 1980	2	NR	NR	NR	2 (NR)	0	0	NR
Woodhurst, 1980	4	NR	NR	0	2 (1 dead, 1 surv, outc NR)	2 (1 dead, 1 surv, outc NR)	0	NR
Hinshaw et al., 1980	NR	NR	NR	NR	0	0	0	NR
Sayama et al., 1981	5	NR	NR	0	4 (4 surv, outc NR)	0	1 (dead)	NR
Bergen et al., 1981	1	0.8	1 M	0	0	0	1 (surv w/ shunt, outc "normal")	30
Ho et al., 1981	3	71.3	1 F, 2 M	2 (2 surv, outc NR)	1 (surv, outc NR)	0	0	NR
Shenkin & Zavala, 1982	6	61.8	2 F, 4 M	0	0	0	6 (6 surv, outc 4 independent, 2 mild–mod disab, 4 w/ shunt)	NR
Taneda et al., 1982	15	59.3	2 F, 13 M	5 (5 dead)	7 (1 dead, 6 surv, no disab)	3 (1 dead, 2 surv, 1 no disab, 1 NR)	0	NR
Harbaugh et al., 1982	1	13	1 M	0	0	0	1 (surv, outc NR)	2
Khan et al., 1983	11	64.2	5 F, 6 M	6 (6 surv, 2 no disab, 4 mild–mod disab)	0	0	5 (1 dead, 4 mild–mod disab)	NR
Horwitz & Ludolph, 1983	2	59	2 M	1 (surv, outc NR)	0	1 (surv, outc NR)	0	NR
Shigemori et al., 1983	1	68	1 F	0	0	0	1 (surv, mod disab)	3
Derlon et al., 1983	1	19	1 M	0	0	1 (+) (surv, outc NR)	0	NR
Duncan & Forman, 1984	1	75	1 M	0	0	0	1 (surv, mild–mod disab w/ shunt)	0.7
Laun et al., 1984	7	47.7	3 F, 4 M	1 (dead)	1 (surv, outc NR)	3 (3 surv, outc NR)	2 (2 surv, outc 1 severe disab, 1 NR)	2.5
Busse et al., 1984	8	52.5	4 F, 4 M	2 (2 dead)	1 (+) (surv, outc NR)	4 (4+) (4 surv, outc NR)	1 (surv, outc NR)	1.2
Keidel et al. 1984	3	60	1 F, 2 M	1 (dead)	0	0	2 (2 surv, outc NR)	1.1
Rousseaux et al., 1985	1	63	1 F	1 (surv, outc NR)	0	0	0	NR
Tomaszek & Rosner, 1985	1	51	1 F	1 (dead)	0	0	0	NR
Cioffi et al., 1985	10	53.6	4 F, 6 M	5 (5 surv, outc NR)	0	1 (surv, outc NR)	4 (1 dead, 3 surv, outc NR)	24
Auer et al., 1986	13	NR	NR	5 (1 dead, 4 surv, outc NR)	7 (7 surv, outc NR)	0	1 (dead)	NR

(continued)

TABLE 3: Summary of available publications reporting on patients with space-occupying cerebellar infarction* (continued)

Authors & Year	No. of Pts†	Mean Age (yrs)	Sex	Conservative Treatment	SDC w/o EVD	SDC w/ EVD	EVD Alone	Mean FU in Mos (range)
Macdonell et al., 1987	3	NR	NR	1 (dead)	2 (1 dead, 1 surv, outc NR)	0	0	NR
Busse & Laun, 1988	24	NR	NR	5 (3 dead, 2 surv, outc NR)	6 (6+) (1 dead, 5 surv, 4 mild–mod disab, 1 severe disab)	7 (7 surv, 6 mild–mod disab, 1 severe disab)	6 (1 dead, 5 surv, 3 mild–mod disab, 2 severe disab)	3
Krieger & Aschoff, 1989	1	45	1 M	0	0	1 (+) (surv, outc NR)	0	NR
Hoyer et al., 1990	9	NR	NR	5 (2 dead, 3 surv, outc NR)	1 (+) (surv, outc NR)	0	3 (1 dead, 2 surv, outc NR)	NR
Andoh et al., 1990	9	NR	NR	NR	NR	NR	9 (surv, outcome NR)	NR
Klugkist & McCarthy, 1991	3	49.3	3 M	0	1 (surv, severe disab)	1 (surv, mild–mod disab)	1 (surv, mild–mod disab)	27.7 (max 54)
Hoyer et al., 1991	8	NR	NR	1 (dead)	0	1 (surv, outc NR)	6 (6 surv, outc NR)	NR
Amarenco et al., 1991	6	NR	NR	6 (6 dead)	0	0	0	NR
Struck et al., 1991	4	NR	NR	2 (2 dead)	1 (surv, GOS 2)	0	1 (surv, outc NR)	NR
Chen et al., 1992	11	54.1	4 F, 7 M	0	0	11 (11 surv, 6 mild–mod disab, 3 severe disab, 2 outc NR)	0	(16–60)
Antonello et al., 1992	1	73	1 M	0	0	0	1 (surv, outc NR)	NR
Bertalanffy & de Vries, 1992	10	61.8	6 F, 4 M	0	0	0	10 (3 dead, 6 surv, outc NR, 1 NR)	NR
Langmayr et al., 1993	15	NR	NR	0	8 (8+) (outc I [no Sx]: 1; II [mild–mod disab]: 2; III [severe disab]: 3; IV (dead): 6)	4 (4+)	3 (outc NR)	3
Kase et al., 1993	10	NR	NR	3 (3 dead)	6 (2+) (6 outc NR)	0	1 (outc NR)	NR
Kase & Wolf, 1993	1	55	1 M	0	0	1 (1 surv, no disab)	0	2
Tulyapronchote et al., 1993	1	62	1 M	0	0	0	1 (surv mild disab)	NR
Krieger et al., 1993	11	52	3 F, 8 M	0	11 (11+) (4 dead, 7 surv, outc NR)	0	0	NR
Tohgi et al., 1993	72	NR	NR	NR	NR	NR	NR	3
Hornig et al., 1994	52	61.2	18 F 34 M	16 (2 dead, 14 surv, outc 11 no or mild disab, 3 NR)	8 (8+) (4 dead, 26 surv, outc 12 NR)	22 14 mild–mod disab,	6 (2 dead, 4 surv mild–mod disab)	NR
Kanis et al., 1994	1	60	1 M	0	0	1 (dead)	0	1
Miyachi et al., 1994	1	49	1 M	0	0	0	1 (dead)	3
Tada et al., 1994	1	65	1 M	1 (surv, outc NR)	0	0	0	1.8
Ito et al., 1994	2	66.5	2 F	0	0	0	2 (2 surv, outc NR)	NR
Miyata et al., 1994	1	11	1 F	0	1 (1+)	0	0	36

(continued)

Space-occupying cerebellar infarction

TABLE 3: Summary of available publications reporting on patients with space-occupying cerebellar infarction* (continued)

Authors & Year	No. of Pts†	Mean Age (yrs)	Sex	Conservative Treatment	SDC w/o EVD	SDC w/ EVD	EVD Alone	Mean FU in Mos (range)
Ogasawara et al., 1995	10	59	3 F, 7 M	0	0	10 (2 dead, 8 surv, outc 3 no disab, 3 mild disab, 1 severe disab, 1 NR)	0	(23–101)
Mathew et al., 1995	19	NR	NR	6 (3 dead, 3 surv, outc NR)	2 (1 dead, 1 surv outc “good”)	2 (2 surv outc “good”)	9 (3 dead, 6 surv outc 5 “good,” 1 mod disab)	NR
Turgut et al., 1996	5	NR	NR	3 (1 dead, 2 mRS ≤3)	2 (1 dead, 1 mRS ≤3)	0	0	NR
Matsumoto et al., 1996	1	61	1 M	0	0	1 (surv, outc NR)	0	12
Sagoh et al., 1997	1	38	1 M	0	0	0	1 (surv, outc NR)	12
Calli et al., 1998	1	42	1 M	0	0	1 (surv, outc NR)	0	NR
Wijdicks et al., 1998	1	47	1 M	0	0	0	1 (surv, outc NR)	NR
Mohsenipour et al., 1999	NR	NR	NR	NR	NR	NR	NR	min 6
Jauss et al., 1999	84	58.5	28 F, 56 M	36 (30 mRS 0–1)	34 (22 mRS 0–1, 12 outc NR)		14 (10 mRS 0–1)	3
Skurnik et al., 1999	NR	NR	NR	NR	NR	NR	NR	NR
Koh et al., 2000 ⁶³	35	NR	NR	26 (1 dead, 25 surv, outc 14 mRS 0–2, 10 mRS 3–4, 1 mRS 5, FU NR)	2 (2 surv, outc 1 mRS 3–4, 1 mRS 5, FU NR)	1 (surv, mRS 3–4, FU NR)	6 (1 dead, 5 surv, outc 2 mRS 0–2, 3 mRS 3–4, FU NR)	16 (1–105)
Koh et al., 2000 ⁶⁴	2	51.5	1 F, 1 M	0	0	2 (1 dead, 1 surv GOS 5)	0	NR
Hirayama et al., 2000	5	NR	NR	0	0	1 (surv, independent)	4 (4 NR)	3
Raco et al., 2003	17	NR	NR	NR	4 (4+) (2 dead, 2 surv, 1 GOS 4, 1 GOS 3)	5 (5+) (1 dead, 4 surv, 3 GOS 5, 1 GOS 4)	8 (8 surv 7 GOS 5, 1 GOS 4)	NR
Pérez Navero et al., 2003	1	4	1 F	1 (dead)	0	0	0	NR
Ganapathy et al., 2003	1	61	1 F	0	0	1 (1+) (mRS 3)	0	NR
Prat et al., 2004	2	70	1 F, 1 M	1 (surv, outc NR)	1 (+) (surv, outc NR)	0	0	NR
Czernicki & Marchel, 2004	5	NR	NR	NR	NR	NR	NR	NR
Rupprecht et al., 2004	1	59	1 M	0	0	0	1 (surv, outc NR)	1
Jensen & St. Louis, 2005	1	48	1 F	1 (surv, outc NR)	0	0	0	NR
Baldauf et al., 2006	10	62	4 F, 6 M	0	0	1 (surv, GOS 2)	9 (9 surv, 1 GOS 2, 4 GOS 3, 2 GOS 4, 2 GOS 5)	NR
Ramos-Zuñiga & Jiménez-Guerra, 2006	8	63.5	3 F, 5 M	3 (3 surv, outc NR)	0	0	5 (5 surv, outc NR)	3

(continued)

TABLE 3: Summary of available publications reporting on patients with space-occupying cerebellar infarction* (*continued*)

Authors & Year	No. of Pts†	Mean Age (yrs)	Sex	Conservative Treatment	SDC w/o EVD	SDC w/ EVD	EVD Alone	Mean FU in Mos (range)
Rosi et al., 2006	12	NR	NR	1 (outc NR)	7 (2 dead, 5 surv, outc NR.)	0	4 (3 dead, 1 surv, outc NR)	NR
Kudo et al., 2007	25	62.6	4 F, 21 M	0	5 (3+) (1 dead, 4 surv, 1 GOS 5, 2 GOS 4, 1 GOS 3)	17 (14+) (1 dead, 16 surv, 9 GOS 5, 4 GOS 4, 3 GOS 3)	3 (1 dead, 2 surv, 1 GOS 5, 1 GOS 3)	NR
Cerimagic et al., 2007	1	46	1 M	0	0	0	1 (surv, outc NR)	4
Yoshimura et al., 2007	5	71.8	2 F, 3 M	0	0	0	5 (1 dead, 4 surv, 2 GOS 5, 1 GOS 4, 1 GOS 2)	3
Hsieh et al., 2008	1	51	1 M	0	0	0	1 (surv, outc NR)	1
Mangubat et al., 2009	4	NR	NR	0	0	2 (1 dead, 1 surv, outc NR)	2 (2 surv, outc NR)	NR
Jüttler et al 2009	56 (25+)	57.7	19 F, 36 M	0	8 (1 dead, 7 surv, 2 no disab, 5 mild–mod disab)	39 (17 dead, 22 surv, 10 no disab, 7 mild–mod disab, 5 severe disab)	9 (4 dead, 5 surv, 4 no disab, 1 mild–mod disab)	96 (41–143)
Pfefferkorn et al., 2009	52	59.2	18 F, 34 M	0	52 (52+) (21 dead, 31 surv, 13 no disab, 14 mild–mod disab, 4 severe disab)		0	55 (12–132)
Lungu et al., 2010	5	NR	NR	0	5 (5+) (1 dead, 4 surv, outc NR)	0	0	12
Tsitsopoulos et al., 2011 ¹¹⁴	10	54.9	2 F, 8 M	0	0	10 (10+) (2 dead, 8 surv, 1 mRS0, 2 mRS1, 3 mRS2, 2 mRS4)	0	57.8 (15–118)
Tsitsopoulos et al., 2011 ¹¹⁵	32	64.3	8 F, 24 M	0	0	32 (32+) (10 dead, 22 surv, 17 mRS 0–2, 5 mRS 3–5)	0	67.5 (14–113)
Montgomery et al., 2012	4	8	4 M	0	0	4 (2+) (2 surv, 2 mild disab, 2 mod disab)	0	36 (12–60)

* Shading indicates combined categories. Abbreviations: disab = disability; FU = follow-up; max = maximum; min = minimum; mod = moderate; NR = not reported; outc = outcome; pts = patients; surv = survived; Sx = symptoms; + = strokectomy.

† Number of patients with space-occupying cerebellar infarction.

3 studies on long-term outcome that include larger numbers of patients with follow-up of more than 1 year.^{56,91,115} In one of these studies, a retrospective single-center case series on SDC plus EVD and strokectomy (removal of necrotic tissue) in 32 patients with expansive unilateral cerebellar infarcts, a low mortality rate (32%) and a high rate of independent outcome (53% mRS score 0–2) was found in long-term survivors.¹¹⁵ In contrast, the 2 other studies, which included 108 patients treated with different strategies, reported a lower long-term survival rate of 60%. Only 27% of survivors showed a favorable outcome (mRS score 0–1) and 38% showed an independent outcome (mRS score 0–2). Age and the presence of brainstem infarction were identified as independent prognostic factors. Interestingly, insertion of an external ventricular drain, either alone or in combination with decompressive craniectomy, seemed

to have no effect on survival or outcome.^{56,91} These results call into question the belief that prognosis in survivors of space-occupying cerebellar infarcts is usually good. Indeed they indicate very heterogeneous outcome results, with 40% of patients dying and a considerable number of patients left with severe neurological deficits. However, the authors of these 2 studies were unable to assess the specific causes of death and severe disability in these patients. Both of the studies are retrospective and single center in nature and therefore also subject to bias.

Suggested Treatment Algorithm

The following treatment algorithm is based on treatment protocols from experienced and expert tertiary centers where large numbers of patients are treated every

Space-occupying cerebellar infarction

year for space-occupying cerebellar infarctions (Fig. 1). Nevertheless, it represents only a recommendation that can be adapted to local standards and conditions.

Alert and clinically stable patients should initially be treated conservatively and monitored closely using clinical scales and repeated neuroimaging.^{54,57,94} If feasible, these patients should be admitted to an ICU or a dedicated stroke unit where neurosurgical interventions can be performed 24 hours a day.

Somnolent or clinically critical patients should ideally be admitted to an ICU, although they may also be cared for in a dedicated stroke unit. In contrast to the stable patients described above, these critically ill patients should not, under any circumstances, be admitted to a general ward. If neuroimaging shows extensive cerebellar stroke in these patients (quantitatively assessed according to the numerical 3-item CT score suggested by Jauss and coworkers⁵²), prophylactic SDC with or without EVD should be considered. Some authors believe that deterioration of consciousness is the strongest predictor for dangerous space occupation and should be a prime factor in the decision to intervene surgically.⁶⁹ However, because clinical signs as well as neuroradiological parameters of deterioration are unspecific or may be detected too late, some authors recommend early or preventive neurosurgical therapy.^{12,69,78} They argue that the risk of EVD and SDC is comparatively small and patients undergoing preventive surgery tend to recover better than patients undergoing emergency treatment after deterioration. Others argue that SDC should not generally be recommended in

patients with a level of consciousness better than coma because its efficacy is unproven and it may cause unnecessary complications.⁵¹ Similarly, prophylactic osmotherapy can be considered if there is radiological evidence of edema, but given the lack of evidence it is not recommended.

In patients whose condition deteriorates, neuroimaging should be performed immediately and frequently to determine the appropriate treatment strategy and, if possible, to rule out extensive brainstem infarction. If the fourth ventricle is blocked and hydrocephalus is present, EVD or SDC or both are recommended, with ventriculostomy to be definitely followed by SDC if the patient's condition does not improve or if it worsens.^{12,41,94} Although ventriculostomy alone is sometimes considered dangerous due to possible upward herniation, this risk seems rather small.^{15,41,47,58,68,69,94,116} Patients with signs of quadrigeminal cistern and/or brainstem compression should undergo immediate SDC combined with or followed by EVD if they have signs of hydrocephalus that do not resolve after SDC.^{12,41,94} Some authors regard the combined approach of ventriculostomy and decompressive surgery as the first choice for patients with occlusive hydrocephalus.^{15,27,44,54}

In comatose patients, neuroimaging and/or brainstem auditory evoked potential (BAEP) and somatosensory evoked potential (SEP) studies should be considered to detect brainstem damage, and some reports have emphasized the potential usefulness of the latter modalities.^{65,66} In patients without brainstem infarction and normal find-

Fig 1. Cerebellar stroke treatment algorithm. Strokectomy refers to removal of necrotic tissue. GCS = Glasgow Coma Scale.

ings on BAEP/SEP studies, immediate decompressive surgery plus EVD should be performed. For patients with extensive brainstem infarction or highly pathological BAEP/SEP findings, conservative treatment is recommended in view of the poor prognosis. If neither MRI nor BAEP/SEP studies can be performed, no general recommendation can be given, and therapeutic decisions have to be made on a case-by-case basis.

Conclusions

Although most cerebellar infarctions have a benign course, the early identification of those patients who are at risk for space-occupying edema formation is essential. In addition to monitoring patients closely and using MRI rather than CT for diagnostic evaluation, one should keep in mind that deterioration can occur late and must not be overlooked. Bilateral decompression craniotomy, including resection of the posterior arch of the atlas and in combination with dura enlargement, is thought to be the treatment of choice, even if patients present with a reduced level of consciousness. The available data, however, do not provide sufficient evidence to suggest the optimal timing of surgery or the optimal surgical procedure. Patients with extensive brainstem damage, however, have a poor outcome, and thus seem not to be good candidates for surgery. Furthermore, recent studies have shown that long-term outcome after space-occupying cerebellar infarction is more heterogeneous than expected, and therefore more studies are needed to identify prognostic factors. Randomized controlled trials would be needed to define the exact value of SDC, ventriculostomy, or a combination of both. Ethically, however, these trials are highly problematic because they might mean withholding from patients a potentially life-saving therapy such as SDC. It is the great challenge for neurosurgeons and neurologists to overcome these problems and create adequately designed trials that answer the open questions about space-occupying cerebellar infarction.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Neugebauer, Witsch, Jüttler. Acquisition of data: Neugebauer, Jüttler. Analysis and interpretation of data: Neugebauer, Zweckberger, Jüttler. Drafting the article: Neugebauer, Witsch, Jüttler. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Neugebauer. Statistical analysis: Neugebauer, Jüttler. Administrative/technical/material support: Neugebauer, Jüttler. Study supervision: Neugebauer, Jüttler.

References

- Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, et al: Guidelines for the early management of adults with ischaemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. **Circulation** **115**:e478–e534, 2007 (Erratum in **Circulation** **116**:e515, 2007)
- Amarenco P, Roullet E, Goujon C, Chéron F, Hauw JJ, Bousser MG: Infarction in the anterior rostral cerebellum (the territory of the lateral branch of the superior cerebellar artery). **Neurology** **41**:253–258, 1991
- Andoh T, Sakai N, Yamada H, Hattori T, Miwa Y, Hirata T, et al: [Cerebellar infarction: analysis of 33 cases.] **No Shinkei Geka** **18**:821–828, 1990 (Jpn)
- Antonello RM, Pasqua M, Bosco A, Torre P: Massive cerebellar infarct complicated by hydrocephalus. **Ital J Neurol Sci** **13**:695–698, 1992
- Arnow PM, Panwalker A, Garvin JS, Rodriguez-Erdmann F: Aspirin, hyperventilation, and cerebellar infarction in sickle cell disease. **Arch Intern Med** **138**:148–149, 1978
- Auer LM, Auer T, Sayama I: Indications for surgical treatment of cerebellar haemorrhage and infarction. **Acta Neurochir (Wien)** **79**:74–79, 1986
- Baldauf J, Oertel J, Gaab MR, Schroeder HW: Endoscopic third ventriculostomy for occlusive hydrocephalus caused by cerebellar infarction. **Neurosurgery** **59**:539–544, 2006
- Bardutzky J, Schwab S: Antiedema therapy in ischemic stroke. **Stroke** **38**:3084–3094, 2007
- Bergen BJ, Batnitzky S, Morantz RA, Price HI: Cerebellar infarction with associated acute hydrocephalus due to vertebral artery occlusion in a child. **Neurosurgery** **8**:383–387, 1981
- Bertalanffy H, de Vries J: Management of cerebellar infarction with associated occlusive hydrocephalus. **Clin Neurol Neurosurg** **94**:19–23, 1992
- Busse O, Laun A: Therapie des raumfordernden Kleinhirnin-farktes. **Akt Neurol** **15**:6–8, 1988
- Busse O, Laun A, Agnoli AL: [Obstructive hydrocephalus in cerebellar infarcts.] **Fortschr Neurol Psychiatr** **52**:164–171, 1984 (Ger)
- Calli C, Katranci N, Guzelbag E, Alper H, Yuntun N: Retro-clival epidural hematoma secondary to decompressive craniectomy in cerebellar infarction: MR demonstration. **J Neuro-radiol** **25**:229–232, 1998
- Cerimagic D, Glavic J, Lovrencic-Huzjan A, Demarin V: Occlusion of vertebral artery, cerebellar infarction and obstructive hydrocephalus following cervical spine manipulation. **Eur Neurol** **58**:248–250, 2007
- Chen HJ, Lee TC, Wei CP: Treatment of cerebellar infarction by decompressive suboccipital craniectomy. **Stroke** **23**:957–961, 1992
- Cioffi FA, Bernini FP, Punzo A, D'Avanzo R: Surgical management of acute cerebellar infarction. **Acta Neurochir (Wien)** **74**:105–112, 1985
- Cohadon F, Guérin A, Castel JP, Leifer C, Caillé M, Leman P: [Pseudo-tumorous softening of the cerebellum.] **Bord Med** **3**:285–297, 1970 (Fr)
- Corcos A, Gaches J, Le Beau J: [Pseudosurgical form of ischemic pontocerebellar edema.] **Ann Med Interne (Paris)** **122**:861–865, 1971 (Fr)
- Cuneo RA, Caronna JJ, Pitts L, Townsend J, Winestock DP: Upward transtentorial herniation: seven cases and a literature review. **Arch Neurol** **36**:618–623, 1979
- Czernicki T, Marchel A: [Results of treatment of cerebellar infarctions.] **Neurol Neurochir Pol** **38**:37–43, 2004 (Polish)
- De Reuck J, Vander Eecken H: Cerebellar infarction and internal hydrocephalus. **Acta Neurol Belg** **78**:129–140, 1978
- Derlon JM, Charbonneau P, Théron J, Houtteville JP, Bazin C: [Edematous cerebellar infarction in the young subject.] **Neurochirurgie** **29**:423–428, 1983 (Fr)
- Duncan GW, Parker SW, Fisher CM: Acute cerebellar infarction in the PICA territory. **Arch Neurol** **32**:364–368, 1975

Space-occupying cerebellar infarction

24. Duncan JS, Forman A: Hydrocephalus after cerebellar infarction. **Br Med J (Clin Res Ed)** **289**:1301–1302, 1984
25. European Stroke Organisation (ESO) Executive Committee: Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. **Cerebrovasc Dis** **25**:457–507, 2008
26. Fairburn B, Oliver LC: Cerebellar softening: a surgical emergency. **BMJ** **1**:1335–1336, 1956
27. Feely MP: Cerebellar infarction. **Neurosurgery** **4**:7–11, 1979
28. Ferguson L, Sundaresan N, Richardson R, Marsan R: Acute hemorrhagic cerebellar infarction following angiography. **Surg Neurol** **5**:176–178, 1976
29. Fischer EG, Strand RD, Gilles FH: Cerebellar necrosis simulating tumor in infancy. **J Pediatr** **81**:98–100, 1972
30. Fisher CM, Karnes WE, Kubik CS: Lateral medullary infarction—the pattern of vascular occlusion. **J Neuropathol Exp Neurol** **20**:323–379, 1961
31. Ford FR, Clark D: Thrombosis of the basilar artery with softening in the cerebellum and brain stem due to manipulation of the neck; a report of two cases with one post-mortem examination, reasons are given to prove that damage to the vertebral arteries is responsible. **Bull Johns Hopkins Hosp** **98**:37–42, 1956
32. Ganapathy K, Girija T, Rajaram R, Mahendran S: Surgical management of massive cerebellar infarction. **J Clin Neurosci** **10**:362–364, 2003
33. George B, Cophignon J, George C, Lounnon J: [Surgical aspects of cerebellar infarctions based upon a series of 79 cases (author's transl).] **Neurochirurgie** **24**:83–88, 1978 (Fr)
34. Géraud G, Guillaume J, Lagarrigue J, Lazorthes Y, Bès A, Géraud J: [Pseudo-tumoral softening of the cerebellum.] **Rev Neurol (Paris)** **134**:183–195, 1978 (Fr)
35. Germain A, Morvan A: Ramollissement cérébelleux pseudo-tumoral. **Bull Soc Med Hosp Paris** **35**:501–524, 1936
36. Goodhart SP, Davison C: Syndrome of the posterior inferior and anterior inferior cerebellar arteries and their branches. **Arch Neurol Psychiatry** **35**:501–524, 1936
37. Graham DI, Adams H: "Idiopathic" thrombosis in the vertebrobasilar arterial system in young men. **BMJ** **1**:26–28, 1972
38. Greenberg J, Skubick D, Shenkin H: Acute hydrocephalus in cerebellar infarct and hemorrhage. **Neurology** **29**:409–413, 1979
39. Harbaugh RE, Saunders RL, Reeves AG: Pediatric cerebellar infarction: case report and review of the literature. **Neurosurgery** **10**:593–596, 1982
40. Heros RC: Cerebellar infarction resulting from traumatic occlusion of a vertebral artery. Case report. **J Neurosurg** **51**:111–113, 1979
41. Heros RC: Surgical treatment of cerebellar infarction. **Stroke** **23**:937–938, 1992
42. Hinshaw DB Jr, Thompson JR, Hasso AN, Casselman ES: Infarctions of the brainstem and cerebellum: a correlation of computed tomography and angiography. **Radiology** **137**:105–112, 1980
43. Hirayama A, Yamasaki S, Miyata M: Clinical use of lidocaine for control of stroke oedema in the posterior cranial fossa accompanied by acute hydrocephalus. **Acta Neurochir Suppl** **76**:317–321, 2000
44. Ho SU, Kim KS, Berenberg RA, Ho HT: Cerebellar infarction: a clinical and CT study. **Surg Neurol** **16**:350–352, 1981
45. Hojer C, Beil C, Neveling M, Szelies B, Haupt WF: [Diagnosis and prognosis of acute cerebellar infarcts. A retrospective study.] **Nervenarzt** **61**:482–490, 1990 (Ger)
46. Hojer C, Bewermeyer H, Huber M, Neveling M, Engel-Riedel W, Haupt WF: Kleinhirnininfarkte und Kleinhirnblutungen. Eine vergleichende, retrospektive Studie an 78 Patienten. **Akt Neurol** **18**:88–94, 1991
47. Hornig CR, Rust DS, Busse O, Jauss M, Laun A: Space-occupying cerebellar infarction. Clinical course and prognosis. **Stroke** **25**:372–374, 1994
48. Horwitz NH, Ludolph C: Acute obstructive hydrocephalus caused by cerebellar infarction. Treatment alternatives. **Surg Neurol** **20**:13–19, 1983
49. Hsieh CY, Lee JS, Yu CY, Chen CH: Bilateral cerebellar infarction in the medial branches of posterior inferior cerebellar arterial territory—using endoscopic third ventriculostomy to relieve acute hydrocephalus. **Clin Neurol Neurosurg** **110**:71–74, 2008
50. Ito M, Sonokawa T, Mishina H, Hishii M, Sato K: Surgical management of comatose patients with cerebellar infarction. **J Clin Neurosci** **1**:251–256, 1994
51. Jauss M, Krieger D, Hornig C, Schramm J, Busse O: Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. **J Neurol** **246**:257–264, 1999
52. Jauss M, Müffelmann B, Krieger D, Zeumer H, Busse O: A computed tomography score for assessment of mass effect in space-occupying cerebellar infarction. **J Neuroimaging** **11**:268–271, 2001
53. Jensen MB, St Louis EK: Management of acute cerebellar stroke. **Arch Neurol** **62**:537–544, 2005
54. Jones HR Jr, Millikan CH, Sandok BA: Temporal profile (clinical course) of acute vertebrobasilar system cerebral infarction. **Stroke** **11**:173–177, 1980
55. Jüttler E, Schellinger PD, Aschoff A, Zweckberger K, Unterberg A, Hacke W: Clinical review: therapy for refractory intracranial hypertension in ischaemic stroke. **Crit Care** **11**:231, 2007
56. Jüttler E, Schweickert S, Ringleb PA, Huttner HB, Köhrmann M, Aschoff A: Long-term outcome after surgical treatment for space-occupying cerebellar infarction: experience in 56 patients. **Stroke** **40**:3060–3066, 2009
57. Kanis KB, Ropper AH, Adelman LS: Homolateral hemiparesis as an early sign of cerebellar mass effect. **Neurology** **44**:2194–2197, 1994
58. Kase CS, Norrving B, Levine SR, Babikian VL, Chodosh EH, Wolf PA, et al: Cerebellar infarction. Clinical and anatomic observations in 66 cases. **Stroke** **24**:76–83, 1993
59. Kase CS, Wolf PA: Cerebellar infarction: upward transtentorial herniation after ventriculostomy. **Stroke** **24**:1096–1098, 1993
60. Keidel M, Galle G, Wiedmayer J, Taghavy A: [Malignant cerebellar infarct.] **Fortschr Neurol Psychiatr** **52**:277–283, 1984 (Ger)
61. Khan M, Polyzoidis KS, Adegbite AB, McQueen JD: Massive cerebellar infarction: "conservative" management. **Stroke** **14**:745–751, 1983
62. Klugkist H, McCarthy J: Surgical treatment of space-occupying cerebellar infarctions—4 1/2 years post-operative follow-up. **Neurosurg Rev** **14**:17–22, 1991
63. Koh MG, Phan TG, Atkinson JL, Wijdicks EF: Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. **Stroke** **31**:2062–2067, 2000
64. Koh MS, Goh KY, Tung MY, Chan C: Is decompressive craniectomy for acute cerebral infarction of any benefit? **Surg Neurol** **53**:225–230, 2000
65. Krieger D, Adams HP, Rieke K, Hacke W: Monitoring therapeutic efficacy of decompressive craniotomy in space occupying cerebellar infarcts using brain-stem auditory evoked potentials. **Electroencephalogr Clin Neurophysiol** **88**:261–270, 1993
66. Krieger D, Aschoff A: [Evoked potentials before and following decompression craniotomy in space-occupying cerebellar infarct.] **Nervenarzt** **60**:36–39, 1989 (Ger)
67. Kubik CS, Adams RD: Occlusion of the basilar artery; a clinical and pathological study. **Brain** **69**:73–121, 1946
68. Kudo H, Kawaguchi T, Minami H, Kuwamura K, Miyata M, Kohmura E: Controversy of surgical treatment for severe cerebellar infarction. **J Stroke Cerebrovasc Dis** **16**:259–262, 2007

69. Langmayr JJ, Buchberger W, Reindl H: [Cerebellar hemorrhage and cerebellar infarct: retrospective study of 125 cases.] **Wien Med Wochenschr** 143:131–133, 1993 (Ger)
70. Laun A, Busse O, Calatayud V, Klug N: Cerebellar infarcts in the area of the supply of the PICA and their surgical treatment. **Acta Neurochir (Wien)** 71:295–306, 1984
71. Lehrich JR, Winkler GF, Ojemann RG: Cerebellar infarction with brain stem compression. Diagnosis and surgical treatment. **Arch Neurol** 22:490–498, 1970
72. Levina G: [Cerebellar infarct in hypertensive crisis.] **Arkhl Patol** 40:68–70, 1978 (Russian)
73. Lindgren SO: Infarctions simulating brain tumours in the posterior fossa. **J Neurosurg** 13:575–581, 1956
74. Lungu G, Mortada J, Stilhart B: [Surgical treatment of cerebellar infarction: five case studies.] **Neurochirurgie** 56:59–62, 2010 (Ger)
75. Macdonell RA, Kalnins RM, Donnan GA: Cerebellar infarction: natural history, prognosis, and pathology. **Stroke** 18:849–855, 1987
76. Mangubat EZ, Chan M, Ruland S, Roitberg BZ: Hydrocephalus in posterior fossa lesions: ventriculostomy and permanent shunt rates by diagnosis. **Neurol Res** 31:668–673, 2009
77. Masdeu JC, Irimia P, Asenbaum S, Bogousslavsky J, Brainin M, Chabriat H, et al: EFNS guideline on neuroimaging in acute stroke. Report of an EFNS task force. **Eur J Neurol** 13:1271–1283, 2006
78. Mathew P, Teasdale G, Bannan A, Oluoch-Olunya D: Neurosurgical management of cerebellar haematoma and infarct. **J Neurol Neurosurg Psychiatry** 59:287–292, 1995
79. Matsumoto K, Kato A, Kohmura E, Fujinaka T, Fukuhara R: Acute cerebellar edema following intra-arterial tissue plasminogen activator thrombolysis for basilar artery occlusion—case report. **Neurol Med Chir (Tokyo)** 36:455–457, 1996
80. Menzies WF: Thrombosis of the inferior cerebellar artery. **Brain** 16:436–439, 1893
81. Miki K, Kurimoto T, Tani S, Ikeda Y, Kawamura Y, Someda K, et al: [On the surgical treatment of cerebellar infarction (author's transl).] **No Shinkei Geka** 6:707–713, 1978 (Jpn)
82. Miyachi S, Okamura K, Watanabe M, Inoue N, Nagatani T, Takagi T: Cerebellar stroke due to vertebral artery occlusion after cervical spine trauma. Two case reports. **Spine (Phila Pa 1976)** 19:83–88, 1994
83. Miyata I, Imaoka T, Masaoka T, Nishiura T, Ishimitsu H: Pediatric cerebellar infarction caused by atlantoaxial subluxation—case report. **Neurol Med Chir (Tokyo)** 34:241–245, 1994
84. Mohsenipour I, Gabl M, Schutzhard E, Twerdy K: Suboccipital decompressive surgery in cerebellar infarction. **Zentralbl Neurochir** 60:68–73, 1999
85. Momose KJ, Leirich JR: Acute cerebellar infarction presenting as a posterior fossa mass. **Radiology** 109:343–352, 1973
86. Montgomery AK, Maixner WJ, Wallace D, Wray A, Mackay MT: Decompressive craniectomy in childhood posterior circulation stroke: a case series and review of the literature. **Pediatr Neurol** 47:193–197, 2012
87. Norris JW, Eisen AA, Branch CL: Problems in cerebellar hemorrhage and infarction. **Neurology** 19:1043–1050, 1969
88. Ogasawara K, Koshu K, Nagamine Y, Fujiwara S, Mizoi K, Yoshimoto T: [Surgical decompression for massive cerebellar infarction.] **No Shinkei Geka** 23:43–48, 1995 (Jpn)
89. Patrick BK, Ramirez-Lassepas M, Synder BD: Temporal profile of vertebrobasilar territory infarction. Prognostic implications. **Stroke** 11:643–648, 1980
90. Pérez Navero JL, Montero-Shiemann C, Velasco Jabalquinto MJ, Ibarra de la Rosa I, Antón Gamero M, Salas Molina J: [Fatal outcome after severe cerebellar infarction due to spontaneous dissection of the vertebral artery.] **An Pediatr (Barc)** 58:385–389, 2003 (Span)
91. Pfefferkorn T, Eppinger U, Linn J, Birnbaum T, Herzog J, Straube A, et al: Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. **Stroke** 40:3045–3050, 2009
92. Pommé B, Terrasse J, Janny P, Gras H, Gibert J: [Pseudotumoral softening of the cerebellum; two cases.] **Sem Hop** 35:791–793, 1959 (Fr)
93. Prat R, Conde FJ, Febles P, Cortés S, Millán-Corada AM: [Space-occupying cerebellar infarcts: surgical or conservative treatment?] **Rev Neurol** 38:133–136, 2004 (Span)
94. Raco A, Caroli E, Isidori A, Salvati M: Management of acute cerebellar infarction: one institution's experience. **Neurosurgery** 53:1061–1066, 2003
95. Ramos-Zuñiga R, Jiménez-Guerra R: Rational management of transient obstructive hydrocephalus secondary to a cerebellar infarct. **Minim Invasive Neurosurg** 49:302–304, 2006
96. Rosi J Jr, de Oliveira PG, Montanaro AC, Gomes S, Godoy R: [Cerebellar infarction: analysis of 151 patients.] **Arq Neuropsiquiatr** 64 (2B):456–460, 2006 (Portugese)
97. Rousseaux M, Devos P, Lesoin F, Petit H: "Pseudotumoral" cystic cerebellar infarction with slow evolution. **Neurosurgery** 16:61–63, 1985
98. Rupprecht TA, Weil S, Winkler PA, Kreuzer E, Pfister HW: Successful cardiac surgery 24 hours after craniotomy in a patient with infective endocarditis and embolic cerebellar infarction: case report. **J Heart Valve Dis** 13:228–230, 2004
99. Sagoh M, Hirose Y, Murakami H, Katayama M, Akaji K, Mayanagi K: Cerebellar infarction with hydrocephalus caused by spontaneous extracranial vertebral artery dissection—case report. **Neurol Med Chir (Tokyo)** 37:538–541, 1997
100. Savitz MH, Katz SS, Nakazawa H: Acute cerebellar infarction producing brainstem compression. **Mt Sinai J Med** 44:299–303, 1977
101. Sayama I, Sakotani AY, Yasui N, Ito Z, Kobayashi T, Nakajima K: [Cerebellar infarction—early prediction to the operative indication of posterior fossa decompression (author's transl).] **No To Shinkei** 33:801–810, 1981 (Jpn)
102. Schmidek HH, Guthikonda M: Acute cerebellar infarction—a potential surgical emergency, in Smith JL (ed): **Neuro-ophthalmology Focus**. New York: Masson Press, 1979, pp 427–432
103. Scotti G, Spinnler H, Sterzi R, Vallar G: Cerebellar softening. **Ann Neurol** 8:133–140, 1980
104. Shenkin HA, Zavala M: Cerebellar strokes: mortality, surgical indications, and results of ventricular drainage. **Lancet** 2:429–432, 1982
105. Shigemori M, Ogata T, Tokutomi T, Yuge T, Watanabe M, Kuramoto S: Massive cerebellar infarction with obstructive hydrocephalus—a case report. **Kurume Med J** 30:81–84, 1983
106. Skurnik Y, Brandiner S, Gandelman G, Shtoege Z: [Cerebellar infarction, clinical presentation, diagnosis and treatment.] **Harefuah** 136:206–210, 1999 (Hebrew)
107. Spinnler H, Sterzi R, Vallar G: [Natural history, treatment and outcome of cerebellar softening (author's transl).] **Riv Patol Nerv Ment** 100:309–328, 1979 (Italian)
108. Struck LK, Biller J, Bruno A, Neiman RF, Yuh WTC, Marsh EE III, et al: Superior cerebellar artery territory infarction. **Cerebrovasc Dis** 1:71–75, 1991
109. Sybert GW, Alvord EC Jr: Cerebellar infarction. A clinicopathological study. **Arch Neurol** 32:357–363, 1975
110. Tada Y, Mizutani T, Nishimura T, Tamura M, Mori N: Acute bilateral cerebellar infarction in the territory of the medial branches of posterior inferior cerebellar arteries. **Stroke** 25:686–688, 1994
111. Taneda M, Ozaki K, Wakayama A, Yagi K, Kaneda H, Irino T: Cerebellar infarction with obstructive hydrocephalus. **J Neurosurg** 57:83–91, 1982
112. Tohgi H, Takahashi S, Chiba K, Hirata Y: Cerebellar infarction. Clinical and neuroimaging analysis in 293 patients. **Stroke** 24:1697–1701, 1993
113. Tomaszek DE, Rosner MJ: Cerebellar infarction: analysis of twenty-one cases. **Surg Neurol** 24:223–226, 1985

Space-occupying cerebellar infarction

114. Tsitsopoulos PP, Tobieson L, Enblad P, Marklund N: Clinical outcome following surgical treatment for bilateral cerebellar infarction. **Acta Neurol Scand** **123**:345–351, 2011
115. Tsitsopoulos PP, Tobieson L, Enblad P, Marklund N: Surgical treatment of patients with unilateral cerebellar infarcts: clinical outcome and prognostic factors. **Acta Neurochir (Wien)** **153**:2075–2083, 2011
116. Tulyapronchote R, Malkoff MD, Selhorst JB, Gomez CR: Treatment of cerebellar infarction by decompression suboccipital craniectomy. **Stroke** **24**:478–480, 1993
117. Turgut M, Ozcan OE, Ertürk O, Sarıbaş O, Erben A: Spontaneous cerebellar strokes. Clinical observations in 60 patients. **Angiology** **47**:841–848, 1996
118. Unterberg A, Juettler E: The role of surgery in ischemic stroke: decompressive surgery. **Curr Opin Crit Care** **13**:175–179, 2007
119. Vereshchagin NV, Chebotareva NM, Kalashnikova LA: [Cerebellar infarct: clinical picture, diagnosis and treatment.] **Zh Nevropatol Psikhiatr Im S S Korsakova** **78**:1609–1614, 1978 (Russian)
120. Wijdicks EFM, Maus TP, Piepgras DG: Cerebellar swelling and massive brain stem distortion: spontaneous resolution documented by MRI. **J Neurol Neurosurg Psychiatry** **65**:400–401, 1998
121. Wood MW, Murphey F: Obstructive hydrocephalus due to infarction of a cerebellar hemisphere. **J Neurosurg** **30**:260–263, 1969
122. Woodhurst WB: Cerebellar infarction—review of recent experiences. **Can J Neurol Sci** **7**:97–99, 1980
123. Yoshimura K, Kubo S, Nagashima M, Hasegawa H, Thminaga S, Yoshimine T: Occlusive hydrocephalus associated with cerebellar infarction treated with endoscopic third ventriculostomy: report of 5 cases. **Minim Invasive Neurosurg** **50**:270–272, 2007

Manuscript submitted October 25, 2012.

Accepted February 20, 2013.

Please include this information when citing this paper: DOI: 10.3171/2013.2.FOCUS12363.

Address correspondence to: Hermann Neugebauer, M.D., Rehabilitation and University Hospital Ulm, Department of Neurology, Oberer Eselsberg 45, 89081 Ulm, Germany. email: hermann.neugebauer@charite.de.