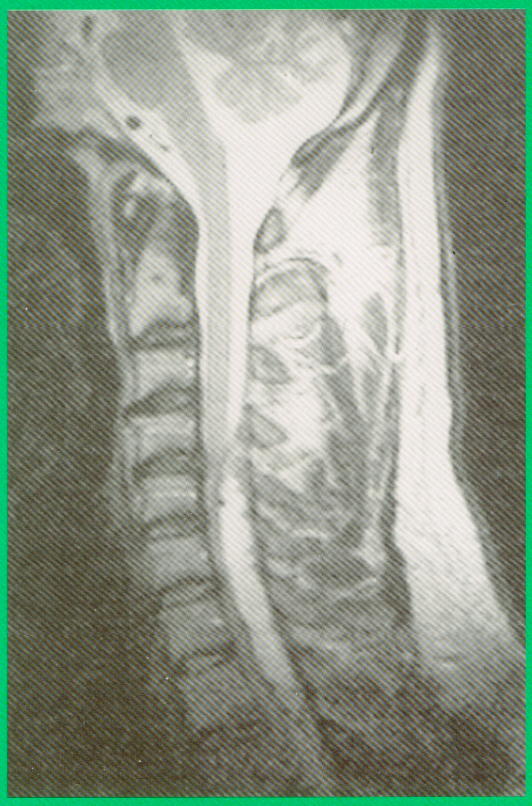
The spontaneous spinal epidural hematoma

Groen, Rob

THE SPONTANEOUS SPINAL EPIDURAL HEMATOMA

A clinical and anatomical study with correlations to the morphology of the internal vertebral venous plexus



R. J. M. GROEN

Stellingen

1. De worstel(l)ing die menig promovendus levert om tot het gewenste aantal stellingen te komen, leidt niet zelden tot een indrukwekkende serie teleurstellingen.

2. De benaming distale spinale spieratrofie (distal spinal muscular atrophy [SMA]) is in strikte zin onjuist, omdat er zowel atrofische als hypertrofische manifestaties bestaan van deze erfelijke aandoening. Het komt het begrip en de logica ten goede als in dit kader de veel minder gebruikte, doch pathofysiologisch juiste, indeling voor hereditaire motor neuropathiëen (HMN) zou worden gehanteerd (J Neurol Sci (1993) 114:81-84).

3. Onbekendheid met de (normale) vasculaire anatomie van de spinale epidurale ruimte lijkt in de literatuur mede te hebben bijgedragen aan onjuiste conclusies over de etiologie van het spontane spinale epidurale hematoom (dit proefschrift).

4. De posterieure interne vertebrale veneuze plexus blijkt, in tegenstelling tot de "klassieke" weergaven, een grote mate van variabiliteit te vertonen, zowel segmentaal als inter-individueel (dit proefschrift).

5. De meerderheid van de spontane spinale epidurale hematomen is het gevolg van een bloeding vanuit de interne vertebrale veneuze plexus (dit proefschrift).

6. De bepalende factoren voor het postoperatieve herstel na een spontane spinale epidurale bloeding zijn: [1] de localisatie van het hematoom, [2] het preoperatieve neurologische beeld en [3] het operatie interval (dit proefschrift).

7. Ook als er op het moment dat de diagnose SSEH wordt gesteld reeds een complete dwarslaesie bestaat, kan een met spoed uitgevoerde decompressie van het ruggenmerg tot een volledig neurologisch herstel leiden (dit proefschrift).

8. In een goed geoutilleerd mortuarium zit verrassend veel leven.

9. De term "Minimaal Invasieve Neurochirurgie" is een fraai hedendaags euphemisme.

10. Een ziekenhuis is een ongezonde werkplek.

11. Opereren leer je door het te doen, niet door er naar te kijken.

12. Beter één interventieradioloog bij de hand, dan tien in de lucht.

Stellingen behorende bij het proefschrift:
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THE SPONTANEOUS SPINAL EPIDURAL HEMATOMA.
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ACADEMISCH PROEFSCHRIFT

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op gezag van de rector magnificus
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in het openbaar te verdedigen
ten overstaan van de promotiecommissie
van de faculteit de geneeskunde
op woensdag 15 oktober 1997 om 13.45 uur
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De Boelelaan 1105

door

Robertus Jacobus Maria Groen

geboren te Groningen

Promotoren: Prof. dr. H.A.M. van Alphen
Prof. dr. H.J. Groenewegen

Copromotor: Dr. P.V.J.M. Hoogland

Contents.

Chapter 1

INTRODUCTION.

1.1 Background of the study.

Among the variety of space occupying lesions within the spinal canal, the spontaneous (or idiopathic) spinal epidural hematoma (SSEH) is a very distinct and intriguing disorder. Jackson, in 1869, was the first who reported a patient suffering a SSEH (22). This 14-year-old girl experienced a painless progressive paresis of the arms and respiratory muscles. She died within 4 days after the onset of the symptoms, due to respiratory insufficiency. Autopsy revealed a large epidural hematoma, extending over the cervical portion of the spine, in particular anteriorly and to the left side. No cause of the hemorrhage was identified (22). After this publication, many cases have been described in the international medical literature, contributing to a large series that, at present, comprises approximately 380 patients.

Despite this multitude of case reports, little is known about the clinical features, the clinical course and the etiology of the SSEH. This lack of knowledge most probably can be explained by the fact that SSEHs are rare. The number of cases that have been treated within one single institution is very small, which has prevented authors from drawing major conclusions about etiology, diagnosis and treatment of SSEH. As a consequence, the theories about the etiology of the SSEH are diverse. The rupture of spinal epidural arteries (6,9), veins (5,13,18,19,23,36), (cryptic) angiomas (35) and arteriovenous malformations (5,10,12-14,17,23,24,28-32,38,41) are mentioned in the literature as a possible source of spontaneous spinal epidural bleeding. Others have suggested the role of pregnancy (7,40,44), arterial hypertension (1,28), atherosclerosis (1) and coagulation disorders (anticoagulants, [hematologic] diseases) (2,3,8,9,15,16,20,27,39,42,43) in the pathogenesis of SSEH. Likewise, mechanical factors (26) and conditions that are assumed to produce an acute increase of pressure in the spinal canal (lifting (11,18,21,25,34), sneezing (33,37), voiding (4) and vomiting (23)) have been mentioned as causative factors. None of these theories has been supported by histopathological, anatomical and/or statistical evidence.

At present, the number of case-reports in the international medical literature still increases. This renewed interest may be related to the introduction of Magnetic Resonance Imaging (MRI), which enables an early and precise definition of the lesion. Due to MRI, more cases with a benign natural course are being detected. However, the main reasons for the clinician to report on this potentially curable disease appear to be the dramatic clinical course of this entity, and the unresolved discussions about its etiology.

1.2 Aim of the thesis.

The aim of this thesis is to describe the clinical features, to determine the factors that are related to the postoperative outcome and to try to elucidate some of the etiological factors in the spontaneous spinal epidural hematoma. The initial assumption was that a rupture of a vessel of the internal vertebral venous plexus is the major cause of SSEH. To substantiate this, we conducted a literature search, paying special attention to the segmental

distribution of the SSEH within the spinal canal and to the vascular anatomy of the spinal epidural space. In addition, the anatomy of the internal vertebral venous plexus was studied after injection of a modern polymere into the vertebral venous system of ten human cadavers.

1.3 Outline of the thesis.

In *Chapter 2*, three patients are presented to illustrate the clinical picture, the diagnostic procedures, the therapeutic approaches and the postoperative course of SSEH. The importance of early diagnosis and undelayed treatment (if necessary) are stressed and the different theories about the etiology are mentioned briefly.

In *Chapters 3 and 4*, a large series of cases that are collected from the international medical literature is described. *Chapter 3* comprises a study of the factors that determine postoperative outcome after SSEH. Based on the data from 333 patients, the prognostic value of sex, age, neurological condition, position of the hematoma, vertebral level and extent of the hematoma and the operative interval is studied. The correlation of these factors with the postoperative recovery are discussed. In *Chapter 4*, the literature data are related to the different theories that have emerged about the etiology of SSEHs. Since (histo)pathological evidence about the source of SSEHs is lacking, and suggestions about the causal relation of (normal) spinal epidural vascular structures with SSEHs have not been supported by anatomical evidence, the literature on the vascular anatomy of the spinal epidural space is reviewed in *Chapter 5*. From these data, a correlation between the SSEH and the morphological aspects of the internal vertebral venous plexus is suspected.

However, details about the segmental and (inter)individual variability of the internal vertebral venous plexus are not available in the literature. Therefore, a human cadaver study was performed, as to find morphological support for the "venous theory" of the etiology of the SSEH. The technical details and the results of this study are presented in *Chapter 6*.

In *Chapter 7*, the results of the anatomical and the literature reviews are discussed. A synthesis of the findings from the previous study is made, focussing on the etiology and pathophysiology of the SSEH.

1.4 References.

1. Ainslie J.P. Paraplegia due to spontaneous extradural or subdural hemorrhage. Br J Surg (1958) 45:565-567.
2. Alderman D.B. Extradural spinal-cord hematoma: report of a case due to dicoumarol and review of the literature. N Engl J Med (1956) 255:839-842.
3. Amyes E.W., Vogel P.J. and Raney R.B. Spinal cord compression due to spontaneous epidural hemorrhage: report of three cases. Bull Los Angeles Neurol Soc (1955) 20:1-8.
4. Bain W. A case of haematorrachis. Br Med J (1897) 2:455.

5. Banerjee T. and Meagher J.N. Spontaneous spinal epidural hemorrhage: case report. South Med J (1974) 67:858-861.
6. Beatty R.M. and Winston K.R. Spontaneous cervical epidural hematoma. A consideration of etiology. J Neurosurg (1984) 61:143-148.
7. Bidzinski J. Spontaneous spinal epidural hematoma during pregnancy: case report. J Neurosurg (1966) 24:1017.
8. Busse O., Hamer J., Paal G. and Piotrowski W. Spontane epidurale spinale Hämatome wahrend und nach Antikoagulantienmedication. Nervenarzt (1972) 43:318-322.
9. Calliauw L., Dhara M., Martens F. and Vannerem L. Spinal epidural hematoma without lesion of the spine. Report of 4 cases. Clin Neurol Neurosurg (1988) 90(2):131-136.
10. Combelles G., Blond S., Lesion F., Bousquet C., Rousseaux M. and Christiaens J.L. Hématomes extra-dureaux rachidiens sans lésion osseuse traumatique. A propos de 9 cas. Neurochirurgie (1983) 29:417-422.
11. Correa A.V. and Breasley B.A.L. Spontaneous cervical epidural hematoma with complete recovery. Surg Neurol (1978) 10:227-228.
12. Cube H.M. Spinal extradural hemorrhage. J Neurosurg (1962) 19:171-172.
13. Dauch W.A. Spinale Epidural-hämatome bei Kindern und Jugendlichen. Neurochirurgia (1986) 29:83-89.
14. Dawson B.H. Paraplegia due to spinal epidural hematoma. J Neurol Neurosurg Psychiat (1963) 26:171-173.
15. Devadiga K.V. and Gass H.H. Chronic lumbar extradural hematoma simulating disc syndrome. J Neurol Neurosurg Psychiat (1973) 36:255-259.
16. Fischbach von R. and Kollar W.A. Spontane spinale epidurale Blutung unter Antikoagulantion. Wien Med Woch (1972) 19/20:5-6.
17. Foo D., Chang Y.C. and Rossier A.B. Spontaneous spinal epidural hemorrhage, anterior cord syndrome, and familial vascular malformation: case report. Neurology (NY) (1980) 30:308-311.
18. Ghanem M.B. and Ivan L.P. Spontaneous spinal epidural hematome in an eight year old boy. Neurology (Minneap) (1978) 28:829-832.
19. Gold M.E. Spontaneous spinal epidural hematome. Radiology (1963) 80:823-828.
20. Goulon M., Nouailhat F., Julian H., Barois A., Gajdos P., Babinet P. and Rapin M. Hématome extradurale cervicale spontané, responsable d'une hémiplégie spinale: guérison après intervention. Soc Med Hop Paris (1967) 118:969-979.
21. Herrmann E., Lorenz R. and Vogelsang H. Zur diagnostik der spinalen epiduralen Hämatome und Abszesse. Radiologe (1965) 5:504-508.
22. Jackson R. Case of spinal apoplexy. Lancet (1869) 2:5-6.
23. Kaplan L.I. and Denker P.G. Acute non-traumatic spinal epidural hemorrhage. Am J Surg (1949) 78:356-361.
24. Koyama T., Igarashi S., Hanakita J. and Handa J. Das spinale epidurale Hämatom. Neurochirurgia (1982) 25:11-13.
25. Lepoire J., Tridon P., Montaut J. and Germain F. L'hématome extra dural rachidien spontané. Neurochirurgie (1959) 7:298-313.

26. Liebeskind A.L., Schwartz K.S., Coffey E.L. and Beresford H.R. Spinal epidural hematoma with delayed appearance of neurological symptoms. Neuroradiology (1975) 8:191-193.
27. London G.W., McKeever P.E. and Wiederholt W.C. Spontaneous spinal epidural hematoma in alcoholism. Ann Int Med (1974) 81(2):266-267.
28. Lougheed W.M. and Hoffman H.J. Spontaneous spinal extradural hematoma. Neurology (1960) 10:1059-1063.
29. Maxwell G.M. and Puletti F. Chronic spinal epidural hematoma in a child: case report. Neurology (1957) 7:597-600.
30. Müller H., Schramm J., Roggendorf W. and Brock M. Vascular malformations as a cause of spontaneous spinal epidural hematoma. Acta Neurochir (Wien) (1982) 62:-297-305.
31. Nichols P. and Manganiello L.O.J. Extradural hematome of the spinal cord. Case reports. J Neurosurg (1956) 13:638-640.
32. Packer N. and Cummins B.H. Spontaneous epidural hemorrhage: a surgical emergency. Lancet (1978) 18:356-358.
33. Pear B.L. Spinal epidural hematoma. Am J Roentgenol Radium Ther Nucl Med (1972) 115:155-164.
34. Rao B.D., Rao K.S., Subrahmanian M.V. and Raghava Reddy M.V. Spinal epidural hemorrhage. Br J Surg (1966) 53:649-650.
35. Robertson W.C. Jr., Lee Y.N. and Edmonson M.B. Spontaneous spinal epidural hematoma in the young. Neurology (1979) 29:120-122.
36. Schicke R. and Seitz D. Spinales epidurales Hämatom unter Antikoagulantien-therapie. Dtsche Med Wochenschr (1970) 6:275-277.
37. Scott B.B., Quisling R.G., Miller C.A. and Kindt G.W. Spinal epidural hematoma. JAMA (1976) 235:513-515.
38. Solero C.L., Fornari M. and Savoiardo M. Spontaneous spinal epidural hematoma arising from ruptured vascular malformation. Case report. Acta Neurochir (Wien) (1980) 53:169-174.
39. Stanley P. and McComb J.G. Chronic spinale epidural hematoma in hemophilia A in a child. Pediatr Radiol (1983) 13:241-243.
40. Suzuki N., Inoue H. and Abematsu N. Acute spontaneous spinal epidural he-morrhage in pregnancy. Nippon Seikeigeka Gakkai Zasshi (1977) 51(3):155-160.
41. Tsai F.Y., Popp A.J. and Waldman J. Spontaneous spinal epidural hematoma. Neuroradiology (1975) 10:15-30.
42. Weigert M. Akutes spinales epidurales Hämatom als Folge von Behandlung mit Antikoagulantien. Nervenarzt (1961) 32:85-89.
43. Winer B.M., Horenstein S. and Starr A.M. Spinal epidural hematoma during anticoagulant therapy. Circulation (1959) 19:735-740.
44. Yonekawa Y., Mehdorn M. and Nishikawa M. Spontaneous spinal epidural hematoma during pregnancy. Surg Neurol (1975) 3:327-328.

Chapter 2

THE SPONTANEOUS SPINAL EPIDURAL HEMATOMA.
Report of three cases.

2.1 Introduction.

Three patients with SSEH are presented, in order to illustrate the clinical picture, the diagnostic procedures, possible therapeutic approaches and the postoperative course of this neurosurgical emergency.

2.2 Case reports.

Case 1. A 29-year-old man was admitted with the clinical picture of a rapidly evolving spinal cord compression. His medical history was uneventful. He worked as a commercial manager. One week before admission he had a minor accident as he slipped over the footway. At 04.00 in the morning of admission he woke up because of severe interscapular pain, which was accompanied by twinkling sensations in the abdomen and both legs. Within fifteen minutes an almost complete paralysis of both legs occurred, starting on the right side.

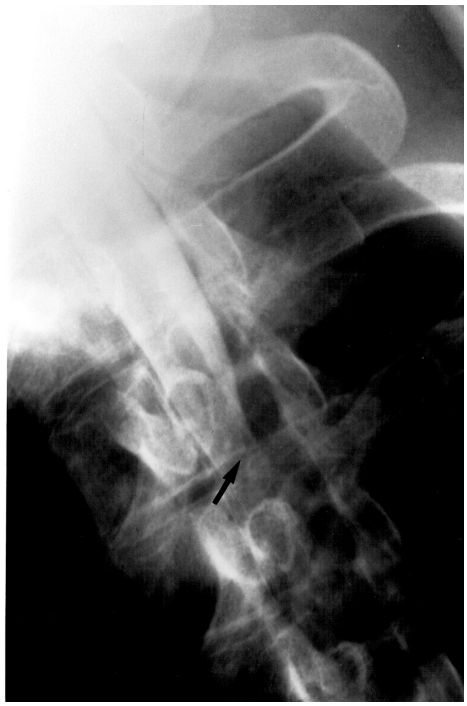
Examination. At the moment of arrival in the hospital, we noticed a severe syndrome of myelum compression with almost complete paraparesis, hypaesthesia and hypalgesia below the nipples, increased tendon reflexes in the legs, extension response of the plantar reflexes, absent abdominal reflexes and urinary retention. General physical examination was normal. Arterial blood-pressure was 120/80 mm Hg. Hematologic analysis ruled out bleeding diathesis. No abnormalities of CSF, blood and urine were detected.

Imaging studies. On myelography, an incomplete block was demonstrated with an upper limit at the level T3 (*Figure 2.1*). The computerized tomography demonstrated an epidural compressive lesion, suggestive for a spinal epidural hematoma extending from T3 to T5.

Operation. Eighteen hours after the onset of the symptoms, laminectomy was performed. As soon as the yellow ligament was opened, an epidural blood clot spontaneously evacuated under remarkable pressure. After removal of the residual hematoma, normal dural pulsations returned. Some profusely bleeding epidural veins were coagulated. Careful exploration with three times magnification could not reveal any abnormal vascular structure. There was no material for histological examination.

Postoperative course. Immediately after the operation a marked improvement of the motor functions was noticed with subsequent amelioration of the sensory functions. Two weeks after the operation the patient was able to walk up and down stairs. Within four months he returned to his job. There were only complaints of some unsteady feelings while running. Neurological examination revealed normal motor function of both legs. Sensory examination seven months after the operation showed an improvement of all modalities. The reflexes in both legs were still elevated, with positive Babinski-signs. The abdominal skin reflexes were present in all segments.

Figure 2.1 Myelography after suboccipital puncture, showing a complete contrast block at T3 in case 1.



Case 2. A 33-year-old Japanese business-man was in generally good health, except for two short episodes of sharp interscapular pain, two years and three weeks before admission, respectively. Since two days he complained of spontaneous, sharp, intermittent, inter-scapular pain, irradiating to the right shoulder. A friend decided to give a massage, by rubbing the painfull area. Within twenty minutes after this procedure, a complete paraplegia developed.

Examination. After presentation in the hospital, a complete paraplegia, priapism, urinary retention and a ptosis and myosis of the right eye were found, with a decreased superficial sensibility below the dermatome T2, absent abdominal skin reflexes and normal knee-, ankle- and plantar-reflexes. This clinical picture was suggestive for spinal cord compression at the level T1 on the right side. General physical examination and arterial blood pressure (110/70 mm Hg) were normal. Blood analysis ruled out hemorrhagic disorders.
Imaging studies. Cervical myelography demonstrated a complete block at the level C7-T1, indicative for an extradural space occupying process. Subsequent lumbar myelographyrevealed the lower limit of this process at T3. CT-scans did confirm these findings (*Figure 2.2 and 2.3*).

Figure 2.2 Computerized tomography scan at T3 after myelography, showing a posterior extradural mass with compression of the spinal cord in case 2.

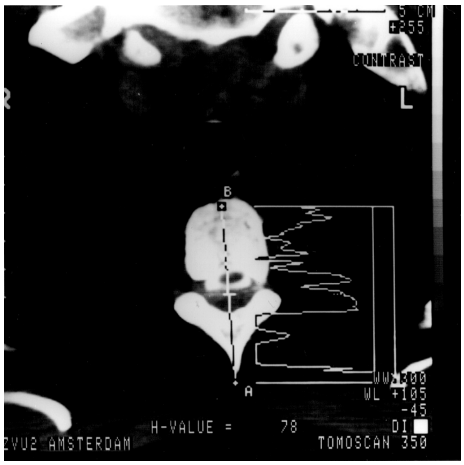


Figure 2.3 Sagittal reconstruction of the CT scan after caudography and myelography, showing a concave lesion extending from T3 to T4, compatible with a spinal epidural hematoma in case 2.

Operation. Nine hours after the onset of the paresis, a right hemilaminectomy at T1 and T2 was performed. Immediately after incision of the yellow ligament, an epidural blood clot spontaneously evacuated and normal dural pulsations returned. No bleeding cause was found. Some epidural vascular structures where removed. Histological examination revealed these to be normal venous structures.

Postoperative course. Improvement of the patient's neurological function was noted immediately. Within two weeks after operation this patient was discharged from the department, without any neurological deficit.

Case 3. A 74-year-old women with a history of arterial hypertension, diabetes mellitus, gout, severe symptomatic atherosclerosis and coagulopathy due to the use of oral anti-coagulants (Sintromitis) was presented. Two days before admission she suffered acute lower backpain, irradiating to the abdomen and the inguinal regions. Progressive numbness was

noticed in both legs, but the patient decided to go to sleep. The next morning she experienced progressive weakness in the lower extremities and appeared to have lost control of bowel and bladder function. Twenty four hours later the patient agreed with transferal to the hospital for further investigation and treatment.

Examination. The lower extremities were plegic and areflexic, and bilateral Babinski signs were elicted. Anal sphincter tone was absent. Allmost complete loss of sensation was noted caudal to the level of the T7 dermatoma. General examination revealed obesitas and arterial hypertension (170/90 mm Hg). Hematologic analysis was normal. Anticoagulant therapy was stopped and coagulopathy had been corrected before admission to the neurosurgical department.

Imaging studies. Plain radiographs of the thoracolumbar spine were unremarkable. Magnetic resonance imaging (MRI) of the thoracic spine demonstrated an isointense concave epidural compressive lesion on T_1 weighted imaging (T_1WI), extending over T5 through T10 (*Figure 2.4*). Severe deformation of the spinal cord was present. T_2 weighted images (T_2WI) of the same area showed a hyperintense epidural lesion, indicative of a subacute hematoma (*Figure 2.5*).

Operation. Emergency laminectomy of T5 down to and including T10 was performed, revealing a solid hematoma in the epidural space. This was gently removed with forceps and by irrigation and aspiration. No vascular anomaly was found during exploration, and histopathological examination of the specimen revealed only a hematoma.

Postoperative course. Postoperatively, the neurological condition slowly improved. She was transfered to a rehabilitation centre. Six months after operation this patient returned home, being allmost independant regarding her activities of daily living. Lower-extremity paresis had improved gradually, enabling mobilization with a playpen. Hypesthesia persisted below the T11 dermatoma. Spontaneous micturition had returned, although intermittent catheterization (twice a day) remained necessary.

Figure 2.4 Sagittal MR view, T_1 - weighted sequence, case 3. A longitudinal line with weak signal, representing the dura, separates the blood clot (arrows) from the spinal cord. The clot is situated dorsal to the dura and has an isointensive signal compared with the cord.

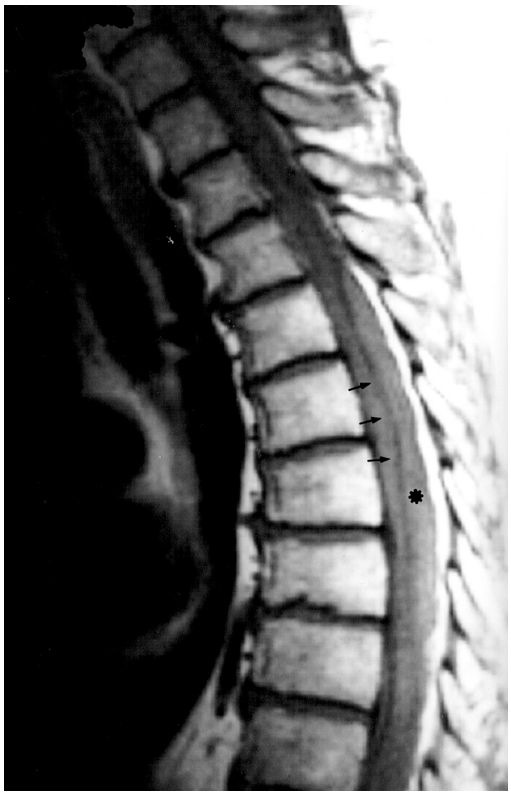
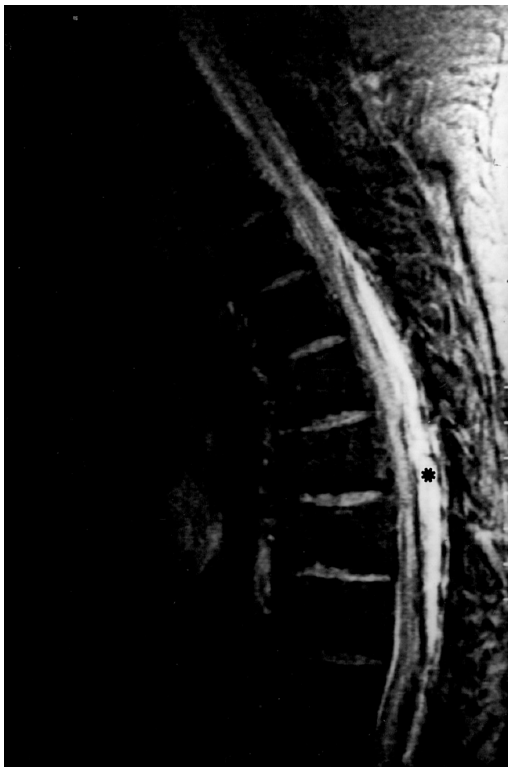


Figure 2.5 Sagittal MR view, T_2 - weighted sequence of the same area. Hyperintense, almost homogeneous epidural lesion, indicative of subacute hemorrhage.



2.3 Discussion.

The spontaneous or idiopathic spinal epidural hematoma is an acute condition. It has been described originally by Jackson in 1869 (18). Up till now, approximately 380 cases of SSEH have been described in the international medical literature. Because of its rarity, many clinicians are unfamiliar with this entity. Although some cases of (chronic) spinal epidural hematomas with slowly progressive neurological symptoms have been described (8,15,30), the classical picture is that of a rapidly progressive spinal cord compression, preceded by acute, severe, local or irradiating spinal pain (7). An increasing number of cases with spontaneous recovery have been described (2,5,6,10,11,14,16,17,20,21,23,27,33-35,37). However, the majority of patients will need operative treatment without delay to reverse the effects of neural compression (7,9,24-26,28). In the past, myelography and computerized tomography have been the diagnostic methods of choice. However, myelography is an invasive and non-specific technique (showing only the presence of an epidural mass with a complete or partial block) and localization of the lesion may be inaccurate. CT imaging (eventually in combination with myelography) can be more specific than myelography if a hyperdense biconvex mass is demonstrated adjacent to the bony spinal canal, but especially in the thoracic area's interpretation of the scans can be very difficult (3). Nowadays, magnetic resonance imaging of the spine has almost replaced CT/myelography. It provides a rapid, noninvasive, accurate method to characterize the epidural lesion as a hematoma, and to delimit its configuration, extent, and effect on the spinal cord and cauda equina (32). T_1 and T_2 signals of SSEH vary, based on the clot-age, size and oxygenation (19). Within the first hours after hemorrhage, T_1WI of the hematoma is isointense with the spinal cord and a heterogeneous signal intensity will be observed in T_2WI. In a later stage, the T_2WI of the hematoma becomes hyperintense and more homogeneous (3), which is highly suggestive for a subacute hematoma. Since the introduction of MRI, the number of reported cases that recover spontaneously is rising. This might be explained by the fact that MRI has greatly facilitated the diagnosis of SSEH, allowing for more cases with a benign natural course to be detected preoperatively. It seems very likely that such cases have often escaped clinical diagnosis in the past, because of the relative paucity of their symptoms and signs, or the very early onset of recovery (19). Consequently, the traditional unreserved pleading for urgent neurosurgical decompression in SSEH may not be justifiable in all cases.

Regarding the etiology of the SSEH, a number of possible factors have been mentioned: bleeding diathesis, hypertension, anticoagulant therapy, atherosclerosis and pregnancy (7). The concept of a minor trauma causing disruption of venous or arterial epidural structures is often referred to (1,4). Physical exertion and other activities that result in an increase of the spinal epidural pressure (like sneezing, coughing, voiding and vomiting) are thought to play an important causal role (7). Nevertheless, several cases of spinal epidural bleeding did develop during sleep (8,18,35). Another subject of continuous discussion is the bleeding source. Ruptures of epidural vascular malformations (12,22), vertebral angiomas (36), cryptic angiomas (31), epidural arteries (4) and the spinal epidural venous plexus (7) are mentioned. Actually, none of these theories has been supported by anatomical and/or histopathological evidence. In most cases, histopathological examination

of the epidural mass revealed only an (organized) hematoma. Few authors advocated the performance of an extensive selective spinal angiography to establish a vascular anomaly in cases in which operative findings were negative (22,36). The controversy of this procedure is illustrated by Ter Spill and Tijssen (36). They reported a vertebro-epidural hemangioma at T6 in a patient who was operated on a spontaneous thoracic epidural hematoma that extended from T8 to T10. The causal relationship between the hemangioma and the spinal epidural hemorrhage is debatable, because of the different location of both lesions in this patient.

Postoperative outcome after SSEH is studied by Foo and Rossier (13). Neurological recovery appeared to vary with the severity of the sensorimotor impairment prior to surgical intervention. However, the absence of sensorimotor functions before operation did not necessarily indicate a poor prognosis, since partial or complete neurological recovery was noted in 45% of these patients. Their study did not indicate a correlation between the duration of neurological deficit before operation (operative interval) and postoperative outcome. On the contrary, McQuarri (26) noticed that the probability of recovery fell below 50%, when operative interval exceeded approximately 36h. All three cases presented above, suffered an allmost complete spinal cord lesion before operation. Laminectomy and evacuation of the hematoma resulted in significant neurological improvement in all patients. Operative interval was 8h in case 2 (complete recovery), 16h in case 1 (allmost complete recovery) and 48 h in case 3 (incomplete recovery), which suggests a correlation between the operative interval and postoperative outcome.

Although SSEH is a rare cause of spinal cord compression, it is essential that the diagnosis is established as early as possible. If a spinal epidural hematoma is suspected, MRI should be performed without delay. The differential diagnosis of SSEH includes acute herniated vertebral disc, vertebral tumor, epidural tumor, spinal subarachnoid hemorrhage, spinal subdural hematoma, epidural abcess, spondylitis, transverse myelitis and aortic dissection. In all these cases, MRI is the diagnostic procedure of choice. After MRI has confirmed the suspicion of SSEH, most patients will need instantaneous operation for spinal cord and/or cauda equina decompression. Conservative treatment should only be reserved for patients with minimal neurological deficit. Obviously, those patients need close monitoring. Disturbances in hemostatis caused by thrombolytic and anticoagulant agents, or due to internal diseases (hemophilia, liver disease, etc.), must be corrected to prevent further hemorrhage, irrespective of whether operation of conservative treatment will be instituted.

2.4 References.

1. Ainslie J.P. Paraplegia due to spontaneous extradural or subdural hemorrhage. Br J Surg (1958) 45:565-567.
2. Anderson T.J. and Donaldson I. MacG. Spontaneous resolution of cervical spinal epidural haematoma. Postgrad Med J (1989) 65:488-490.

3. Avrahami E, Tadmor R., Ram Z., Feibel M. and Itzhak Y. MR demonstration of spontaneous acute epidural hematoma of the thoracic spine. Case reports. Neuroradiology (1989) 31:89-92.
4. Beatty R.M. and Winston K.R. Spontaneous cervical epidural hematoma. A consideration of etiology. J Neurosurg (1984) 61:143-148.
5. Bernsen P.L.J.A., Haan J., Vielvoye G.J. and Peelinck K.M.J. Spinal epidural hematoma visualized by magnetic resonance imaging. Neuroradiology (1988) 30:280.
6. Brawn L.A., Bergval U.E.G. and Davies-Jones G.A.B. Spontaneous spinal epidural haematoma with spontaneous resolution. Postgrad Med J (1986) 62:885-887.
7. Bruyn G.W. and Bosma N.J. Spinal epidural hematoma, in Vinken en Bruyn,eds. Handbook of clinical neurology (1976). Vol.26. Amsterdam: North-Holland publishing company. pp. 1-30.
8. Chavany J.A., Taptas J.N. and Pecker J. A propos d'une varieté exceptionelle d'hematorachis: l'hematome epidurale spontané. Presse Med (1949) 62:869-871.
9. Chen H.-H. and Hung C.-C. Spontaneous spinal epidural hematoma: report of seven cases. J Formosan Med Assoc (1992) 91:214-218.
10. Clarke D.B, Bertrand G. and Tampieri D. Spontaneous sinal epidural hematoma causing paraplegia: resolution and recovery without surgical decompression. Neurosurgery (1992) 30:108-111.
11. Crabbe D.C.G., Mendelow A.D. and Pharoh P. Cervical spinal extradural haematoma causing a transient Brown-Sequard syndrome. J Neurol Neurosurg Psych (1992) 55:239.
12. Cube H.M. Spinal extradural hemorrhage. J Neurosurg (1962) 19:171-172.
13. Foo D. and Rossier A.B. Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas. Surg Neurol (1981) 15:398-401.
14. Futuwatari K, Matsuoka S. and Kowada M. A case of spinal epidural hematoma associated with idiopathic thrombocytopenic purpura. No Shinkei Geka (1991) 19:1187-1190.
15. Hehman K. and Norrel H. Massive chronic spinal epidural hematoma in a child. Am J Dis Child (1968) 116:308-310.
16. Hernandez D., Viñuela F. and Feasby T.E. Recurrent paraplegia with total recovery from spontaneous spinal hematoma. Ann Neurol (1982) 11:623-624.
17. Imamura T. and Tsuburaya K. Chrinic cervical epidural hematoma diagnosed by magnetic resonance imaging. No to Shinkei (1990) 42:857-861.
18. Jackson R. Case of spinal apoplexy. Lancet (1869) 2:5-6.
19. Jamjoom Z.A.B. Acute spontaneous spinal epidural hematoma: the influence of magnetic resonance imaging on diagnosis and treatment. Surg Neurol (1996) 46:345-349.
20. Kato S., Seki H. and Koshu K. Acute cervical spinal epidrual hematoma with spontaneous resolution. Case report. Neurol Med Chir (Tokyo) (1994) 34:23-26.
21. Kingery W.S., Seibel M., Date E.S. and Marks M. The natural resolution of a lumbar spontaneous epidural hematoma and associated radiculopathy. Spine (1994) 19:67-69.

22. Koyama T., Igarashi S., Hanakita J. and Handa J. Das spinale epidurale Hämatom. Neurochirurgia (1982) 25:11-13.
23. Laissy J.-P., Milon P., Freger P., Hattab N., Creissard P. and Thiebot J. Cervical epidural hematomas: CT diagnosis in two cases that recovered spontaneously. AJNR (1990) 11:394-396.
24. Lord G.M. and Mendoza N. Spontaneous spinal epidural haematoma: a cautionary tale. Arch Emerg Med (1993) 10:339-342.
25. Matsumae M., Shimoda M., Shibuya N., Ueda M., Yamamoto I. and Sato O. Spontaneous cervical epidural hematoma. Surg Neurol (1987) 28:381-384.
26. McQuarrie I.G. Recovery from paraplegia caused by spontaneous spinal epidural hematoma. Neurology (1978) 28:224-228.
27. Narawong D., Gibbons V.P., McLaughlin J.R., Bouhasin J.D. and Kotagal S. Conservative management of spinal epidural hematoma in hemophilia. Ped Neurol (1989) 4:169-171.
28. Packer N. and Cummins B.H. Spontaneous epidural hemorrhage: a surgical emergency. Lancet (1978) 18:356-358.
29. Pan G., Kulkarni M., MacDougall D. and Miner M.E. Traumatic epidural hematoma of the cervical spine: diagnosis with magnetic resonance imaging. Case report. J Neurosurg (1988) 68:798-801.
30. Pear B.L. Spinal epidural hematoma. Am J Roentgenol 1972; 115:155-64.
31. Robertson W.C. Jr., Lee Y.N. and Edmonson M.B. Spontaneous spinal epidural hematoma in the young. Neurology (1979) 29:120-122.
32. Rothfus W.E., Chidid M.K., Deed Z.L., Abla A.A., Maroon J.C. and Sherman R.L. MR imaging in the diagnosis of spontaneous spinal epidural hematomas. J Comput Assist Tomogr (1987) 11:851-854.
33. Saito S., Katsube H. and Kobayashi Y. Spinal epidural hematoma with spontaneous recovery demonstrated by magnetic resonance imaging. Spine (1994) 19:483-486.
34. Sheikh A.A. and Abildgaard C.F. Medical management of extensive spinal epidural hematoma in a child with factor lX deficiency. Ped Emerg Care (1994) 10:26-29.
35. Shenkin H.A., Horn R.C. and Grant F.C. Lesions of the spinal epidural space producing cord compression. Arch Surg (1945) 51:125-146.
36. Spill ter H.W. and Tijssen C.C. Spinal epidural hematoma due to a vertebro-epidural hemangioma. Clin Neurol Neurosurg (1989) 91 N°1:91-93.
37. Yoneyama Y., Fukuda M., Itoh K., Yarita H. and Murata K. A case of acute spinal epidural haematoma with spontaneous resolution and its MRI. Clin Neurol (Jap) (1989) 29:754-757.

Chapter 3

OPERATIVE TREATMENT OF SPONTANEOUS SPINAL EPIDURAL HEMATOMAS.
A study of the factors determining postoperative outcome

3.1 Introduction.

The spontaneous spinal epidural hematoma (SSEH) (also referred to as idiopathic spinal epidural hematoma) is a rarely occurring condition. The classical clinical picture is that of acute onset of severe, often radiating, back pain followed by signs and symptoms of a rapidly evolving nerve root and/or spinal cord compression. Spontaneous recovery after SSEH has been reported (80), although this remains exceptional. In most cases, prompt surgery is necessary. Several studies have been performed on operative treatment of spinal epidural hematomas. McQuarrie (111) evaluated 35 patients with SSEH for factors favoring recovery after surgery. Foo and Rossier (46) studied 158 patients to predict the postoperative outcome after spinal epidural hemorrhage, including SSEHs after lumbar puncture, epidural anesthesia or trauma and vertebral neoplasm. Von Klossek and Hüller (190) also studied a large number of SSEHs; their results, however, are difficult to interpret because of incomplete documentation of the cases. Recently, Lawton et al. (85) reported on their experience with surgical management of 30 spinal epidural hematomas, but only five of those cases fit into the definition of SSEH. To further clarify the factors affecting postoperative outcome in SSEH, in the present study an extensive literature rearch is reviewed.

3.2 Patients and methods.

From the literature, the data of 333 cases of SSEH were collected, that were reported adequately and confirmed by surgery or autopsy (*Table 3.1*). Hematomas occurring after trauma with spinal dislocation or fractures, or after epidural anesthesia, diagnostic lumbar puncture, or surgery, were excluded. Spinal epidural hematomas in association with spinal subdural and/or subarachnoid hemorrhage, or combinations with tumors in the spinal canal, were also left out of the study.

The results of surgical treatment were studied with regard to age, preoperative neurological condition, position of the hematoma, the vertebral level and extent of the hematoma, and the operative interval (the time between onset of the neurological dysfunction and surgery). In the majority of patients with complete preoperative sensorimotor loss, we were able to also study the timespan from onset of the initial symptom (which was always pain in this group) to complete sensorimotor loss (compression speed). Unfortunately, those data were missing in most reports of patients with incomplete preoperative neurological deficit.

In most reports, the follow-up interval was not specified and/or accurate description of the degree of sensorimotor recovery was lacking. Consequently, it was decided to list the neurological postoperative condition into three categories: (1) unchanged, (2) incomplete sensory and/or motor recovery and (3) complete sensorimotor recovery. Grouped variables were analyzed statistically using the Chi-square test. The level of significance for rejecting the null hypothesis was fixed at $P < 0.05$.

Table 3.1 Summary of 333 cases of SSEH[a].

Author	Year	Sex	Age	History	Level	Position	Neurology	Interval	Recovery	Histopathology
Ainslie	1958	F	73	Ht	T8-T10	post	SM compl	36 h	unch/died	unknown
		M	70	Ht	C7	post	SM compl	--	died	hematoma
		F	63	Ht	C2-C4	postlat	SM incompl	6 d	unch/died	unknown
Alderman	1956	M	57	Ht,Ac	L1-L3	post	SM incompl	6 d	M incompl	unknown
Amyes	1955	M	57		D11-L4	post	SM incompl	6 d	SM compl	unknown
Amyot	1969	M	1,8		C5-C7	postlat	SM incompl	4 w	M incompl	unknown
Aring	1952	M	62	Ht,Ac	L3-L5	postlat	SM incompl	12 d	SM incompl	hematoma
Avrahami	1989	F	56		T8-T10	post	McompSincomp	unknown	SM compl	unknown
Bain	1897	F	18		C2-C3	postlat	SM compl	--	died	hematoma
Banerjee	1974	M	34		C7-T1	post	SM incompl	24 h	SM compl	hematoma
		M	22		T7-T9	post	SM incompl	24 h	SM compl	hematoma
Bareño	1987	M	46	Ht	C3-C6	postlat	SM incompl	36 h	SM compl	hematoma
Beatty	1984	F	65		C2-C7	postlat	SM incompl	unknown	SM incompl	hematoma
		M	37	SLE	C3-C7	postlat	SM incompl	48 h	SM incompl	hematoma
Bidzinski	1966	F	26	Pregnant	T2-T5	post	SM compl	31 h	SM incompl	hematoma
Binnert	1971	F	28	Pregnant	C6-T4	post	SM compl	5 d	SM incompl	hematoma
Bollar	1988	M	56		T9-T12	post	SM compl	24 h	unknown	hematoma
Bruyn	1976	M	50	Ac	L2-L5	circ	SM compl	unknown	SM incompl	unknown
		M	69	CML	C6-T5	postlat	SM compl	18 h	unchanged	unknown
		M	60	Moschc.	T11-L3	post	SM compl	unknown	SM incompl	unknown
		F	55	Ac	L3-S1	post	SM incompl	unknown	unknown	unknown
		F	57		T2-T4	post	SM compl	unknown	SM incompl	unknown
Busse	1972	F	57	Ac	T10-T12	postlat	SM compl	9 d	SM incompl	hematoma
		M	48	Ac	T9-L1	postlat	SM incompl	48 h	SM incompl	unknown
Caldarelli	1994	F	2		C5-T4	post	SM compl	24 h	SM compl	hematoma
		M	1,3		C4-C7	post	SM incompl	48 h	SM compl	hematoma
Calliauw	1988	M	13		C7-T9	ant	S incompl	unknown	SM compl	hematoma
		M	20		C7-T3	post	SM incompl	4 w	SM incompl	hematoma
		M	39	Hemoph.	T4-T8	post	SM incompl	48 h	SM incompl	unknown
		M	63		T12-L3	post	SM incompl	8 d	SM incompl	hematoma
Carrea	1954	F	4		T5-T10	post	SM incompl	9 d	SM compl	hematoma
Carroll	1981	F	72	Ht,Ac,DM	C3-C6	post	SincompMcomp	24 h	SM compl	unknown
Cecotto	1961	F	16		T10-T12	postlat	SM compl	unknown	ScompMincomp	unknown
Chavany	1949	F	70		L1-L4	post	SM compl	3 w	SM incompl	unknown
Chen	1992	M	19		T3-T4	post	M compl	48 h	M compl	hematoma
		M	28	Ac	T10-L1	post	M compl	28 h	M incompl	hematoma
		F	11		L3-L5	post	M incompl	4 d	M incompl	hematoma
		F	65	Ht	T10-L1	post	M incompl	7 d	M incompl	hematoma
		F	40		C7-T4	post	M incompl	19 h	M incompl	hematoma
		M	30		T3-T5	post	M compl	7 h	unchanged	hematoma
		M	53	Ht	T11-L2	post	M incompl	2 h	M compl	hematoma
Chou	1988	F	75	Ht	C3-C6	postlat	Brown Seq.	3 d	SincompMcomp	hematoma
Combelles	1983	F	73		C6-T2	postlat	SM compl	48 h	SM incompl	hematoma
		F	67	Ac	C3-C4	postlat	SM compl	17 d	unch/died	unknown
		F	28	Pregnant	C4-C7	postlat	SM compl	3 d	SM incompl	unknown
		M	39	Ac	C3-T5	post	SM compl	--	died	unknown
		M	67	Ac	L2-L4	post	SM incompl	4 d	SM incompl	unknown
		F	57	Ac	T11-L1	post	SM compl	24 h	unchanged	hematoma
		M	63	Ac	C4-C6	postlat	SM compl	4 d	SM incompl	unknown
		F	72		T10-T12	postlat	SM compl	24 h	SM incompl	unknown
Cooper	1967	F	14		C3-C5	post	SM incompl	12 h	SM compl	unknown
Correa	1978	M	78		C2-T2	post	SincompMcomp	24 h	SM compl	unknown
Costabile	1984	M	65	Ht	C3-T2	postlat	Brown Seq.	28 h	SM compl	hematoma
		M	72	Ac	T10-T11	post	SM incompl	24 h	SM incompl	hematoma
Crisi	1990	F	58		C3-C5	postlat	Brown Seq.	24 h	SM incompl	hematoma
Cromwell	1977	M	1,5	Hemoph.	C2-T8	post	SM incompl	6 d	SM incompl	unknown
Cube	1962	M	29		C6-T1	post	SM compl	48 h	SM incompl	angioma
Dauch	1986	M	11		T3	post	SM compl	5 h	SM incompl	hematoma

Table 3.1 (continued).

Author	Year	Sex	Age	History	Level	Position	Neurology	Interval	Recovery	Histopathology
Davies	1992	M	56		T9-T11	post	M incompl	24 h	M compl	hematoma
Dawson	1963	F	19		T2-T6	post	SincompMcomp	12 h	SM compl	angioma
		M	15		L2-L5	post	SM incompl	12 h	SM compl	angioma
Delmas	1980	F	70		L4-L5	postlat	rad pain	6 m	complete	hematoma
Demierre	1991	M	28		C3-C7	postlat	Brown Seq.	5 d	SM compl	unknown
		F	70	Ht,DM	C3-C5	postlat	Brown Seq.	--	died	unknown
		F	64	Ht	C3-T1	postlat	SM incompl	24 h	SM compl	unknown
Devadiga	1973	M	41		L5	postlat	rad SMincomp	8 d	SM compl	hematoma
		F	65	Ac	L5	postlat	rad SMincomp	3 d	SM compl	hematoma
Enomoto	1980	M	52		T2-T4	postlat	SM compl	4 d	SM compl	unknown
Farias	1994	F	75		C2-C4	postlat	SM incompl	48 h	SM compl	unknown
		F	76	Ht	C4	postlat	Brown Seq.	3 d	SM compl	unknown
Fischerbach	1972	M	57	Ac	C7-T3	ant	SM compl	24 h	SM incompl	unknown
Flaschka	1990	F	38		T4-T5	postlat	SincompMcomp	12 h	SM compl	hematoma
		F	27		T2-T4	post	SM incompl	48 h	SM compl	angioma
		M	59	Ac	L1-L2	post	SM incompl	24 h	SM compl	hematoma
		M	62		T1-T3	post	SM compl	8 h	SM compl	hematoma
Fliedner	1977	F	72	Ht,DM	T11-L1	postlat	SM compl	24 h	ScompMincomp	hematoma
Foo	1980	M	33		C2-C7	post	SincompMcomp	19 h	unchanged	angioma
	1981	M	36		T2-T4	post	SincompMcomp	24 h	SM comp	hematoma
Fosselle	1978	M	61	Ht	C3-C5	postlat	SM incompl	6 h	SM incompl	hematoma
Franscini	1994	M	43	Ac	T10-T12	post	pain	unknown	complete	hematoma
Freger	1986	M	1,4	Hemoph.	T10-L4	post	SM incompl	unknown	SM incompl	unknown
Galzio	1980	M	58		T1-T6	post	SM compl	9 h	SM compl	hematoma
Gauthier	1963	F	34		T8-T10	post	SM incompl	12 h	SM compl	hematoma
Ghanem	1978	M	8		T1-T3	post	SM compl	12 h	SM compl	hematoma
Giagheddu	1964	M	60	Ht	C5-C6	post	SincompMcomp	24 h	SM compl	hematoma
Girard	1975	F	46	Ht,Ac,DM	T11-L4	postlat	SM incompl	3 d	SM incompl	unknown
Gold	1963	F	62	Ht	T9-L2	postlat	ScompMincomp	48 h	unchanged	unknown
		M	72	Ht	C5-C7	postlat	ScompMincomp	48 h	SM inc/died	unknown
		M	62		C7-T2	postlat	SM compl	48 h	unch/died	unknown
		F	54	Ht	C5-C6	post	SM incompl	24 h	SM compl	unknown
		F	67	Ac	T7-T12	postlat	M incompl	8 d	M inc/died	hematoma
Goulon	1967	F	79	Ht	C6-T1	post	SM incompl	6 d	SM compl	hematoma
Groen	1990	M	29		T3-T5	post	SM incompl	16 h	SM incompl	hematoma
		M	33		T1-T2	post	SincompMcomp	8 h	SM compl	hematoma
		F	73	Ht,Ac	T5-T10	post	SM incompl	48 h	SM incompl	hematoma
Grollmus	1975	M	15		C6-T1	postlat	SM incompl	12 h	SM compl	hematoma
		M	29		C6-T1	postlat	SM compl	6 h	SM compl	hematoma
Gruszkiewicz	1987	M	44	Ht,Ac	T1-T3	post	SM incompl	8 d	ScompMincomp	hematoma
		M	34		T5-T7	post	SM incompl	9 h	SM compl	hematoma
Hack	1984	F	28	Pregnant	T11-L2	post	SM compl	12 h	unchanged	hematoma
Harris	1969	M	66		L5-S1	postlat	rad Sincompl	2 m	complete	hematoma
Haykal	1984	F	65		C3-C6	postlat	unknown	unknown	unknown	hematoma
		M	37	SLE	C2-C5	postlat	unknown	unknown	unknown	hematoma
Hehman	1968	M	1,8		C5-T9	postlat	SincompMcomp	23 d	SM incompl	hematoma
Herrmann	1965	M	38		T4-T9	postlat	ScompMincomp	unknown	unknown	angioma
Hirai	1970	M	44		C3-C5	ant	SM compl	48 h	SM incompl	unknown
Horne	1977	F	27		C7-T1	postlat	Brown Seq.	16 h	SincompMcomp	unknown
		F	78	Ht,DM	T10-T11	post	SM compl	24 h	SM incompl	unknown
Houtteville	1975	M	58	Ac	T11-L3	post	SincompMcomp	4 d	unch/died	hematoma
Huff	1994	M	41	Cocaine	C1-T3	post	SM compl	20 h	unchanged	unknown
Jackson F	1963	F	1,2	Whooping cough	T1-T5	post	M incompl	unknown	M compl	hematoma

Table 3.1 (continued).

Author	Year	Sex	Age	History	Level	Position	Neurology	Interval	Recovery	Histopathology
Jackson R	1869	F	14		C1-C7	post	M compl	--	died	hematoma
Jacobson	1966	F	61	Ht	T2-T4	ant	SM compl	30 h	SM incompl	hematoma
		F	60	Ht,Ac	T12-L2	post	SM compl	48 h	SM incompl	unknown
Jacobson	1966	M	48	Ac	C5-C6	post	SincompMcomp	unknown	SM incompl	unknown
Jacquet	1990	F	82		C3-C7	postlat	SM compl	unknown	SM incompl	hematoma
Jones	1956	M	12	Hemoph.	C6-T1	post	SM compl	unknown	unchanged	unknown
Joseph	1993	F	17		C7-T2	post	SM incompl	17 h	SM incompl	unknown
Jost	1970	M	63	Ac	L1	post	SM incompl	5 d	SM inc/died	unknown
Kania	1990	F	63		T3-T11	post	SM compl	65 h	SM incompl	hematoma
Kaplan	1949	F	39		T5-T6	postlat	SM compl	5 d	unchanged	hematoma
		M	43		T9-L1	circ	SM compl	4 d	unchanged	hematoma
Khatib	1966	M	86		T12-L4	postlat	SM incompl	unknown	SM incompl	unknown
Klossek von	1984	M	36		C5-C7	post	SM compl	8 h	SM compl	unknown
		F	40	Ac	L2-L5	postlat	SM incompl	6 d	SM incompl	unknown
Koyama	1982	M	18		T1	postlat	SM compl	28 h	SM compl	angioma
Krolick	1991	M	69	Ht,Ac	C2-C6	post	SM compl	4 h	unch/died	unknown
Kuchiwaki	1973	M	67	Ht	T11-L1	post	SM compl	64 h	SM incompl	unknown
Lazorthes	1968	F	62		T4-T5	post	SM compl	3 d	unch/died	unknown
		F	23		C4-C5	post	SM compl	36 h	unchanged	hematoma
		F	72	Ac	T5-T6	post	SM compl	6 d	unchanged	unknown
Lecuire	1966	F	34		T6-T8	post	SM compl	80 h	SM compl	hematoma
Lepoire	1961	M	32		C5-C7	postlat	SM compl	50 h	unch/died	unknown
		M	19		C6-T2	postlat	SM compl	3 d	unch/died	unknown
		M	15		C6-T1	post	SM compl	8 h	SM compl	unknown
		M	33		C7-T2	post	SM compl	18 h	SM incompl	unknown
Levitan	1983	F	58		L4	postlat	rad Mincomp	2 m	unknown	unknown
		F	90		L3	postlat	SM incompl	6 w	unknown	unknown
Lévy	1964	M	67	Ht,Ac	L2-L4	postlat	SM incompl	3 d	SM incompl	hematoma
		F	58	Ac	C5-T1	postlat	SM compl	48 h	SM inc/died	hematoma
Licata	1988	F	54		T12-L2	post	SincompMcomp	unknown	died	hematoma
		F	70		C4-C6	post	SincompMcomp	unknown	died	hematoma
		M	1,5		T1-T2	post	SM incompl	unknown	SM compl	hematoma
		M	80		T12-L3	post	SM incompl	unknown	SM incompl	hematoma
		M	76		L4-L5	postlat	SM incompl	unknown	died	hematoma
		F	33		L5-S1	postlat	rad Mincomp	unknown	M compl	hematoma
		M	65		L3-L4	postlat	rad SMincomp	unknown	SM compl	hematoma
Liebeskind	1975	M	23		C7-T2	postlat	SM incompl	8 d	ScompMincomp	hematoma
Lin	1961	F	53	Ht	L1-L3	post	SincompMcomp	48 h	unchanged	hematoma
Lizuka	1972	F	62	Ac	T9-T11	post	SM compl	48 h	SM incompl	hematoma
Locke	1976	M	36	Ht,Ac	T5-T6	post	SM compl	24 h	ScompMincomp	hematoma
London	1974	M	50	Alcohol	C2-S1	circ	SM compl	--	died	hematoma
Lord	1993	M	68	Ht	C4-C5	postlat	SM incompl	6 h	SM compl	unknown
Lorenzo di	1990	M	37	Ht,Ac	T7-T10	postlat	SM compl	7 d	unchanged	hematoma
Lougheed	1960	M	33		T3-T6	post	SM compl	3 d	ScompMincomp	angioma
		F	74	Ht	T10-L1	post	SincompMcomp	10 d	SM incompl	unknown
		M	67	Ht	T11-L5	post	SM incompl	12 h	SM compl	unknown
		M	57		T3-T5	postlat	SM compl	10 h	SM incompl	hematoma
		F	54		T5-T10	postlat	SM compl	4 d	unchanged	unknown
		M	55	Ht	T11-S1	post	SM incompl	3 d	SM incompl	unknown
Lowrey	1959	F	71	RA	C5-T5	post	M incompl	48 h	M incompl	unknown
		M	23		T11	post	SM incompl	48 h	SM incompl	unknown
		M	52		T12-L2	post	SM compl	40 h	unchanged	unknown
MacFarlane	1957	M	15	Hemoph.	T5-L4	post	SM compl	48 h	unchanged	unknown
Machado	1989	M	88		L3-L4	postlat	rad pain	4 m	complete	hematoma
Mahieu	1994	F	26	Pregnant	C3-T1	postlat	Brown Seq.	3 h	SM compl	hematoma

Table 3.1 (continued).

Author	Year	Sex	Age	History	Level	Position	Neurology	Interval	Recovery	Histopathology
Major	1991	M	54		T12	postlat	SM compl	65 h	SM incompl	unknown
		M	22		C4	post	SM compl	6 h	unchanged	unknown
		F	15		T1	postlat	SM compl	3 d	unchanged	unknown
		F	38		C6-C7	postlat	Brown Seq.	48 h	SM incompl	unknown
		M	12	Hemoph.	T2	post	SM compl	5 h	SM compl	unknown
		M	57	Ac	T12-L2	post	SM incompl	96 h	SM compl	unknown
		M	56		C6-T1	postlat	Brown Seq.	6 h	SM compl	unknown
Markham	1967	F	79	DM	T8-T11	post	SM compl	12 h	ScompMincomp	hematoma
		M	45		C7-T1	circ	SM incompl	18 h	SM inc/died	hematoma
		F	56		T10-T12	postlat	SM compl	30 h	SM compl	hematoma
Marmey	1990	M	35		C5-T2	post	SM incompl	12 h	SM compl	unknown
Matsumae	1987	M	16		C4-C7	post	SM incompl	60 h	ScompMincomp	hematoma
Mattle	1987	M	69	Ac	C3-T6	post	SincompMcomp	90 h	SM incompl	unknown
		M	49	Ac	T1-T6	post	SM incompl	12 h	SM compl	unknown
		F	63	Ac	T12-L3	post	SM compl	24 h	unchanged	unknown
		M	46	Ac	L1-L3	post	SincompMcomp	3 d	SM incompl	unknown
		F	63	Ac	C3-T2	post	SincompMcomp	24 h	unch/died	unknown
		M	46	Ac	C7-T1	circ	Brown Seq.	1 w	SM compl	unknown
		M	42		C7-T1	post	SincompMcomp	30 h	SM incompl	unknown
		M	38		L3-L4	post	SM incompl	12 h	SM compl	unknown
		M	60	Alcohol	T8	ant	SM compl	20 h	SM incompl	unknown
Maxwell	1957	M	4		T2-T3	postlat	SincompMcomp	8 d	SM incompl	hematoma
Mayer	1963	F	17		C7-T1	post	SM compl	18 h	SM incompl	angioma
		M	49		C3-T1	post	SM incompl	24 h	SM compl	hematoma
		F	65	Ht	T7-L3	post	SM incompl	5 h	SM compl	unknown
McQuarrie	1978	F	33	Ac	C4-T3	postlat	SM compl	36 h	SM compl	unknown
		F	55	Ht	T11	post	SincompMcomp	30 h	SM incompl	unknown
		F	32		C7-T2	post	SincompMcomp	12 h	SM incompl	unknown
Mérienne	1973	F	21		T8-T10	post	SincompMcomp	12 h	SM incompl	hematoma
		M	49	Ht,Ac	T9-T11	post	SM incompl	unknown	unknown	hematoma
Mishima	1989	M	76	Ac	T11-L3	post	SM compl	17 h	SM compl	hematoma
Miyasaka	1977	M	47		T1-T3	post	SM compl	8 d	unchanged	angioma
Mohazab	1993	F	39	SLE	C1-T4	post	SM compl	unknown	SM incompl	unknown
Mracek	1980	M	66		T9-T11	post	SM compl	24 h	unchanged	hematoma
		F	62	Ht	T8-T11	post	SM compl	4 d	unchanged	unknown
		F	72	Ac	L3-L5	post	SM incompl	6 d	unch/died	hematoma
		M	52		T8-T11	post	SM compl	48 h	SM incompl	hematoma
Müller	1982	F	71	DM	C5-C7	ant	Brown Seq.	18 d	SM compl	angioma
		M	65		T5-T10	postlat	SM compl	24 h	unchanged	angioma
		F	59		T9-T10	post	SM compl	12 h	SM incompl	hematoma
		F	53		T9-T11	post	SM incompl	12 h	unchanged	angioma
Murata	1984	M	75	Ht	C2-C5	postlat	Brown Seq.	3 d	SM incompl	hematoma
Mustafa	1987	F	70	Hemoph.	C5-C6	postlat	Brown Seq.	unknown	SM compl	hematoma
	1988	M	49	Ac	L4-L5	postlat	SM incompl	3 d	unknown	hematoma
Nagel	1989	F	7		C3-T1	post	SM incompl	unknown	SM compl	hematoma
Nehls	1984	M	74		L3-L4	postlat	rad SMincomp	2 m	SM compl	hematoma
Nichols	1956	M	15		C6-T1	post	SM compl	12 h	SM compl	hematoma
Ogawa	1985	F	25		T3-T4	ant	M compl	8 h	M compl	unknown
Oldenkott	1966	M	50	Ac	T7-T9	post	SM compl	unknown	SM inc/died	unknown
Packer	1978	F	13		C7-T7	postlat	SM compl	10 h	SM compl	angioma
	1978	M	17		T9	postlat	SM incompl	6 d	SM incompl	angioma
Paliard	1967	F	64	Ht,Ac	C6-T4	postlat	SM compl	30 h	unch/died	unknown
Panitz	1975	F	26	Puerp.	T2	postlat	SM compl	12 h	ScompMincomp	hematoma
Parman	1980	M	34		T8-T10	postlat	SincompMcomp	12 h	ScompMincomp	unknown
Pear	1972	M	15		T5-T6	postlat	SM incompl	unknown	SM compl	hematoma
		M	68	Ht	T11-T12	postlat	SM incompl	unknown	SM incompl	unknown
		M	66		L5-S1	postlat	SM incompl	unknown	SM compl	hematoma
		F	75		L3-L4	postlat	SM incompl	unknown	unknown	hematoma
		M	76	Ac	C4-C6	post	M incompl	24 h	M compl	hematoma
		M	47	Ht,Ac	T2	post	SincompMcomp	24 h	unknown	unknown
		M	73	Ac	L1-S1	post	SM incompl	unknown	SM incompl	unknown

Table 3.1 (continued).

Author	Year	Sex	Age	History	Level	Position	Neurology	Interval	Recovery	Histopathology
Penar	1987	M	48		C5-T3	post	SincompMcomp	6 d	SM compl	hematoma
		F	54		T1-T4	post	SM compl	24 h	SM compl	angioma
Pendl	1971	M	13		T5-T8	post	SM incompl	unknown	SM compl	hematoma
		M	71	Ht	T8-T10	post	SM compl	24 h	unchanged	hematoma
			63		L5	postlat	rad Sincomp	6 w	S compl	hematoma
		M	46	Ac	T9-L1	postlat	SM incompl	24 h	SM compl	hematoma
Peserico	1959	F	74		C5-C7	post	SM incompl	48 h	SM compl	hematoma
Petrov	1979	M	56	Ht,Ac	T4-T12	post	SM compl	10 h	SM compl	hematoma
Phillips	1981	M	21		T2-T6	ant	SM incompl	10 h	SincompMcomp	hematoma
Plagne	1961	M	38		T1-T4	ant	SM compl	48 h	unchanged	unknown
Piontud	1979	M	63	Ac	L4-S2	postlat	SM incompl	3 d	SM incompl	unknown
Pommé	1959	M	55		L3-L4	post	SM compl	6 d	SM incompl	unknown
Posnikoff	1968	F	2,5		C5-T5	postlat	M incompl	unknown	M compl	hematoma
Quequet	1987	F	76		C4-T2	postlat	SM incompl	5 d	SM incompl	unknown
		F	63	Ht	T12-L2	post	M incompl	unknown	M compl	hematoma
Rao	1966	M	17		L4-L5	postlat	rad Mincomp	4 d	M compl	hematoma
Rathe	1969	M	54		C4-C6	post	SM compl	unknown	ScompMincomp	hematoma
Rebello	1966	M	11		C3-C7	postlat	SM compl	3 d	ScompMincomp	hematoma
		M	20		C6-C7	postlat	ScompMincomp	17 h	SM incompl	hematoma
Reddy	1972	F	60		T4-T5	post	SM incompl	12 h	SM compl	unknown
Robertson	1979	F	6		T1-T3	post	SincompMcomp	4 h	SM incompl	hematoma
Rose	1990	M	87	Ac	T8-T10	post	SincompMcomp	42 h	SM incompl	hematoma
Rothfus	1987	M	65		C6-T8	post	SM incompl	48 h	SM compl	hematoma
		F	78		C2-C3	postlat	SM incompl	48 h	SM incompl	hematoma
		F	50		L3-S1	postlat	SM incompl	3 d	SM compl	hematoma
Russman	1971	F	53	Ht	C4-C7	post	Brown Sequard	36 h	SincompMcomp	hematoma
Santa	1990	F	61	Ht	T10-T12	post	SM incompl	6 h	SM compl	hematoma
Sasaki	1987	M	71	DM	T10-L2	postlat	SM compl	36 h	SM incompl	hematoma
Scharfetter	1972	F	65	Ht	T6-L1	post	ScompMincomp	18 h	unchanged	unknown
		M	60	Ht	T9-L1	post	SM incompl	3 d	SM incompl	unknown
		F	63	Ac	T1-L3	post	SM compl	--	died	hematoma
Scheil	1990	F	62	Ac	T7-T9	circ	SM compl	48 h	SM incompl	hematoma
		M	46	Ac	T7-T12	circ	SM compl	12 h	SM incompl	hematoma
		M	16		C7-T2	postlat	SM incompl	4 d	SM compl	hematoma
		M	75		L3-L4	post	SM incompl	3 w	SM compl	hematoma
Scheil	1990	M	58	Ac	C3-C7	postlat	SM incompl	17 d	SM incompl	hematoma
		M	71		L4-L5	postlat	rad SMincomp	2 m	SM compl	hematoma
		F	37		T8-T11	post	SM compl	7 h	SincompMcomp	angioma
Schicke	1970	M	60	Ac	T12-L1	postlat	Brown Seq.	2 w	SM incompl	unknown
Schiffer	1984	M	56		L1-L3	post	SM incompl	6 d	SM incompl	unknown
Schultz	1953	M	24		T2-T4	post	SincompMcomp	24 h	SM compl	unknown
		M	79		L2-L4	circ	SincompMcomp	12 d	SM compl	hematoma
		F	56		T10-T12	post	SM compl	29 h	SM incompl	hematoma
		M	34		T9-L1	postlat	SM compl	8 d	SM incompl	hematoma
Scott	1976	F	63	Ht	C3-T2	circ	SincompMcomp	30 h	SM incompl	unknown
		M	52		C5-T3	circ	SincompMcomp	12 h	SM incompl	unknown
Segelov	1967	M	47	Ac	T1	post	SM incompl	12 h	SM compl	unknown
Servadei	1987	F	74	Ht	T8-T11	post	SM compl	4 d	SM incompl	hematoma
Shen	1992	M	17		C4-C6	post	SincompMcomp	20 h	SM compl	hematoma
		M	30		T10-L1	postlat	SincompMcomp	20 h	SM incompl	hematoma
		F	75		C3-C7	postlat	Brown Seq.	48 h	SM compl	hematoma
		M	59		T5-T10	post	SM compl	48 h	SM incompl	hematoma
	1995	M	58	Ht	C2-C6	postlat	Brown Seq.	18 h	SM compl	hematoma
Shenkin	1945	M	1,8		T1-T7	post	SM compl	2 w	SM compl	hematoma
		M	42		T2-T5	post	SincompMcomp	1 w	unchanged	hematoma
Simmons	1978	M	68	Ht,Ac	C3-T2	circ	SM compl	12 h	SM incompl	hematoma
Solero	1980	M	38		C5-T1	post	SincompMcomp	32 h	SM compl	angioma

Table 3.1 (continued).

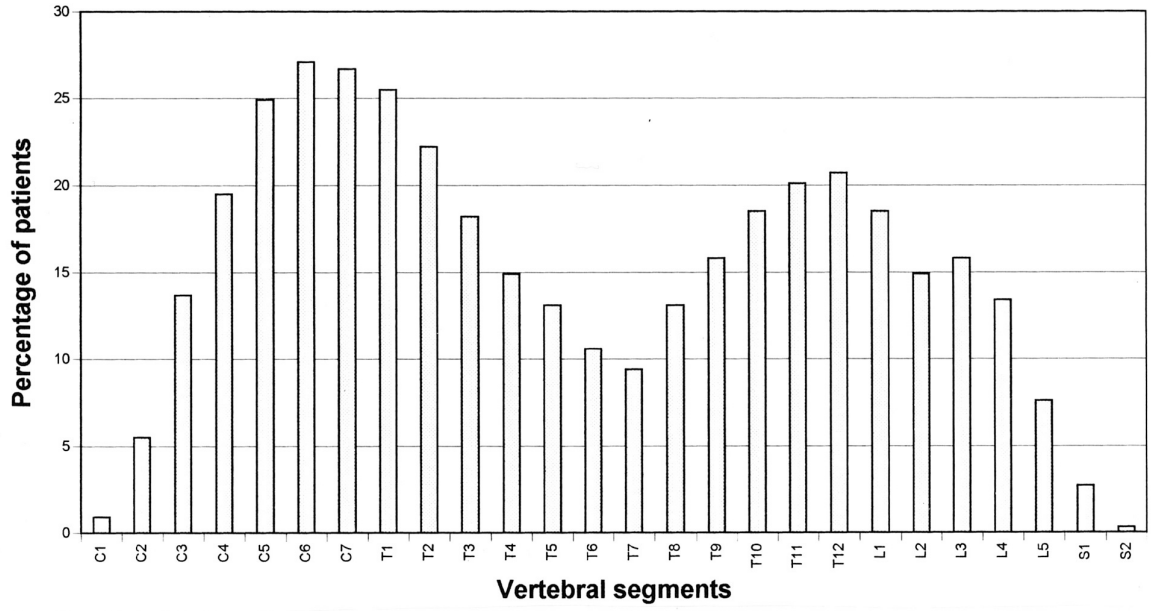
Author	Year	Sex	Age	History	Level	Position	Neurology	Interval	Recovery	Histopathology
Soustiel	1988	F	64	Ac	T12-L1	circ	SincompMcomp	10 h	SM incompl	unknown
Spanu	1987	F	57		T12-L4	postlat	ScompMincomp	unknown	SM incompl	unknown
		F	68		T12-L3	postlat	SincompMcomp	unknown	SM incompl	unknown
Spill ter	1989	F	15		T8-T10	post	SincompMcomp	12 h	SM compl	hematoma
Spurny	1964	F	67	Ht,Ac	T9-L2	postlat	SincompMcomp	3 d	SM incompl	unknown
		M	70	Ac	T11-L4	circ	SM incompl	3 d	SM incompl	unknown
Sreerama	1973	M	45	Ac	L1-L5	post	M compl	6 h	M incompl	unknown
Stanley	1983	M	13	Hemoph.	T5-T8	lat	SM incompl	48 h	SM incompl	hematoma
Strain	1964	M	57	Ac	C2-T2	post	SM compl	48 h	SM incompl	unknown
Suzuki H	1993	M	61		T11	post	SM incompl	4 d	SM compl	hematoma
Suzuki N	1977	F	29	Pregnant	T1-T3	circ	SM compl	4 d	SM incompl	unknown
Suzuki S	1968	F	23		T6-T9	ant	M compl	31 h	unchanged	unknown
Svien	1950	M	67		L3-L4	postlat	M incompl	unknown	M compl	hematoma
Telerman	1972	M	48	Ac	T12-L4	post	SincompMcomp	24 h	SM incompl	unknown
Tewari	1992	F	11		C4-C7	postlat	SM compl	24 h	SM incompl	hematoma
		M	8		T10-L4	post	SM incompl	50 d	SM incompl	hematoma
		M	5		C5-T4	post	SM compl	5 d	SM incompl	hematoma
Tsai	1975	M	60	Ht	C4-C7	postlat	Brown Seq.	10 h	SM incompl	unknown
		F	20		T3-T4	post	SM compl	6 h	unchanged	hematoma
		M	17		L3-L4	lat	rad SMincomp	4 w	SM compl	unknown
Twerdy	1984	F	30		T8-T12	post	SM compl	24 h	SM compl	hematoma
		M	68	Ac	L1-L2	post	SincompMcomp	2 w	SM incompl	hematoma
Valleé	1982	F	1,8		C5-C7	post	SM incompl	48 h	SM compl	hematoma
Vapalahti	1975	M	63	Ac	T12-L1	post	SincompMcomp	24 h	SM incompl	unknown
		F	63	Ac	T9-T12	post	SM compl	12 h	unchanged	unknown
		M	66		T12-L4	post	SM incompl	24 h	SM incompl	unknown
Ventureyra	1979	M	8		T1-T3	post	SM compl	12 h	SM compl	hematoma
VerBrugghen	1946	M	75		C5-C7	post	SM compl	12 h	SM incompl	unknown
Verhagen	1986	M	70	Ac	C4-T2	postlat	SincompMcomp	24 h	SM incompl	unknown
Watts	1976	F	73		T11-L2	post	SincompMcomp	12 h	SM incompl	hematoma
Weigert	1961	M	43	Ac	T10-T12	post	SM compl	53 h	unchanged	hematoma
Whaley	1962	M	52	Ac	T10-T12	postlat	Brown Seq.	24 h	SM incompl	unknown
Williams	1987	F	81		T12-L1	postlat	SM incompl	48 h	SM compl	hematoma
		F	7		C5-T1	postlat	SM incompl	3 d	SM incompl	hematoma
Winer	1959	F	70	Ht,Ac,DM	T6-T11	post	SM compl	45 h	unchanged	unknown
Wittebol	1984	F	6		C6-T6	postlat	SM compl	18 h	SM incompl	hematoma
		M	75		C4-T1	post	SM compl	9 h	SM compl	unknown
		M	52		C4-C7	postlat	Brown Seq.	unknown	SM incompl	hematoma
		F	70	Ht,Ac,DM	T1-T3	circ	SincompMcomp	3 d	unchanged	unknown
Yettou	1995	F	29		T1-T2	post	SM compl	14 h	SM incompl	unknown
		M	27		T4-T8	post	SM compl	8 h	unchanged	unknown
Yonekawa	1975	F	20	Pregnant	C4-C6	post	SM compl	17 h	SM incompl	hematoma
Yu	1986	M	63		T1-T3	post	SM incompl	6 h	SM compl	hematoma
		M	31		T2-T4	postlat	Brown Seq.	unknown	SM incompl	hematoma
Zilkha	1983	M	62		C3-C6	postlat	SM incompl	10 h	SM compl	hematoma
		M	67	Ht,Ac	T11-L3	postlat	SM compl	48 h	SM incompl	unknown
Zouaoui	1980	M	14	Hemoph.	T12-L3	post	SincompMcomp	24 h	SM incompl	unknown
Zuccarello	1980	M	45	Ac	C4-T1	postlat	SM compl	24 h	unchanged	hematoma
Zupruk	1989	M	86		C3-C7	postlat	Brown Seq.	12 h	SM compl	unknown

[a]Ac,anticoagulants; Ant,anterior; Circ,circular; CML,chronic myelogenous leukemia; compl,complete; DM,diabetes mellitus; Hemoph, hemophilia; Ht, hypertension; incompl, incomplete; M, motor; Moschc, Moschcowitz's disease; Post, posterior; Postlat, posterolateral; Puerp, puerperium (postpartum); RA,rheumatoid arthritis; Rad, radicular; S, sensory; SLE, systemic lupus erythematosus.

3.3 Results.

This study comprised 333 patients, 137 females and 196 males (ratio [M:F], 1.4:1), between 1 and 90 years old (mean, 47.8 yr). All vertebral segments were affected; however, the cervicothoracic and thoracolumbar areas predominated (*Fig. 3.1*). Spinal cord compression (SSEHs restricted to vertebral segments C1-L1), occurred in 257 patients (77%), cauda equina compression (L2-S2) in 21 patients (6%), and both cord and caudal compression were present in 43 cases (13%). Isolated lumbar and/or sacral root compression occurred in 12 patients (4%).

Figure 3.1. Segmental distribution in SSEH (N = 333).



The preoperative neurological condition is listed in *Table 3.2.* In 193 patients (58%), sensory and/or motor (SM) deficit was incomplete and in 123 patients (37%) SSEH resulted in complete sensorimotor loss. Isolated radicular symptoms appeared in 15 patients (4%) and preoperative symptoms were not known in 2 cases (1%).

Table 3.2 Preoperative neurological condition after SSEH[a].

Only radicular symptoms	15
SM incomplete deficit	113
Brown Sequard syndrome (SM incomplete)	23
S complete M incomplete deficit	6
M complete S incomplete deficit	51
SM complete deficit	123
Unknown	2
Total	333

[a]S, sensory; M, motor.

The hematomas were removed surgically in 327 patients. In six patients, the hematoma was diagnosed at autopsy. In 276 patients, surgery was performed within a time interval of 2 hrs to 6 mnts following the onset of symptoms; in 51 patients, the operation interval was not specified.

Postoperative outcome is listed in *Table 3.3*. In 12 patients (4%), postoperative outcome was unknown. In 49 patients (15%), the neurological condition did not improve after surgery. In 147 patients (44%), postoperative recovery was incomplete and 115 patients (34%) recovered completely. Six patients died before surgery and four patients died immediately after surgery. Therefore, no postoperative neurological data were available from these ten patients (3%).

Table 3.3 Postoperative outcome after SSEH[a].

Unchanged	49
SM incomplete recovery	128
S complete M incomplete recovery	14
M complete S incomplete recovery	5
SM complete recovery	115
Died (before or shortly after operation)	10
Unknown	12
Total	333

[a]S, sensory; M, motor.

Another 18 patients died several hours to months after surgery; cardiovascular complications (pulmonary embolism [7] or myocardial infarction [3]) and respiratory insufficiency [7] were the major causes of death. Cardiovascular complications were highly associated with perioperative interruption of anticoagulant therapy (8 of 12 patients) (*Table 3.4*); however, this small number of patients did not allow statistical analysis. In 17 deaths, SSEH was located in the cervical [11] or cervicothoracic [6] vertebral area. Mortality correlated ($P < 0.05$) with SSEH in those areas (*Table 3.5*).

Table 3.4 Relation of cardiovascular risk factors to causes of death[a].

Causes of death	Ac	Ht	Ac + Ht	none	Total
Pulmonary embolism	5	-	1	1	7
Myocardial infarction	1	1	1	-	3
Respiratory insufficiency	-	2	-	5	7
Others/unknown	4	2	-	5	11
Total	10	5	2	11	28

[a]Ac, anticoagulants; Ht, hypertension.

Table 3.5 Relation of mortality to vertebral segment in SSEH[a].

Location	Deaths	Survivors	Total
Cervical	10 (16)	51 (84)	61
Cervicothoracic	7 (11)	56 (89)	63
Thoracic	4 (4)	109 (96)	113
Thoracolumbar	3 (6)	49 (94)	52
Lumbar	3 (8)	34 (92)	37
Total	27 (8)	299 (92)	326

[a]Number of patients (percent); $P < 0.05$.

All patients who experienced only radicular symptoms recovered completely after surgery, regardless of the location of the hematoma or the operative interval.

Postoperative outcome (complete, incomplete, or unchanged) after spinal cord decompression was related to the preoperative neurological condition (complete or incomplete sensory and/or motor deficit) and the operative interval (*Table 3.6*). From these data, the corresponding mean operation intervals, sample standard deviations (SSD), and standard errors of the mean (SEM) were calculated. In patients with complete recovery, mean operation interval was shortest (34.5 h). The 95% confidence limits for those intervals were calculated. The mean operative interval in patients with complete recovery is significantly shorter than in patients with incomplete or no improvement after spinal cord decompression. Mean operative interval in patients who recovered completely after complete sensorimotor loss was significantly shorter compared with the five other subgroups. The data for incomplete recovery do not differ from the unchanged outcome categories; ranges overlap completely between those groups, and standard deviations are relatively similar. No statistical difference was found between the operative intervals from both groups. This suggests that the outcome (in those groups) is determined by the level of preoperative neurological deficit (*Table 3.6*). We found evidence for this assumption after calculation of the data in *Table 3.7*; postoperative outcome after incomplete sensory and/or motor deficit was significantly better compared to recovery after complete loss of sensorimotor functions (Chi-square = 41.4, $P < 0.0005$).

Table 3.6 Outcome related to preoperative neurological deficit and operative interval in spinal cord compression for SSEH (N = 252).

Outcome	Complete recovery		Incomplete recovery		Unchanged	
	83		121		48	
Neurological deficit	SMcompl	SMincompl	SMcompl	SMincompl	SMcompl	SMincompl
	23	60	50	71	37	11
Interval (hours) range	[5-96]	[2-216]	[5-216]	[4-672]	[4-192]	[12-168]
mean	22.7826	39.0166	46.8600	101.4225	49.9189	72.0909
SSD	23.4423	45.9197	43.6904	139.6409	46.0919	57.3941
SEM	4.8880	5.9282	6.1787	16.5723	7.5774	12.3049
mean	34.5180		78.8760		55.0000	
SSD	41.4494		113.4992		49.1610	
SEM	4.5496		10.3181		7.0957	

Table 3.7 Relation of outcome to preoperative neurological condition[a].

Deficit	Unchanged	Incomplete recovery	Complete recovery	Total
SM incomplete	12 (7)	88 (49)	80 (44)	180
SM complete	37 (32)	59 (50)	21 (18)	117
Total	49 (16)	147 (50)	101 (34)	297

[a]Number of patients (percent); $P < 0.0005$. S, sensory; M, motor.

The relationship between preoperative neurological condition, operative interval, and postoperative outcome was evaluated. In patients with complete preoperative sensorimotor loss, results were significantly better if surgery was performed <36 hours after onset of the neurological deficit (Chi-square = 6.99, $P < 0.05$) (*Table 3.8*). When the "critical" operative interval was fixed at >36 hours, no statistically significant difference in postoperative results could be observed. In patients with incomplete sensorimotor deficit, favorable outcomes correlated highly with surgery in ≤48 hours (Chi-square = 11.99, $P < 0.005$) (*Table 3.9*); this correlation disappeared when the "critical" operative interval was fixed at >48 hours. No correlation could be observed between the spinal level (vertebral segments) and postoperative outcome (*Table 3.10 and 3.11*). Nevertheless, postoperative recovery from complete sensorimotor loss caused by SSEH involving the T1-T7 vertebral segments was significantly better than the outcome after complete neurological deficit caused by spinal cord compression at the T8-T12 vertebral segments (Chi-square = 7.6, $p < 0.025$) (see *Table 3.10*). The mean operation intervals did not differ significantly, but the mean age was significantly higher in patients with hematomas at T8-T12 (57.2 versus 34.4 yr; [standard error of the mean 4.5723; estimated 95% confidence interval, 32-14 yr]).

Table 3.8 Relation of outcome in complete sensorimotor deficit to operative interval[a].

Interval	Unchanged	Incomplete recovery	Complete recovery	Total
≤36 h	18 (29)	26 (42)	18 (29)	62
>36 h	18 (38)	25 (53)	4 (9)	47
Total	36 (33)	51 (47)	22 (20)	109

[a]Number of patients (percent); $P < 0.05$.

In the group of patients with complete preoperative sensorimotor loss caused by spinal cord compression, the time was scored that elapsed until the transverse lesion was complete. A very quick deterioration (within minutes) occured in 22 patients. In 27 patients, the evolution to complete sensorimotor deficit lasted ≥12 hours (mean, 59.6 h [12-360 h]). However, the speed of neurological deterioration did not affect postoperative neurological outcome (Chi-square = 4.1 [not significant]) (*Table 3.12*), which might be related to the long interval until surgery was performed in both groups (mean, 43.1 h [6-96 h] and 43.8 h [6-96 h], respectively). Finally, the extent of the hematoma (i.e., the number of spinal segments involved) did not correlate with preoperative neurological deficit or with postoperative outcome (*Table 3.13 and 3.14*).

Table 3.9 Relation of outcome in incomplete sensorimotor deficit to operative interval[a].

Interval	Unchanged	Incomplete recovery	Complete recovery	Total
≤48 h	7 (7)	39 (39)	54 (54)	100
>48 h	4 (7)	38 (67)	15 (26)	57
Total	11 (7)	77 (49)	69 (44)	157

[a]Number of patients (percent); $P < 0.005$.

Table 3.10 Relation of outcome after complete sensorimotor deficit to spinal area (N = 115)[a].

Spinal area	Unchanged	Incomplete recovery	Complete recovery	Total
Cervical	5 (33)	9 (60)	1 (7)	15
Cervicothoracic	7 (27)	12 (46)	7 (27)	26
Thoracic (T1-T7)	9 (29)	10 (32)	12 (39)	31
Thoracic (T8-T12)	7 (29)	15 (63)	2 (8)	24
Thoracolumbar	6 (40)	8 (53)	1 (7)	15
Lumbar	-	4 (100)	-	4
Total	34 (30)	58 (50)	23 (20)	115

[a]Number of patients (percent).

Table 3.11 Relation of outcome after incomplete sensorimotor deficit to spinal area (N = 172)[a].

Spinal area	Unchanged	Incomplete recovery	Complete recovery	Total
Cervical	2 (5)	17 (44)	20 (51)	39
Cervicothoracic	1 (3)	17 (47)	18 (50)	36
Thoracic (T1-T7)	2 (8)	9 (34)	15 (58)	26
Thoracic (T8-T12)	1 (5)	10 (53)	8 (42)	19
Thoracolumbar	3 (9)	23 (67)	8 (24)	34
Lumbar	2 (11)	10 (56)	6 (33)	18
Total	11 (6)	86 (50)	75 (44)	172

[a]Number of patients (percent).

Table 3.12 Relation of outcome to the rapidity of neurological deterioration untill complete sensorimotor deficit[a].

Interval	Unchanged	Incomplete recovery	Complete recovery	Total
Minutes	12 (55)	9 (40)	1 (5)	22
≥12 h	11 (41)	9 (33)	7 (26)	27
Total	23 (47)	18 (37)	8 (16)	49

[a]Number of patients (percent); $P < 0.2$ (not significant).

Table 3.13 Relation of extent of hematoma to neurological deficit[a].

Number of segments	1	2	3	4	5	6	7	≥8	Total
SM incomplete deficit	9	35	59	32	19	21	7	11	193
SM complete deficit	9	13	41	22	14	8	6	10	123
Total	18	48	100	54	33	29	13	21	316

[a]$P < 0.5$ (not significant). SM, sensorimotor.

Table 3.14 Relation of extent of hematoma to postoperative outcome[a].

Number of segments	1	2	3	4	5	6	7	≥8	Total
Unchanged	2	6	16	9	5	6	2	3	49
SM incomplete recovery	8	19	43	33	17	13	5	9	147
SM complete recovery	8	28	37	10	11	9	5	7	115
Total	18	53	96	52	33	28	12	19	311

[a]$P < 0.4$ (not significant). SM, sensorimotor.

3.4 Discussion.

SSEH usually presents as an acute syndrome with severe pain, followed by signs and symptoms of spinal cord and/or nerve root compression. Recognition of the symptoms and rapid diagnostic evaluation is essential to minimize delay in surgery. Magnetic resonance imaging (MRI), allowing accurate noninvasive multiplanar investigation of the regions of interest, has replaced myelography and computed tomographic scanning as the imaging technique of first choice for diagnosis of SSEH. The cause of SSEH has been discussed in the literature, but to date it cannot be decided from the available data whether the origin of the bleeding is arterial or venous. Bleeding diathesis and arterial hypertension are conditions that are often associated with SSEH; however, the pathogenesis of those conditions remains obscure. As a result, there are no arguments to exclude those patients from this series of SSEHs.

Several factors are purported to affect postoperative neurological outcome. Three studies were published on this subject, but specification of the data was incomplete or statistical objectives were missing in those articles (46,111,190). Recently, Lawton et al. (85) presented their experience with operative treatment of 30 patients with spinal epidural hematomas, but only five patients fit into the definition of SSEH. As a result, comparison of the studies was problematic.

3.4.1 Sex and age.

Several authors stated that the final result after surgery, proper postoperative care, and intense rehabilitation is related to the sex and age of the patient (14,74). In contrast, McQuarri concluded that age did not influence the probability of recovery (111). In the present study (including the material from Refs. 14 and 74), the overall neurological outcome in all 333 patients did not correlate with age or sex.

3.4.2 Localization of the hematoma.

For several reasons, it might be expected that the prognosis after spinal cord compression will correlate with the vertebral level of the hematoma. The intraspinal space that is available for the spinal cord varies with the anteroposterior and the side-to-side dimensions. The interpeduncular space in the thoracic area is significantly smaller compared with the cervical and lumbar vertebral canal; as a result the spinal canal is narrowest between the third and ninth thoracic vertebrae (39,41). Because the surplus of perimedullary space is lowest in the thoracic area (100), a lesion intruding into the relatively small thoracic spinal canal can produce changes that in the larger cervical or lumbar space would not cause significant encroachment on the spinal cord and/or cauda equina.

With regard to the vascular supply of the spinal cord, three main arterial territories

have been distinguished. Both the upper (or cervicothoracic) and lower (or thoracolumbar) territories are richly vascularized, with large collateral arteries. The intermediate or middle-thoracic territory (spinal cord segments T4-T8) has very few collaterals and usually is supplied by only one artery. This makes the middle part of the spinal cord more vulnerable to ischaemia (87). Because of the ascensus of the spinal cord, in adults the vertebrae T3-T7 correspond with spinal cord segments T4-T8 (23). From those osseous and vascular details, we deduced that SSEHs involving the T3-T7 vertebrae should harbor a higher risk forstructural damage of the spinal cord.

Surprisingly, in patients suffering complete preoperative sensorimotor loss, favorable recovery correlated highly with hematomas that were restricted to the T1-T7 vertebrae when compared with SSEHs at T8-T12 ($P < 0.025$) (see *Table 3.10*). The operation interval cannot be responsible for this difference, because mean operative interval appeared not to vary significantly between both groups. Position and extent of the hematomas were similar in both groups, as were sex and medical history. The only significant difference occurred with age; mean age was significantly higher in the group of patients with SSEHs at T8-T12 (57.2 versus 34.4 yr). Although age did not seem to correlate with postoperative outcome in the whole series, younger patients in this subgroup experienced favorable outcome after thoracic spinal cord decompression compared with older patients.

The results after incomplete preoperative neurological deficit did not show significant segmental differences (see *Table 3.11*). Comparison of the segmental distribution of SSEH in deaths and survivals revealed a statistically significant correlation ($P < 0.05$) between mortality and cervical location of the hematomas (*Table 3.5*). In those patients, pulmonary embolism or myocardial infarction (especially after interruption of anticoagulant treatment and subsequent correction of the coagulopathy) and neurogenic respiratory insufficiency were the main reported causes of death. Sawin et al. (152) recently described two patients with SSEH after coronary thrombolysis with tissue-type plasminogen activator. The authors stressed the importance of correction of coagulopathies before performing decompressive laminectomy, which carries a high risk for fatal perioperative thromboembolic complications.

3.4.3 Position of the hematoma.

It has been demonstrated experimentally in monkeys that the effect of epidural masses on the intrinsic blood supply of the spinal cord is influenced by the position of the compressing lesion (141). Anterior and posterior masses obstructed central perforating arteries and lateral masses did not, possibly because of the tethering action of the dentate ligaments and anterolaterally emerging nerve root sheaths that tend to resist cord displacement (141). However, no correlation could be found in the literature between the position of the hematoma and the preoperative neurological deficit in spinal cord compression.

3.4.4 Size of the hematoma.

Foo and Rossier (46) have mentioned that the results of surgical decompression are better when the hematoma is small and confined to one vertebral segment only. However, after application of the Chi-square test on their data we were not able to find any correlation. This was confirmed by statistical analysis of the data from all 333 SSEHs.

3.4.5 Preoperative neurological status.

In the present study, postoperative neurological recovery highly correlates with sensorimotor impairment before surgical intervention. All patients suffering only radicular symptoms made a complete neurological recovery after decompression. In spinal cord compression incomplete preoperative sensorimotor deficit correlated highly with favorable postoperative outcome, no matter how slight the preservation of sensory and/or motor function was before surgery. Complete preoperative sensorimotor loss seemed to carry a high risk for persisting neurological deficit after decompression. Nevertheless, total recovery from a complete transverse spinal cord lesion may occur, even after longstanding compression (40,163).

3.4.6 Force and duration of spinal cord compression.

In experimental animal studies performed by Tarlov and collegues, functional recovery after acute extradural balloon compression of the spinal cord was found to depend on the magnitude of the compression force as well as on its duration (176-178). With large compression forces (complete inflation of a balloon with a diameter approximately the size of the spinal canal), full recovery of function occurred when the compression was released in ≤1 minute. When compression was applied for longer periods, functional recovery usually did not occur. When minimal compressive force was used to produce complete sensorimotor loss (inflation of small balloons just to the point of complete paralysis), recovery occurred even after 2 hours of compression. When the spinal cord was compressed gradually instead of suddenly, the time limits for recovery also increased. When motor function was lost completely but pain sensation was preserved, the duration of spinal cord compression compatible with recovery also was much longer (176).

SSEH in humans resembles the experimental animal models from Tarlov and Klinger (178) because the hematoma causes extradural spinal cord compression. The time interval from the onset of symptoms to complete sensorimotor deficit approximates the force of spinal cord compression (176-178), and the interval from neurological deficit to surgery approximates the duration of spinal cord compression (111). We studied the operative interval in all patients, as well as the compression force in patients with complete preoperative sensorimotor loss.

Postoperative neurological outcome was highly correlated with operative interval (*Table 3.8 and 3.9*). In patients with complete preoperative neurological deficits, favorable outcome was achieved when surgery was performed in ≤36 hours ($p < 0.05$). In patients with incomplete preoperative sensorimotor deficits, favorable outcome correlated highly with operative decompression in ≤48 hours ($P < 0.005$). These data correspond with the findings of Tarlov (176). It should be emphasized that the intervals mentioned above are not absolute; they show that there is limited time to enable significant neurological recovery after incomplete sensorimotor deficit due to SSEH, which is even more critical in patients with complete sensorimotor loss.

A correlation between neurological outcome and the speed of neurological deterioration in spinal cord compression that resulted in complete sensorimotor deficit could not be found. In 22 patients with a very rapidly evolving paralysis (minutes) and in 27 patients with slow progression (≥12 h until complete sensorimotor deficit), postoperative outcome did not correlate. This is possibly related to the relatively small number of patients involved. However, it is important to realize that the average interval from completeness of the deficit until surgery was 43 hours in both groups. Because we have shown that late decompression after complete sensorimotor deficit (operative interval >36 h) correlated highly with unfavorable outcome (*Table 3.8*), conclusions about the impact of the compression force on postoperative outcome after spinal cord compression can not be drawn from this series. Based on these observations, we cannot agree with the statements of Foo and Rossier (46), Von Klossek and Hüller (190), and Flaschka et al. (44) that rapid evolution of the paresis in SSEH results in unfavorable postoperative recovery.

3.5 Conclusions.

The major factors determining neurological recovery after SSEH are the localization of the hematoma (vertebral segments involved), the preoperative neurological condition, and the operative interval. Mortality after SSEH correlated highly with the cervical localization, especially in patients with hypertension and those undergoing anticoagulant therapy. Among those patients, pulmonary embolism was one of the main causes of death. For this reason, anticoagulants should not be discontinued completely and agressive correction of coagulopathies perioperatively should be avoided in this category of patients. The prognosis of a radicular syndrome caused by a SSEH is excellent. Although the vascular and osseous details suggest a higher vulnerability and susceptibility of the T3-T7 spinal cord for compression and mass lesions, postoperative outcome did not correlate with the spinal cord segments involved in SSEH. Our findings indicate that local compression, rather than vascular obstruction and subsequent spinal cord anoxia, is the main factor in producing compression paralysis.

In contrast with suggestions in the literature, sex, age, size of the hematoma, and position of the hematoma in the spinal canal did not seem to affect postoperative outcome after SSEH. In spinal cord compression, the critical factors for recovery after SSEH include the time span between the establishment of the neurological symptoms and surgery as well

as the degree of the neurological deficit. In patients with complete preoperative sensorimotor deficit, surgery in ≤36 hours correlated with favorable outcome; in patients with incomplete preoperative sensorimotor deficit, favorable outcome correlated with surgery in ≤48 hours. Based on the present study, the impact of the rapidity of neurological deterioration untill complete sensorimotor loss on postoperative recovery remains unknown. Statistical analysis of this item was not possible in the present study, because operative intervals were too long (mean interval >42 hours), which in itself does result in unfavorable outcome. Only careful documentation and prompt surgery of future patients with SSEH may lead to clinical evidence about a correlation between the compression speed of the spinal cord and postoperative neurological outcome.

3.6 References.

1. Ainslie J.P. Paraplegia due to spontaneous extradural or subdural hemorrhage. Br J Surg (1958) 45:565-567.
2. Alderman D.B. Extradural spinal-cord hematoma: report of a case due to dicoumarol and review of the literature. N Engl J Med (1956) 255:839-842.
3. Amyes E.W., Vogel P.J. and Raney R.B. Spinal cord compression due to spontaneous epidural hemorrhage: report of three cases. Bull Los Angeles Neurol Soc (1955) 20:1-8.
4. Amyot R., Hardy J., Chagnon J. and Lefrançois R. Hematome epidural rachidien spontané chez un enfant de 22 mois. Union Med Canad (1969) 98:1302-1306.
5. Aring C.D. Neurological pathological conference. Dis Nerv Syst (1952) 13:53-60.
6. Avrahami E., Tadmor R., Ram Z., Feibel M. and Itzhak Y. MR demonstration of spontaneous acute epidural hematoma of the thoracic spine. Case reports. Neuroradiology (1989) 31:89-92.
7. Bain W. A case of haematorrachis. Br Med J (1897) 2:455.
8. Banerjee T. and Meagher J.N. Spontaneous spinal epidural hemorrhage: case report. South Med J (1974) 67:858-861.
9. Bareño E.U. and Schlamich A. L'hématome épidural cervical spontané. A propos d'un cas. Neurochirurgie (1987) 33:66-70.
10. Beatty R.M. and Winston K.R. Spontaneous cervical epidural hematoma. A consideration of etiology. J Neurosurg (1984) 61:143-148.
11. Bidzinski J. Spontaneous spinal epidural hematoma during pregnancy: case report. J Neurosurg (1966) 24:1017.
12. Binnert D., Thierry A., Michiels R., Solchot P. and Perrin M. Présentation d'un nouveau cas d'hématome extra-dural rachidien spontané observé au cours d'un accouchement. J Med Lyon (1971) 1307-1309.
13. Bollar Zabala A., Gelabert Gonzalez M., Garcia Allut A. and Martinez Rumbo R. Spontaneous spinal epidural hematoma diagnosed by MRI. J Neurosurg Sci (1988) 32:153-155.

14. Bruyn G.W. and Bosma N.J. Spinal extradural hematoma. In: Vinken en Bruyn, eds. Handbook of clinical neurology (1976). Vol.26. Amsterdam: North-Holland publishing company 1-30.
15. Busse O., Hamer J., Paal G. and Piotrowski W. Spontane epidurale spinale Hämatome wahrend und nach Antikoagulantienmedication. Nervenarzt (1972) 43:318-322.
16. Caldarelli M., Di Rocco C. and La Marca F. Spontaneous spinal epidural hematoma in toddlers: description of two cases and review of the literature. Surg Neurol (1994) 41:325-329.
17. Calliauw L., Dhara M., Martens F. and Vannerem L. Spinal epidural hematoma without lesion of the spine. Report of 4 cases. Clin Neurol Neurosurg (1988) 90(2):131-136.
18. Carrea R., Girado M. and Eurnekian A. Hematoma cronico epidural y subdural espinal. Medicina (1954) 6:379-397.
19. Carroll K.L. The epidural defect on myelography: some unusual causes. Henry Ford Hosp Med (1981) 29(2):87-92.
20. Cecotto C., Ruberti R. and Benedetti A. Paraplegia acuta da ematoma epidurale spinale spontaneo. Rev Anat Patol (1961) 19:351-357.
21. Chavany J.A., Taptas J.N. and Pecker J. A propos d'une varieté exceptionelle d'hematorachis: l'hematome epidurale spontané. Presse Med (1949) 62:869-871.
22. Chen H.-H. and Hung C.-C. Spontaneous spinal epidural hematoma: report of seven cases. J Formosan Med Assoc (1992) 91:214-218.
23. Chipault A. Rapports des apophyses épineuses avec la moelle les racines médullaires et les méninges. Thesis. Paris, Battaille et cie. (1894).
24. Chou D.-Y., Guang-Shiung C. and Yang D.-Y. Spontaneous cervical epidural hematoma presented with Brown-Sequard syndrome: a case report. Kaosiung J Med Sci (1988) 4:58-62.
25. Combelles G., Blond S., Lesion F., Bousquet C., Rousseaux M. and Christiaens J.L. Hématomes extra-dureaux rachidiens sans lésion osseuse traumatique. A propos de 9 cas. Neurochirurgie (1983) 29:417-422.
26. Cooper D.W. Spontaneous spinal epidural hematoma. Case report. J Neurosurg (1967) 26:343-345.
27. Correa A.V. and Breasley B.A.L. Spontaneous cervical epidural hematoma with complete recovery. Surg Neurol (1978) 10:227-228.
28. Costabile G., Husag L. and Probst C. Spinal epidural hematoma. Surg Neurol (1984) 21:489-492.
29. Crisi G., Colombo A., Scarpa M., Falasca A. and Angiari P. Gadolinium-DTPA-enhanced MR imaging in the diagnosis of spinal epidural haematoma. Neuroradiology (1990) 32:64-66.
30. Cromwell L.D., Kerber C. and Ferry P.C. Spinal cord compression and hematoma: an unusual complication in a hemophiliac infant. Am J Roentgenol (1977) 128:847-849.
31. Cube H.M. Spinal extradural hemorrhage. J Neurosurg (1962) 19:171-172.

32. Dauch W.A. Spinale Epidural-hämatome bei Kindern und Jugendlichen. Neurochirurgia (1986) 29:83-89.
33. Davies K.G. and Weeks R.D. Acute spontaneous spinal epidural haematoma with temporary resolution. Br J Neurosurg (1992) 6:63-66.
34. Dawson B.H. Paraplegia due to spinal epidural hematoma. J Neurol Neurosurg Psychiat (1963) 26:171-173.
35. Delmas M., Senegas J., Longy M., Le Bras M. and Moretti G. Hématome extradural rachidien et lombo sciatique atypique. La Nouv Presse Méd (1980) 9:1172.
36. Demierre B., Unger P.-F. and Bongioanni F. Sudden cervical pain: spontaneous spinal epidural hematoma. Am J Emerg Med (1991) 9:54-56.
37. Devadiga K.V. and Gass H.H. Chronic lumbar extradural hematoma simulating disc syndrome. J Neurol Neurosurg Psychiat (1973) 36:255-259.
38. Di Lorenzo N., Rizzo A. and Fortuna A. Spontaneous spinal epidural hematoma: preoperative diagnosis by MRI. Clin Neurol Neurosurg (1990) 92:357-359.
39. Elsberg C.A. and Dyke C.G. Diagnosis and localization of tumors of spinal cord by means of measurements made on X-ray films of vertebrae, and the correlation of clinical and X-ray findings. Bul Neurol Inst New York (1936) 3:359.
40. Enomoto T., Maki Y., Nakagawa K. and Nose T. Spontaneous spinal epidural hematoma. Report of a case. Neurol Surg (Jap) (1980) 8:875-880.
41. Epstein B.S. The spinal cord, in: Epstein (ed): The spine. A radiological text and atlas. Philadelphia: Lea & Febiger (1976).
42. Farias J.P., Almeida Lima J. and Lobo Antunes J. Subacute cervical epidural hematomas. Surg Neurol (1994) 42:414-416.
43. Fischbach von R. and Kollar W.A. Spontane spinale epidurale Blutung unter Antikoagulantion. Wien Med Woch (1972) 19/20:5-6.
44. Flaschka G., Sutter B., Ebner F., Klein G.E. and Tilz G. Das spinale Epiduralhämatom. Langzeitergebnisse von 4 eigenen Fällen. Nervenarzt (1990) 61:629-633.
45. Fliedner E. and Sartor K. Ueber ein akutes spontanes spinales epidurales Hämatom. Fallbericht. Nerveratzt (1977) 48:603-607.
46. Foo D. and Rossier A.B. Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas. Surg Neurol (1981) 15:398-401.
47. Foo D., Chang Y.C. and Rossier A.B. Spontaneous spinal epidural hemorrhage, anterior cord syndrome, and familial vascular malformation: case report. Neurology (NY) (1980) 30:308-311.
48. Fosselle E. and Vander Eecken H. Idiopathic spinal epidural haematoma. Lancet (1978) 2:42.
49. Franscini L., Ballmer P.E., Sturzenegger M., Beer H., Tuncdogan E. and Straub W. Arch Intern Med (1994) 154:2769-2771.
50. Freger P., Meneses M., Creissard P., Vanier J.-P., Tadie M. and Godlewski J. L'hématoma épidural intrarachidien chez l'hémophile. Neurochirurgie (1986) 32:486-489.
51. Galzio R.J., Zenobii M. and D'Ecclesia G. Spontaneous spinal epidural hematoma: report of a case with complete recovery. Surg Neurol (1980) 14:263-265.

52. Gauthier G. L'hematome extradural rachidien sans fracture de la colonne: revue de 56 cas verifies. Psychiat Neurol (Basel) (1963) 146:149-175.
53. Ghanem M.B. and Ivan L.P. Spontaneous spinal epidural hematome in an eight year old boy. Neurology (Minneap) (1978) 28:829-832.
54. Giagheddu M., Napoleone F. and Pasolini F. Gli ematomi extradurali spinali: rassegna bibliografica e descrizione di un nuovo caso clinico. Minerva Neurochir (1964) 8:37-40.
55. Girard P.-L., Cournil C. and Dumas M. Hématome épidural rachidien spontané. Bull Soc Méd Afr Noire Lang Franç (1975) 20:256-265.
56. Gold M.E. Spontaneous spinal epidural hematome. Radiology (1963) 80:823-828.
57. Goulon M., Nouailhat F., Julian H., Barois A., Gajdos P., Babinet P. and Rapin M. Hématome extradurale cervicale spontané, responsable d'une hémiplégie spinale: guérison après intervention. Soc Med Hop Paris (1967) 118:969-979.
58. Grollmus J. and Hoff J. Spontaneous spinal epidural hemorrhage: good results after early treatment. J Neurol Neurosurg Psychiat (1975) 38:89-90.
59. Gruszkiewicz J., Doron Y., Lemberger B., Borovich B. and Feinsod M. Acute spinal extradural hematoma. Neurochirurgia (1987) 30:88-90.
60. Guthkelch A.N. Hemangiomas involving the spinal epidural space. J Neurol Neurosurg Psych (1948) 11:199-210.
61. Hack I., Cademartori M.S., Mamani R.S., Beltrame C.M. and Cademartori C.G. Hematoma epidural espinal espontaneo durante a gravidez. Registro de um caso. Arq Neuropsiq (1984) 42:41-44.
62. Harris M.E. Spontaneous epidural spinal hemorrhage. Am J Roentgenol (1969) 105:383-385.
63. Haykal H.A., Wang A.-M., Zamani A.A. and Rumbaugh C.L. Computed tomography of spontaneous acute cervical epidural hematoma. J Comput Assist Tomogr (1984) 8:229-231.
64. Hehman K. and Norrel H. Massive chronic spinal epidural hematoma in a child. Am J Dis Child (1968) 116:308-310.
65. Herrmann E., Lorenz R. and Vogelsang H. Zur diagnostik der spinalen epiduralen Hämatome und Abszesse. Radiologe (1965) 5:504-508.
66. Hirai H., Takamatsu H., Takahashi H. and Matsumoto N. A case of spontaneous spinal epidural hematoma. Rinsho Shinkeigaku (1970) 10:145-150.
67. Horne J.G. and Muller P. Spontaneous spinal extradural hematoma. Can J Surg (1977) 20:379-384.
68. Houtteville J.-P., Lechevalier B., Godard F., Blet J.N., Charrieras C. and Laschon M.C. Hématome épidural dorso-lombaire secondaire à un traitement anticoagulant. Nouv Presse Med (1975) 4:3195-3196.
69. Huff J.S. Spinal epidural hematoma associated with cocaine abuse. Am J Emerg Med (1994) 12:350-352.
70. Jackson F.E. Spontaneous spinal epidural hematoma coincident with whooping cough: case report. J Neurosurg (1963) 20:715-717.
71. Jackson R. Case of spinal apoplexy. Lancet (1869) 2:5-6.

72. Jacobson I., Maccabe J.J., Harris P. and Dott N.M. Spontaneous spinal epidural hemorrhage during anticoagulant therapy. Br Med J (1966) 1:522-523.
73. Jacquet G., Godard J. and Steimle R. Hématoma extra-dural rachidien spontané. A propos d'une localisation cervicale. Est Med (1990) 10:121-124.
74. Jellinger K. Neuropathology of cord injuries, in: Vinken en Bruyn (eds): Handbook of clinical neurology. Vol.25. Amsterdam: North-Holland Publishing Co. (1976) 72-76.
75. Jones R.K. and Knighton R.S. Surgery in hemophiliacs with special reference to the central nervous system. Ann Surg (1956) 144:1029-1034.
76. Joseph A.P. and Vinen J.D. Acute spinal epidural hematoma. J Emerg Med (1993) 11:437-441.
77. Jost F. and Siehr U. Spinales epidurales Hämatom unter Antikoagulantienbehandlung. Dtsch Med Wochenschr (1970) 95:2350.
78. Kanai M., Egashira M., Murata T., Iwai Y. and Seka M. Spontaneous spinal epidural hematoma diagnosed by MRI: a case report. No Shinkei Geka (1989) 17:1073-1076.
79. Kaplan L.I. and Denker P.G. Acute non-traumatic spinal epidural hemorrhage. Am J Surg (1949) 78:356-361.
80. Kato S., Seki H. and Koshu K. Acute cervical spinal epidural hematoma with spontaneous resolution. Case report. Neurol Med Chir (Tokyo) (1994) 34:23-26.
81. Khatib R. and Cook A.W. Spontaneous spinal epidural hematoma. NY State J Med (1966) 66:989-992.
82. Koyama T., Igarashi S., Hanakita J. and Handa J. Das spinale epidurale Hämatom. Neurochirurgia (1982) 25:11-13.
83. Krolick M.A. and Cintron G.B. Spinal epidural hematoma causing cord compression after tissue plasminogen activator and heparin therapy. South Med J (1991) 84:670-671.
84. Kuchiwaki M.H., Furuse M. and Nakitani Y. A case of spontaneous spinal epidural hematoma. Clin Neurol (Tokyo) (1973) 13(5):296-300.
85. Lawton M.T., Porter R.W., Heiserman J.E., Jacobowitz R., Sonntag V.K.H. and Dickman C.A. Surgical management of spinal epidural hematoma: relationship between surgical timing and neurological outcome. (1995) J Neurosurg 83:1-7.
86. Lazorthes G., Boulard C., Espagno J., Arbus L. and Lazorthes Y. L'hematome epidural rachidien spontané (3 observations). Rev Med Toulouse (1968) 4:475-477.
87. Lazorthes G., Gouaze A., Zadeh J.O., Santini J.J., Lazorthes Y. and Burdin P. Arterial vascularization of the spinal cord. Recent studies of the anastomotic substitution pathways. J Neurosurg (1971) 35:253-262.
88. Lecuire J., Léger G., Goutelle A., Gentil J.P. and Dechaume J.P. Hématorachis spontané avec paraplégie opéré et guéri. Lyon Chir (1966) 62:67-72.
89. Lepoire J., Tridon P., Montaut J. and Germain F. L'hématome extra dural rachidien spontané. Neurochirurgie (1959) 7:298-313.
90. Levitan L.H. and Wiens C.W. Chronic lumbar extradural hematoma: CT findings. Radiology (1983) 148:707-708.

91. Licata C., Zoppetti M.C., Perini S.S., Bazzan A., Gerosa M. and Da Pian R. Spontaneous spinal haematomas. Acta Neurochir (Wien) (1988) 95:126-130.
92. Liebeskind A.L., Schwartz K.S., Coffey E.L. and Beresford H.R. Spinal epidural hematoma with delayed appearance of neurological symptoms. Neuroradiology (1975) 8:191-193.
93. Lin Th. Paraplegia caused by epidural hemorrhage of the spine. J Int Coll Surg (1961) 36:742-749.
94. Lizuka J. Intracranial and intraspinal haematomas associated with anticoagulant therapy. Neurochirurgia (1972) 1:15-25.
95. Locke G.E., Giorgio A.J., Biggers S.L., Johnson A.P. and Salem F. Acute spinal epidural hematoma secondary to aspirin-induced prolonged bleeding. Surg Neurol (1976) 5:293-296.
96. London G.W., McKeever P.E. and Wiederholt W.C. Spontaneous spinal epidural hematoma in alcoholism. Ann Int Med (1974) 81(2):266-267.
97. Lord G.M. and Mendoza N. Spontaneous spinal epidural haematoma: a cautionary tale. Arch Emerg Med (1993) 10:339-342.
98. Lougheed W.M. and Hoffman H.J. Spontaneous spinal extradural hematoma. Neurology (1960) 10:1059-1063.
99. Lowrey J.J. Spinal epidural hematomas. Experiences with three patients. J Neurosurg (1959) 16:508-513.
100. Lusted L.B. and Keats T.E. Atlas of roentgenographic measurement. Chicago, Year Book Medical Publishers Inc. (1978) 129-132.
101. MacFarlane R.G., Mallam P.C., Witts L.J., Bidwell E., Biggs R., Fraenkel G.J., Honey G.E. and Taylor K.B. Surgery in haemophilia. The use of animal anti-haemophilic globulin and human plasma in thirteen cases. Lancet (1957) 251-259.
102. Machado de Almeida G., Bianco E. and Kasunori Shibata M.. 5ncapsulated spinal epidural hematoma in an octogenarian. Surg Neurol (1989) 32:394-395.
103. Mahieu X., Kridelka F., Pintiaux A., Hans P. and Brichant J.F. Hématome extradural cervical spontané de la femma enciente. J Gynecol Obstet Biol Reprod (1994) 23:99-102.
104. Major O., Sipos L., Czirják S., Benoist Gy., Horváth M. and Pásztor E. Spontaneous spinal epidural haematomas. Acta Neurochir (Wien) (1991) 11:40-42.
105. Markham J.W., Lynge H.N. and Stahlman G.E.B. The syndrome of spontaneous spinal epidural hematoma. Report of three cases. J Neurosurg (1967) 26:334-342.
106. Marmey G., Doyon D. and David Ph. Diagnostic IRM d'un hématome épidural cervical. A propos d'une observation. J Radiol (1990) 71:549-553.
107. Matsumae M., Shimoda M., Shibuya N., Ueda M., Yamamoto I. and Sato O. Spontaneous cervical epidural hematoma. Surg Neurol (1987) 28:381-384.
108. Mattle H., Sieb J.P., Rohner M. and Mumenthaler M. Nontraumatic spinal epidural and subdural hematomas. Neurology (1987) 37:1351-1356.
109. Maxwell G.M. and Puletti F. Chronic spinal epidural hematoma in a child: case report. Neurology (1957) 7:597-600.
110. Mayer J.A. Extradural spinal hemorrhage. Case report. Can Med Ass J (1963) 89:1034-1037.

111. McQuarrie I.G. Recovery from paraplegia caused by spontaneous spinal epidural hematoma. Neurology (1978) 28:224-228.
112. Mérienne L., Herreman G., Godeau P. and Couteaux B. Les hématomes extra-dureaux rachidiens non traumatiques. Gaz Med France (1980) 80:5589-5600.
113. Mishima K., Aritake K., Morita A., Miyagawa N., Segawa H. and Sano K. A case of acute spinal epidural hematoma in a patient with antiplatelet therapy. No Shinkei Geka (1989) 17:849-853.
114. Miyasaka Y., Morii S., Takiga H., Ohwada T. and Yada K. A case of acute spinal epidural hematoma due to a hemorrhage of spinal epidural arteriovenous malformations. No Shinkei Geka (1977) 5:655-661.
115. Mohazab H., Langer B. and Spigos D. Spinal epidural hematoma in a patient with lupus coagulopathy: MR findings. Case report. AJR (1993) 160:853-854.
116. Mracek Z. Spontaneous spinal epdirual hematoma. Experiences with four patients and a review of the literature. Zbl Neurochir (1980) 41:19-30.
117. Müller H., Schramm J., Roggendorf W. and Brock M. Vascular malformations as a cause of spontaneous spinal epidural hematoma. Acta Neurochir (Wien) (1982) 62:-297-305.
118. Murata T., Ohhata K., Tsujikawa S., Sumimoto T., Kitano S., Shirahata N., Hakuba A. and Choi S.K. A case of spontaneous spinal epidural hematoma presenting Brown-Séquard syndrome. No Shinkei Geka (1984) 12:1195-1200.
119. Mustafa M.H. and Bernstein R.A. Spontaneous spinal epidural hematoma, Brown-Séquard syndrome and factor Xl deficiency. Ann Int Med (1987) 106:477-478.
120. Mustafa M.H. and Gallino R. Spontaneous spinal epidural hematoma causing cord compression after streptokinase and heparin therapy for acute coronary artery occlusion. South Med J (1988) 81:1202-1203.
121. Nagel M.A., Taff I.P., Cantos E.L., Patel M.P., Maytal J. and Berman D. Spontaneous spinal epidural hematoma in a 7-year-old girl. Diagnostic value of magnetic resonance imaging. Clin Neurol Neurosurg (1989) 91:157-160.
122. Nehls D.G., Shetter A.G., Hodak J.A. and Waggener J.D. Chronic spinal epidural hematoma presenting as lumbar stenosis: clinical, myelographic and computed tomographic features. A case report. Neurosurgery (1984) 14:230-233.
123. Nichols P. and Manganiello L.O.J. Extradural hematome of the spinal cord. Case reports. J Neurosurg (1956) 13:638-640.
124. Ogawa T., Abe S., Nakahara S., Sekino H. and S. Tani. Spontaneous extradural hematoma localized in the ventral side of the thoracic spinal cord. Case report. No Shinkei Geka (1985) 13:439-443.
125. Oldenkott P. and Driesen W. Spontanes epidurales Hämatom im Burstwirbelkanal wärhend Antikoagulantienlangzeitbehandlung. Med Welt (1966) 17:305-307.
126. Packer N. and Cummins B.H. Spontaneous epidural hemorrhage: a surgical emergency. Lancet (1978) 18:356-358.
127. Paliard P., Mottin J., Goutelle A., Ducluzeau R., Perrot E. and Bouletreau S. Hématoma extradural médullaire au cours d'un traitement anticoagulant. Lyon Med (1967) 37:395-399.

128. Panitz K., Neundörfer B., Taglieber U. and Susemihl D. Spontanes spinales epidurales Hämatom nach Totgeburt bei Schwangerschaftshepatose. Münch Med Wochenschr (1975) 117(9):329-332.
129. Parman S.C. Spontaneous spinal epidural hematoma. Ann Emerg Med (1980) 9:368-370.
130. Pear B.L. Spinal epidural hematoma. Am J Roentgenol Radium Ther Nucl Med (1972) 115:155-164.
131. Penar P.L., Fisher D.K., Goodrich I., Bloomgarden G.M. and Robinson F. Spontaneous spinal epidural hematoma. Int Surg (1987) 72:218-221.
132. Pendl G., Gangberger J.A. and Horcajada J. Das spinale epidurale Hämatom. Acta Neurochir (1971) 24:207-217.
133. Peserico L. and Svien H.J. Spontaneous epidural hematoma of the cervical region. Proc Mayo Clinic (1959) 34:309-312.
134. Petrov V., Collignon J. and Stevenaert A. Hématomes spineaux extra-duraux et sous-duraux au cours d'un traitement par anticoagulants. Acta Neurol Belg (1979) 79:398-408.
135. Phillips T.W., Kling T.F. and McGillicuddy J.E. Spontaneous ventral spinal epidural hematoma with anterior cord syndrome: report of a case. Neurosurgery (1981) 9:440-443.
136. Plagne R. L'hématome extra-dural rachidien non traumatique (Hématome épidural spontané. Thèse. Clermont-Ferrand, G. de Bussac (1961) pp.32-35.
137. Pointud Ph., Uzzan B. and Stilhart B. Syndrome de la queue de cheval par hématome épidural rachidien au cours d'un traitement anticoagulant. Nouv Presse Med (1979) 8:4047.
138. Pommé B., Montrieul B. and Girard J. Hématome extradural lombaire spontané. Soc Fr Neurol (1959) 101:69-71.
139. Posnikoff J. Spontaneous spinal epidural hematoma of childhood. J Pediatr (1968) 73:178-183.
140. Quequet P.M. and Mizon R.J.P. Hématomes épidureaux rachidiens non traumatiques (2 Cas): Apport du scanner X. Rev Neurol (Paris) (1987) 143:143-146.
141. Ramsey R. and Doppman J.L. The effects of epidural masses on spinal cord blood flow. An experimental study in monkeys. Radiology (1973) 107:99-103.
142. Rao B.D., Rao K.S., Subrahmanian M.V. and Raghava Reddy M.V. Spinal epidural hemorrhage. Br J Surg (1966) 53:649-650.
143. Rathe S. Spontaneous spinal epidural hemorrhage. J Indian Med Assoc (1969) 52:222-223.
144. Rebello M.D. and Dastur H.M. Spinal epidural hemorrhage. Neurology (India) (1966) 14:135-145.
145. Reddy D.R. and Rao B.D. Some observations on spinal epidural hematoma. Indian J Med Sci (1972) 26:437-439.
146. Robertson W.C. Jr., Lee Y.N. and Edmonson M.B. Spontaneous spinal epidural hematoma in the young. Neurology (1979) 29:120-122.

147. Rose K.D., Croissant P.D., Parliament C.F. and Levin M.B. Spontaneous spinal epidural hematoma with associated platelet dysfunction from excessive garlic ingestion: a case report. Neurosurgery (1990) 26:880-882.
148. Rothfus W.E., Chidid M.K., Deed Z.L., Abla A.A., Maroon J.C. and Sherman R.L. MR imaging in the diagnosis of spontaneous spinal epidural hematomas. J Comput Assist Tomogr (1987) 11:851-854.
149. Russman B.S. and Kazi K.H. Spinal epidural hematoma and the Brown-Séquard syndrome. Neurology (1971) 21:1066-1068.
150. Santa M., Sulla I. and Fagul'a J. Spontaneous spinal epidural hematoma. Zentbl Neurochir (1990) 51:164-165.
151. Sasaki M., Ishida K., Nogaki H. and Nagao T. Idiopathic thoraco-lumbar spinal epidural hematoma. Case report and review of literature. No Shinkei Geka (1987) 15:537-541.
152. Sawin P.D., Traynelis V.C. and Follett K.A. Spinal epidural hematoma following coronary thrombolysis with tissue plasminogen activator. Report of two cases. J Neurosurg (1995) 83:350-355.
153. Scharfetter F. Das spontane spinale epidurale Hämatom in der Differentialdiagnose des akuten spinalen Insultes. Dtsche Med Wochenschr (1972) 1:13-16.
154. Scheil F., Larkamp U. and Mutharoglu U. Das spontane spinale epidurale Hämatom. Neurochirurgia (1990) 33:45-49.
155. Schicke R. and Seitz D. Spinales epidurales Hämatom unter Antikoagulantientherapie. Dtsche Med Wochenschr (1970) 6:275-277.
156. Schiffer J., Razon N., Milo R., Harish Z. and Avidan D. Spinal epidural hematoma. Posttraumatic in ankylosing spondylitis. Orthopaedic Rev (1984) 13:592-595.
157. Schultz E.C., Johnson A.C., Brown C.A. and Mosberg W.H. Paraplegia caused by spontaneous spinal epidural hemorrhage. J Neurosurg (1953) 10:608-616.
158. Scott B.B., Quisling R.G., Miller C.A. and Kindt G.W. Spinal epidural hematoma. JAMA (1976) 235:513-515.
159. Segelov J.N. Spinal epidural haematoma. A report of two cases. Pacific Med Surg (1967) 75:169-171.
160. Servadei F., Trevisan C., Bianchedi G. and Padovani R. Spontaneous dorsal epidural haematoma: usefulness of magnetic resonance imaging and importance of operative treatment even in cases with complete paraplegia. Acta Neurochir (Wien) (1987) 89:137-139.
161. Shen C.-C., Wang Y.-C., Yang D.-Y., Wang F.-H. and Chou D.-Y. Acute spontaneous (idiopathic) spinal epidural hematoma: four cases report and review of the literature. Acta Neurol Sinica (1992) 1:248-253.
162. Shen C.-C., Wang Y.-C., Yang D.-Y., Wang F.-H. and Shen B. B.-H. Brown-Séquard syndrome associated with Horner's syndrome in cervical epidural hematoma. Spine (1995) 20:244-247.
163. Shenkin H.A., Horn R.C. and Grant F.C. Lesions of the spinal epidural space producing cord compression. Arch Surg (1945) 51:125-146.
164. Simmons E.H. and Grobler L.J. Acute spinal epidural hematoma. J Bone Joint Surg (1978) 60:395-396.

165. Solero C.L., Fornari M. and Savoiardo M. Spontaneous spinal epidural hematoma arising from ruptured vascular malformation. Case report. Acta Neurochir (Wien) (1980) 53:169-174.
166. Soustiel J.F., Goldsher D. and Feinsod M. L'hématome épidural aigu rachidien circonferentiel. A propos d'un cas. Neurochirurgie (1988) 34:352-354.
167. Spanu G., Messina A.L., Rodriguez y Baena R., Introzzi G., Gaetani P. and Silvani V. Spinal epidural hematoma without vertebral fracture or dislocation. Report of two cases and a review of the literature. Riv Neurol (1987) 57:239-244.
168. Spurney O.M., Rubin S. and Wu W.Q. Spinal epidural hematoma during anticoagulant therapy. Report of two cases. Arch Intern Med (1964) 114:103-107.
169. Sreerama V., Ivan L.P., Dennery J.M. and Richard M.T. Neurosurgical complications of anticoagulant therapy. Can Med Assoc J (1973) 108:305-307.
170. Stanley P. and McComb J.G. Chronic spinale epidural hematoma in hemophilia A in a child. Pediatr Radiol (1983) 13:241-243.
171. Strain R.E. Spinal epidural hematoma in patients on anticoagulant therapy. Ann Surg (1964) 159:507-509.
172. Suzuki H., Yamamoto Y., Hoshino T. and Yoneda C. A case of spontaneous spinal epidural hematoma with spontaneous remission before surgery. No Shinkei Geka (1993) 21:1119-1123.
173. Suzuki N., Inoue H. and Abematsu N. Acute spontaneous spinal epidural hemorrhage in pregnancy. Nippon Seikeigeka Gakkai Zasshi (1977) 51(3):155-160.
174. Suzuki S., Aihara H., Hori S. and Suzuki J. Spontaneous spinal epidural hematoma; report of a case. No To Shinkei (1968) 20:478-483.
175. Svien H.J., Adson A.W. and Dodge H.W. Lumbar extradural hematoma. Report of a case simulating protruded disc syndrome. J Neurosurg (1950) 7:587-588.
176. Tarlov I.M. Spinal cord compression studies. Time limits for recovery after gradual compression in dogs. A.M.A. Arch Neurol Psych (1954) 71:588-597.
177. Tarlov I.M. and Herz E. Spinal cord compression studies. Outlook with compression paralysis in man. A.M.A. Arch Neurol Psych (1954) 72:43-59.
178. Tarlov I.M. and Klinger H. Spinal cord compression studies. Time limits for recovery after acute compression in dogs. A.M.A. Arch Neurol Psych (1954) 71:271-290.
179. Telerman-Toppet N., Moerman C. and Noterman J. L'hématome épidural spinal spontané, complication rare de la thérapeutique anticoagulante. Acta Neurol Belg (1972) 72:118-129.
180. Ter Spill H.W. and Tijssen C.C. Spinal epidural hematoma due to a vertebro-epidural hemangioma. Clin Neurol Neurosurg (1989) 91:91-93.
181. Tewari M.K., Tripathi L.N., Mathuriya S.N., Khandelwal N. and Kak V.K. Spontaneous spinal extradural hematoma in children. Report of three cases and a review of the literature. Child's Nerv Syst (1992) 8:53-55.
182. Tsai F.Y., Popp A.J. and Waldman J. Spontaneous spinal epidural hematoma. Neuroradiology (1975) 10:15-30.
183. Twerdy K., Plangger C. and Mayr U. Das spontane spinale epidurale Hämatom. Nervenarzt (1984) 55:96-98.

184. Umbach W. and Kunft H.-D. Vascular tumors of the spinal cord. In: Vinken en Bruyn, eds. Handbook of clinical neurology. (1976) Vol.20. Amsterdam: North-Holland publishing company. pp. 435-480.
185. Vallée B., Besson G., Gaudin J., Person H., Le Fur J.-M. and Le Guyader J. Spontaneous spinal epidural hematoma in a 22-month-old girl. J Neurosurg (1982) 56:135-138.
186. Vapalahti M. and Kuurne T. Acute paraplegia caused by a spontaneous extradural hematoma of the conus medullaris area. Acta Chir Scand (1975) 141:484-487.
187. Ventureyra E.C.G., Ghanem Q. and Ivan L.P. Spontaneous spinal epidural hematoma in a youngster. Childs Brain (1979) 5:103-108.
188. VerBrugghen A. Extradural spinal hemorrhage. Ann Surg (1946) 123:154-159.
189. Verhagen I.T.H.J. and ter Weeme C.A. Een patiënt met een cervicaal gelocaliseerd epiduraal hematoom als gevolg van antistollingsbehandeling. Ned Tijdschr Geneeskd (1986) 130:2134-2135.
190. Von Klossek H. and Hüller E. Zur problematiek der spontanen spinalen Epiduralhämatome. Zeitbl Neurochirurgie (1984) 45:116-123.
191. Watts C. and Porto L. Recognizing spontaneous spinal epidural hematoma. Geriatrics (1976) 97-98.
192. Weigert M. Akutes spinales epidurales Hämatom als Folge von Behandlung mit Antikoagulantien. Nervenarzt (1961) 32:85-89.
193. Whaley R.L. and Lindner D.W. Spontaneous spinal epidural hemorrhage associated with anticoagulant therapy. Report of a case. Grace Hosp Bull (1962) 40:27-32.
194. Williams C.E. and Nelson M. The varied computed tomographic appearances of acute spinal epidural haematoma. Clin Radiol (1987) 38:363-365.
195. Winer B.M., Horenstein S. and Starr A.M. Spinal epidural hematoma during anticoagulant therapy. Circulation (1959) 19:735-740.
196. Wittebol M.C. and Veelen van C.W.M. Spontaneous spinal epidural hematoma. Etiological considerations. Clin Neurol Neurosurg (1984) 86:265-270.
197. Yettou H., Vinikoff L., Marchal J.-C. and Auque J. L'hématome épidural rachidien spontané. A propos de 2 nouvelles observations. Neurochirurgie (1995) 41:58-61.
198. Yonekawa Y., Mehdorn M. and Nishikawa M. Spontaneous spinal epidural hematoma during pregnancy. Surg Neurol (1975) 3:327-328.
199. Yu Y.L., Chin K.F., Wen H.L. and Woo E. Spontaneous spinal epidural haematoma. Clin Neurol Neurosurg (1986) 88:131-134.
200. Zilkha A., Irwin G. A. and Fagelman D. Computed tomography of spinal epidural hematoma. AJNR (1983) 4:1073-1076.
201. Zouaoui A., Hirsch J.F., Ganzengel C., Pierre-Kahn A. and Renier D. Les accidents neurologiques de l'hémophilie chez l'enfant. Neurochirurgie (1980) 26:285-289.
202. Zuccarello M., Scanarini M., D'Avilla D., Andrioli G.C. and Gerosa M. Spontaneous spinal extradural hematoma during anticoagulant therapy. Surg Neurol (1980) 14:411-413.
203. Zupruk G.M. and Mehta Z. Brown-Séquard syndrome associated with posttraumatic cervical epidural hematoma: case report and review of the literature. Neurosurgery (1989) 25:278-280.

Chapter 4

THE SPONTANEOUS SPINAL EPIDURAL HEMATOMA.
A study of the etiology.

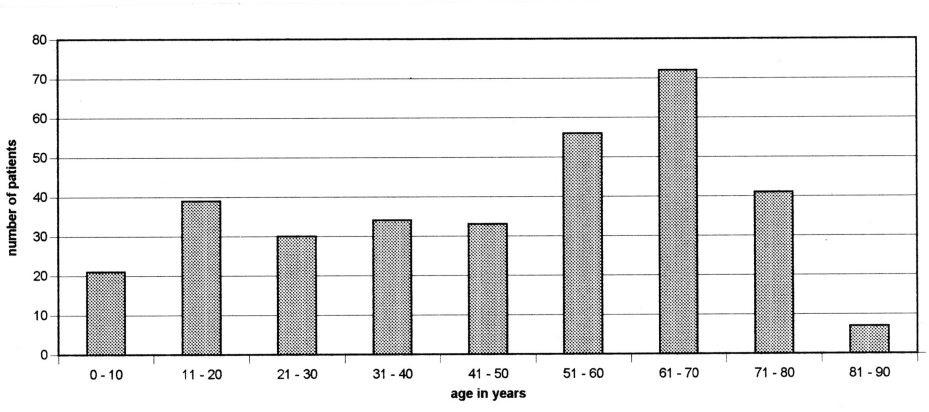
4.1 Introduction.

By definition, the etiology of the spontaneous spinal epidural hematoma (SSEH) is obscure. Since the initial description by Jackson (31) and Bain (4), many authors have reported their cases, contributing to the series of SSEH in the international medical literature. As possible etiological factors, minor trauma (14,19,29,61,65), whooping cough (31), sneezing (48,57), voiding (4), vomiting (34), lifting (15,25,30,36,50) (all assumed to produce an acute increase of the pressure in the spinal epidural veins), pregnancy (8,60,70), hypertension (1,39), atherosclerosis (1), anticoagulants (2,10,19,20,27,68,69) or bleeding diathesis (3,11,38,59) are mentioned. In other cases none of these factors were present (12,32,36,51). A vascular anomaly is thought to be an important source of SSEH (5,13,14,16-18,23,34,35,41,44,45,47,57,58,63). Nevertheless, in most postoperatively investigated hematomas no histological evidence for a vascular malformation was found. Other authors (1,3,26,32,36,37,48) have the opinion that the internal vertebral venous plexus is the major bleeding source. In contrast, Beatty and Winston (7) and Bareño and Schlamich (6) consider an arterial genesis of the SSEH as most likely. A large number of acute spinal epidural hematomas has been found to be spontaneous in origin (22,26,39,40,56). In the present chapter the literature on the etiology of the SSEH will be reviewed.

4.2 Patients and methods.

This study is based on the data that are described in the previous chapter (*Chapter 3*). Details of the patients are listed in *Table 3.1* (see *Chapter 3*). This data base concerned 136 females and 197 males, aged between 14 months and 90 years [mean 47.3 years]. The male/female ratio is 1.4:1. The age distribution is shown in *Figure 4.1.* The largest number of patients (50.8%) is aged between 50 and 80 years. Only few patients older than 80 years have been reported, possibly because of the small contribution of this age-group to the population.

Figure 4.1 Age distribution in 333 SSEH patients.



4.3 Results.

182 patients had no medical history. Hypertension was present is 39 patients and 20 patients were both hypertensive and adjusted on oral anticoagulants. A group of 69 patients had increased bleeding tendency due to the use of oral anticoagulants (57 patients), alcohol abuse (2 patient), hemophilia (9 patients) or M. Moschcowitz (1 patient). The history of the entire group of 333 patients is described in *Table 4.1.*

Table 4.1 History of 333 patients with SSEH.

History	Score
Healthy	182
Coagulopathies	
*iatrogenic (Ac)	57
*disease (Hemoph/Liver disease/Moschc)	12
Hypertension (Ht)	39
Both Ht and Ac	20
Systemic disease (SLE/DM/RA/etc.)	7
Pregnancy	7
Cardiovascular disaese	2
Malignancy	2
Intoxication	1
Toxicosis	1
Whooping cough	1
Resp. disease	1
Fam. hemangioma	1
Total	333

The relationship between the age and the existence of hypertension and/or the use of anticoagulants is described in *Table 4.2* (n = 116). Hypertension was present in 39 patients. Fifty seven patients used oral anticoagulants and 20 patients were hypertensive as well as adjusted on oral anticoagulants.

Table 4.2 Age related to hypertension and/or the use of anticoagulants in 116 out of 333 patients with SSEH.

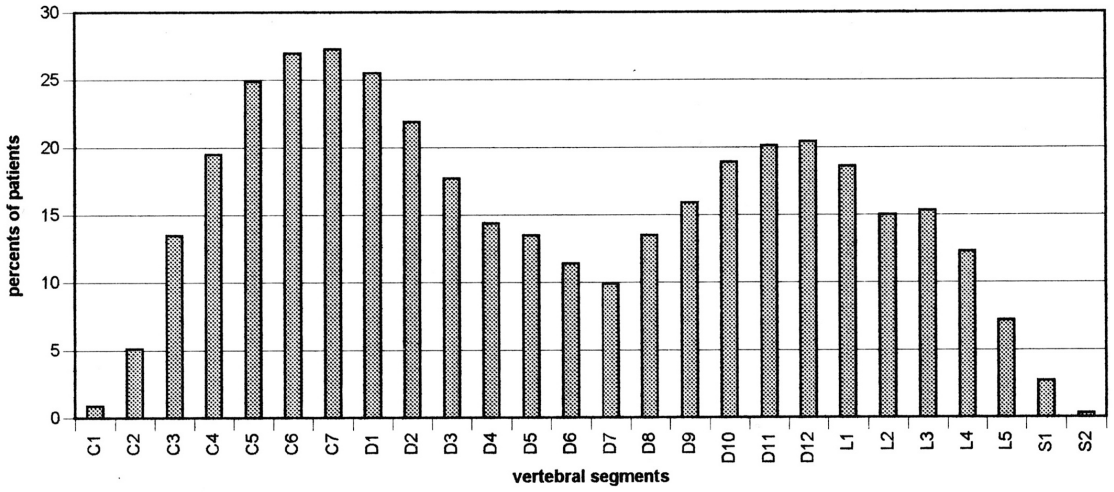
Age(yr)	Hypertension	Anticoagulants	Both	Total
31-40		3	2	5
41-50	1	16	4	21
51-60	10	12	3	25
61-70	19	19	10	48
71-80	9	6	1	16
81-90		1		1
Total	39	57	20	116

In 328 cases (98%) the hematoma was confirmed by operation and in five cases after autopsy. The predominant site of the SSEH was posterior (55%), or posterolateral (37%) in the spinal epidural space. Only two hematomas (0.6%) were located laterally, ten (3%) hematomas anteriorly and 15 hematomas were circular (4.5%).

The SSEHs were ranged according to the spinal segments involved. The segmental distribution of SSEH within the spinal canal is reproduced in *Figure 4.2.* The total number of segments involved is 1309, resulting in an avarage of 3.9 segments per patient.
Hematomas at the levels C5 down to and including T2 comprise one third (32.2%) of the

total number of segments. A second peak appeared at the thoracolumbar area (T10 - L1). In patients with a coagulopathy, the avarage number of segments involved is 4.5 per patient [403 segments in 89 patients], versus 3.7 segments in the remaining group of patients [906 segments in 244 patients].

Figure 4.2 Segmental distribution in 333 SSEH patients.



Histopathological investigation after operation or autopsy was performed in 176 cases (52.8%). Only in 18 patients (5.4%) this showed a more or less defined "vascular anomaly". These findings are listed separately and specified in detail in *Table 4.3.* According to the histological accounts, in eight patients a (hem)angioma was diagnosed and in ten patients an unspecified "arterial and/or venous malformation" was found.

Table 4.3 Summary of 18 patients with abnormal findings after histopathological examination in SSEH.

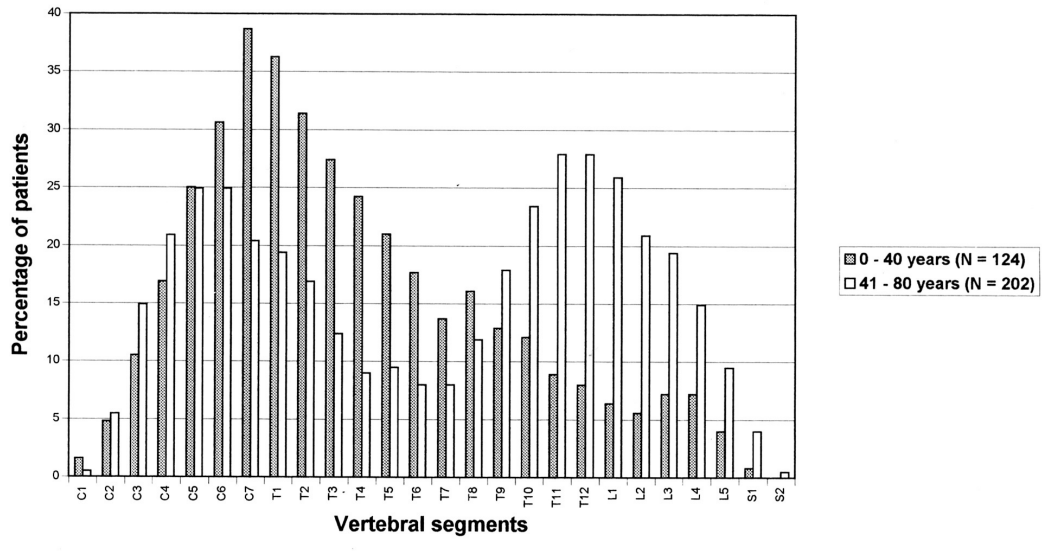
Author	Sex	Age	Level	Operative findings	Histopathology
Cube	M	29	C6-T1	two angiomatous lesions	dilated vascular channels; ***hemangioma***
Dawson	F	19	T2-T6	distended venous channels	vascular nodule, vascular channels; ***venous angioma***
Dawson	M	15	L2-L5	infected hematoma with with tangled bloodvessels	vascular nodule with vascular channels; ***angioma***
Flaschka	F	27	T2-T4	angiomatous lesion	***arteriovenous angioma***
Foo	M	33	C2-C7	hematoma	arteries and veins; ***vascular malformation***
Herrmann	M	38	T4-T9	enlarged veins	unclassified; ***angioma or venectasia***
Koyama	F	18	T1	abnormal vessels	channels with many veins and capillaries; ***venous angioma***
Lougheed	M	33	T3-T6	profuse bleeding	numerous veins with little muscle and thin fibrous walls; ***vascular malformation***
Mayer	F	17	C7-T1	clot attached to fat and vessels	dilated vessels; ***angioma***
Miyasaka	M	47	T1-T3	numerous dilated epidural vessels	***arteriovenous malformation***
Müller	F	71	C5-C7	organized clot	arteries and veins; ***arteriovenous angioma***
Müller	M	65	T5-T10	venous convolution	***vascular malformation***
Müller	F	53	T9-T11	unknown	pathological veins; ***vascular malformation***

Table 4.3 (Continued).

Author	Sex	Age	Level	Operative findings	Histopathology
Packer	F	13	C7-T7	abnormal coiled vessels over lamina D4-D6	clotted blood, 3-4 arteries; ***vascular malformation***
Packer	M	17	T9	abnormal vessels	multiple thick channels; ***arteriovenous malformation***
Penar	F	54	T1-T4	hematoma	small fragments of vessels showing ectasia and thinning of the wals; ***vascular anomaly***
Scheil	F	37	T8-T11	hematoma	***hemangioma***
Solero	M	38	C5-T1	bleeding epidural vessels	thick-walled venous vessels; ***venous malformation***

The relationship between level, age (in decades) and sex was analyzed. There appeared to be an equal distribution between both sexes, according to age and level. On the contrary, the segmental distribution showed to depend strongly on the age. The localization of a SSEH at the lower thoracic and lumbosacral segments appeared to be an exception, at ages below 40 years (n = 124). This phenomenon is exemplified in *Figure 4.3*, as well as the segmental distribution in the patients aging between 40 and 80 years (n = 202). Seven patients older than 80 years were left out. The interval chosen (40 years), results from the remarkable change in the level distribution that can be observed after the fourth decade. This suggests the existence of two separate groups among the series of SSEH.

Figure 4.3 Segmental distribution related to age in SSEH.



4.4 Discussion.

In the past decades several studies have been published on the spinal epidural hematoma (9,22,24,40,51,63,67). The present review of 333 patients, strictly concerning SSEH, pointed out some differences in comparison to the findings of other studies. Some authors (22) included patients with (non-spontaneous) spinal epidural hemorrhage after epidural anaesthesia, lumbar puncture or spinal tumors. In other articles documentation

was incomplete, which made critical evaluation difficult (67).

With regard to the age-distribution and sex-ratio, the discussion will be brief. The data suggest that the SSEH occurs at every age, and is more frequent in males (1.4 : 1). Some authors (9,17,33) made a differentiation between children (0 - 18 years) and adults. However, no arguments were given to support this distinction. The number of SSEH in the present series is highest between the age of 50 and 80 years (50.8%). Many authors came to this conclusion (9,13,17,22,63,67), but up to this moment the significance of this observation is unclear.

4.4.1 Medical history.

In quite a number of reports, arterial hypertension and anticoagulant therapy are mentioned as important pathogenetic factors (1,2,33,39,42,46,48,68,69). Ainslie (1) considers that the increase of bleedings after the age of 50 years is related to the coexistence of hypertension and the use of anticoagulants. Some others (9,55) doubt this strongly. According to the histories of the patients in the present series, indeed a relationship is suggested between the occurrence of a SSEH and the coexistence of hypertension and/or the use of anticoagulants. Of 195 patients aging between 30 and 70 years, 99 (50.8%) had cardiovascular problems, from which 46 patients (23.6%) suffered hypertension (*Table 4.2*). However, comparison of these findings with the data of the Hypertension Detection and Follow-up Program Cooperative Group (1977) makes it obvious that there is no difference between the percentage of hypertension in our group and the percentage of hypertensive persons (aging 30-69 years) in a population of 158,906 individuals (25.3%) that underwent home-screening of the blood pressure (31). Therefore, we think that conclusions about a causal relationship between hypertension and SSEH can not be drawn. The role of anticoagulants likewise seems uncertain, but we have no data to support this idea. As could be expected, the average number of segments that are involved appeared to be higher in patients with coagulopathies.

4.4.2 Bleeding source.

The etiology of the SSEH has been discussed by many authors. Some of them support the theory of the epidural hemorrhage being caused by a rupture of a spinal epidural vein in the venous plexus encircling the spinal dura. They postulate the rupture of a weakened vessel in a pre-existing abnormal epidural venous plexus as the basic pathologic process (5,17,26,34) and regard the internal vertebral venous plexus as a "locus minoris resistentiae" (25,55).

Liebeskind (37) stated that the cervico-thoracic and the thoraco-lumbar junctions are the sites of least resistance in the normal curved spine, suggesting mechanical factors to be of etiological and localizational importance. Indeed, in our series the epidural hematomas are most common at these segments.

Beatty and Winston (7) assumed an arterial hemorrhage, in a review of 43 cases of spontaneous cervical epidural hematoma. Robertson et al. (52) mentioned a rupture of a so-called cryptic vascular anomaly as a possible etiologic factor. A rupture of such a small cryptic epidural angiomatous structure could be responsible for some cases of SSEH (52).

In many publications the authors propose the existence of a true vascular anomaly as the cause of SSEH (5,13,16-18,23,34,35,39,41,44,45,47,58,63). Such an anomaly might well explain the occasional occurrence of back pain and radicular pain without neurologic deficit which have preceded the final hemorrhagic accident by months or years in some cases of SSEH (33). The inability to reveal a hemangioma by pathological examination not necessarily means that such malformations should be ignored as the possible source of SSEH (16). An unrecognized vascular anomaly might be demonstrated more frequently by the use of serial histological sections (39). The failure to visualize these malformations in SSEH also could be related to the small size of these anomalies or to thrombosis after the initial episode of bleeding (19).

In the literature the vertebral hemangioma, in particular the purely epidural hemangioma, is regarded a very rare condition. Most cases of spinal epidural hemangiomas are vertebral angiomas extending into or invading the vertebral epidural space (28,66). Purely epidural hemangiomas almost always seem to occur in the thoracic spinal canal, and predominantly in females (64). The histological structure varies from cavernous hemangioma to less mature hemangio-endothelioma and hemangio-blastoma (28). The clinical signs of hemangiomas of the spinal epidural space are those of progressive paraplegia of varying rapidity of onset, but usually this develops within a few months. Some cases with temporary remission are known. Only one case of sudden and complete paraplegia was noticed and was shown at autopsy to have resulted from an epidural hemorrhage from the vessels of an epidural vascular tumor (28). Scharfetter (53) considered it remarkable that an acute epidural hemorrhage has never been mentioned in publications about spinal epidural vascular malformations. With regard to the clinical history of spinal (epidural) (hem)angio-mas and the doubtful results of histological examination in SSEH mentioned in this review, the assumed predominant role of a vascular malformation in the pathogenesis of the SSEH may be questioned. This opinion seems to be strengthened by the report of a SSEH in a patient with a vertebro-epidural hemangioma (postoperatively diagnosed by angiography) several segments above the actual spinal epidural hematoma (62).

4.5 Conclusions.

(1) Summarizing the different causes mentioned in literature, the possible ruptures of epidural veins, arteries, cryptic angiomas, vascular malformations or hemangiomas and spinal angiomas are advocated. However, none of the authors managed to give supportive evidence for their theories, neither statistically nor on an anatomical basis.

(2) The SSEH at the thoracolumbar area mainly occurs after the fourth decade. Before that age, an epidural hematoma extending to or originating from these vertebral segments is an exception.

(3) Despite many suggestions in the literature, arterial hypertension does not play a role in the pathogenesis of the SSEH.

(4) The vertebral and/or epidural hemangioma plays an insignificant role in the etiology of the SSEH.

4.6 References.

1. Ainslie J.P. Paraplegia due to spontaneous extradural or subdural hemorrhage. Br J Surg (1958) 45:565-567.
2. Alderman D.B. Extradural spinal-cord hematoma: report of a case due to dicoumarol and review of the literature. N Engl J Med (1956) 255:839-842.
3. Amyes E.W., Vogel P.J. and Raney R.B. Spinal cord compression due to spontaneous epidural hemorrhage: report of three cases. Bull Los Angeles Neurol Soc (1955) 20:1-8.
4. Bain W. A case of haematorrachis. Br Med J (1897) 2:455.
5. Banerjee T. and Meagher J.N. Spontaneous spinal epidural hemorrhage: case report. South Med J (1974) 67:858-861.
6. Bareño E.U. and Schlamich M.A. L'hématome épidural cervical spontané. A propos d'un cas. Neurochirurgie (1987) 33:66-70.
7. Beatty R.M. and Winston K.R. Spontaneous cervical epidural hematoma. A consideration of etiology. J Neurosurg (1984) 61:143-148.
8. Bidzinski J. Spontaneous spinal epidural hematoma during pregnancy: case report. J Neurosurg (1966) 24:1017.
9. Bruyn G.W. and Bosma N.J. Spinal extradural hematoma. In: Vinken en Bruyn, eds. Handbook of clinical neurology (1976). Vol.26. Amsterdam: North-Holland publishing company. pp. 1-30.
10. Busse O., Hamer J., Paal G. and Piotrowski W. Spontane epidurale spinale Hämatome wahrend und nach Antikoagulantienmedication. Nervenarzt (1972) 43:318-322.
11. Calliauw L., Dhara M., Martens F. and Vannerem L. Spinal epidural hematoma without lesion of the spine. Report of 4 cases. Clin Neurol Neurosurg (1988) 90(2):131-136.
12. Chavany J.A., Taptas J.N. and Pecker J. A propos d'une varieté exceptionelle d'hematorachis: l'hematome epidurale spontané. Presse Med (1949) 62:869-871.
13. Combelles G., Blond S., Lesion F., Bousquet C., Rousseaux M. and Christiaens J.L. Hématomes extra-dureaux rachidiens sans lésion osseuse traumatique. A propos de 9 cas. Neurochirurgie (1983) 29:417-422.

14. Cooper D.W. Spontaneous spinal epidural hematoma. Case report. J Neurosurg (1967) 26:343-345.
15. Correa A.V. and Breasley B.A.L. Spontaneous cervical epidural hematoma with complete recovery. Surg Neurol (1978) 10:227-228.
16. Cube H.M. Spinal extradural hemorrhage. J Neurosurg (1962) 19:171-172.
17. Dauch W.A. Spinale Epiduralhämatome bei Kindern und Jugendlichen. Neurochirurgia (1986) 29:83-89.
18. Dawson B.H. Paraplegia due to spinal epidural hematoma. J Neurol Neurosurg Psychiat (1963) 26:171-173.
19. Devadiga K.V. and Gass H.H. Chronic lumbar extradural hematoma simulating disc syndrome. J Neurol Neurosurg Psychiat (1973) 36:255-259.
20. Fischbach von R. and Kollar W.A. Spontane spinale epidurale Blutung unter Antikoagulantion. Wien Med Woch (1972) 19/20:5-6.
21. Flaschka G., Sutter B., Ebner F., Klein G.E. and Tilz G. Das spinale Epiduralhämatom. Langzeitergebnisse von 4 eigenen Fällen. Nervenarzt (1990) 61:629-633.
22. Foo D. and Rossier A.B. Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas. Surg Neurol (1981) 15:398-401.
23. Foo D., Chang Y.C. and Rossier A.B. Spontaneous spinal epidural hemorrhage, anterior cord syndrome, and familial vascular malformation: case report. Neurology (NY) (1980) 30:308-311.
24. Gauthier G. L'hematome extradural rachidien sans fracture de la colonne: revue de 56 cas verifies. Psychiat Neurol (Basel) (1963) 146:149-175.
25. Ghanem M.B. and Ivan L.P. Spontaneous spinal epidural hematome in an eight year old boy. Neurology (Minneap) (1978) 28:829-832.
26. Gold M.E. Spontaneous spinal epidural hematome. Radiology (1963) 80:823-828.
27. Goulon M., Nouailhat F., Julian H., Barois A., Gajdos P. Babinet P. and Rapin M. Hématome extradurale cervicale spontané, responsable d'une hémiplégie spinale: guérison après intervention. Soc Med Hop Paris (1967) 118:969-979.
28. Guthkelch A.N. Hemangiomas involving the spinal epidural space. J Neurol Neurosurg Psych (1948) 11:199-210.
29. Hehman K. and Norrel H. Massive chronic spinal epidural hematoma in a child. Am J Dis Child (1968) 116:308-310.
30. Herrmann E., Lorenz R. and Vogelsang H. Zur diagnostik der spinalen epiduralen Hämatome und Abszesse. Radiologe (1965) 5:504-508.
31. Ingelfinger J.A., Mosteller F. and Thibodeau L.A. Biostatistics in clinical medicine. New York: Macmillan publishing company (1987) 95.
32. Jackson R. Case of spinal apoplexy. Lancet (1869) 2:5-6.
33. Jellinger K. Neuropathology of cord injuries, in: Vinken en Bruyn (eds): Handbook of clinical neurology. Vol.25. Amsterdam: North-Holland Publishing Co. (1976) 72-76.
34. Kaplan L.I. and Denker P.G. Acute non-traumatic spinal epidural hemorrhage. Am J Surg (1949) 78:356-361.

35. Koyama T., Igarashi S., Hanakita J. and Handa J. Das spinale epidurale Hämatom. Neurochirurgia (1982) 25:11-13.
36. Lepoire J., Tridon P., Montaut J. and Germain F. L'hématome extra dural rachidien spontané. Neurochirurgie (1959) 7:298-313.
37. Liebeskind A.L., Schwartz K.S., Coffey E.L. and Beresford H.R. Spinal epidural hematoma with delayed appearance of neurological symptoms. Neuroradiology (1975) 8:191-193.
38. London G.W., McKeever P.E. and Wiederholt W.C. Spontaneous spinal epidural hematoma in alcoholism. Ann Int Med (1974) 81(2):266-267.
39. Lougheed W.M. and Hoffman H.J. Spontaneous spinal extradural hematoma. Neurology (1960) 10:1059-1063.
40. Markham J.W., Lynge H.N. and Stahlman G.E.B. The syndrome of spontaneous spinal epidural hematoma. Report of three cases. J Neurosurg (1967) 26:334-342.
41. Maxwell G.M. and Puletti F. Chronic spinal epidural hematoma in a child: case report. Neurology (1957) 7:597-600.
42. Mayer J.A. Extradural spinal hemorrhage. Case report. Can Med Ass J (1963) 89:1034-1037.
43. Miyasaka Y., Morii S., Takiga H., Ohwada T. and Yada K. A case of acute spinal epidural hematoma due o a hemorrhage of spinal epidural arteriovenous malformations. No Shinkei Geka (1977) 5:655-661.
44. Müller H., Schramm J., Roggendorf W. and Brock M. Vascular malformations as a cause of spontaneous spinal epidural hematoma. Acta Neurochir (Wien) (1982) 62:-297-305.
45. Nichols P. and Manganiello L.O.J. Extradural hematome of the spinal cord. Case reports. J Neurosurg (1956) 13:638-640.
46. Oldenkott P. and Driesen W. Spontanes epidurales Hämatom im Burstwirbelkanal wärhend Antikoagulantienlangzeitbehandlung. Med Welt (1966) 17:305-307.
47. Packer N. and Cummins B.H. Spontaneous epidural hemorrhage: a surgical emergency. Lancet (1978) 18:356-358.
48. Pear B.L. Spinal epidural hematoma. Am J Roentgenol Radium Ther Nucl Med (1972) 115:155-164.
49. Penar B.L., Fisher D.K., Goodrich I., Bloomgarden G.M. and Robinson F. Spontaneous spinal epidural hematoma. Int Surg (1987) 72:218-221.
50. Rao B.D., Rao K.S., Subrahmanian M.V. and Raghava Reddy M.V. Spinal epidural hemorrhage. Br J Surg (1966) 53:649-650.
51. Rebello M.D. and Dastur H.M. Spinal epidural hemorrhage. Neurology (India) (1966) 14:135-145.
52. Robertson W.C. Jr., Lee Y.N. and Edmonson M.B. Spontaneous spinal epidural hematoma in the young. Neurology (1979) 29:120-122.
53. Scharfetter F. Das spontane spinale epidurale Hämatom in der Differentialdiagnose des akuten spinalen Insultes. Dtsche Med Wochenschr (1972) 1:13-16.
54. Scheil F., Larkamp U. and Mutharoglu U. Das spontane spinale epidurale Hämatom. Neurochirurgia (1990) 33:45-49.

55. Schicke R. and Seitz D. Spinales epidurales Hämatom unter Antikoagulantien-therapie. Dtsche Med Wochenschr (1970) 6:275-277.
56. Schultz E.C., Johnson A.C., Brown C.A. and Mosberg W.H. Paraplegia caused by spontaneous spinal epidural hemorrhage. J Neurosurg (1953) 10:608-616.
57. Scott B.B., Quisling R.G., Miller C.A. and Kindt G.W. Spinal epidural hematoma. JAMA (1976) 235:513-515.
58. Solero C.L., Fornari M. and Savoiardo M. Spontaneous spinal epidural hematoma arising from ruptured vascular malformation. Case report. Acta Neurochir (Wien) (1980) 53:169-174.
59. Stanley P. and McComb J.G. Chronic spinale epidural hematoma in hemophilia A in a child. Pediatr Radiol (1983) 13:241-243.
60. Suzuki N., Inoue H. and Abematsu N. Acute spontaneous spinal epidural hemorrhage in pregnancy. Nippon Seikeigeka Gakkai Zasshi (1977) 51(3):155-160.
61. Svien H.J., Adson A.W. and Dodge H.W. Lumbar extradural hematoma. Report of a case simulating protruded disc syndrome. J Neurosurg (1950) 7:587-588.
62. Ter Spill H.W. and Tijssen C.C. Spinal epidural hematoma due to a vertebro-epidural hemangioma. Clin Neurol Neurosurg (1989) 91:91-93.
63. Tsai F.Y., Popp A.J. and Waldman J. Spontaneous spinal epidural hematoma. Neuroradiology (1975) 10:15-30.
64. Umbach W. and Kunft H.-D. Vascular tumors of the spinal cord. In: Vinken en Bruyn, eds. Handbook of clinical neurology (1976). Vol.20. Amsterdam: North-Holland publishing company. pp. 435-480.
65. VerBrugghen A. Extradural spinal hemorrhage. Ann Surg (1946) 123:154-159.
66. Vogelsang H. Intraosseous spinal venography. Amsterdam, Exerpta medica (1970).
67. Von Klossek H. and Hüller E. Zur problematiek der spontanen spinalen Epiduralhämatome. Zeitbl Neurochirurgie (1984) 45:116-123.
68. Weigert M. Akutes spinales epidurales Hämatom als Folge von Behandlung mit Antikoagulantien. Nervenarzt (1961) 32:85-89.
69. Winer B.M., Horenstein S. and Starr A.M. Spinal epidural hematoma during anticoagulant therapy. Circulation (1959) 19:735-740.
70. Yonekawa Y., Mehdorn M. and Nishikawa M. Spontaneous spinal epidural hematoma during pregnancy. Surg Neurol (1975) 3:327-328.

Chapter 5

THE VASCULAR ANATOMY OF THE SPINAL EPIDURAL SPACE.
Considerations on the etiology of the spontaneous spinal epidural hematoma.

5.1 Introduction.

In the literature, several theories have emerged about the etiology of SSEH (see *Chapter 4*). However, none of these theories has been supported by convincing anatomical and/or statistical evidence. In the present chapter a review of the anatomy of the spinal epidural space will be presented, in particular of the venous and arterial structures, so as to form the basis for further study on the etiology of the SSEH.

5.2 The vascular anatomy of the spinal epidural space.

5.2.1 The spinal epidural space.

The dura mater is composed of internal and external laminae (4,13). The internal lamina of the dura mater (the dura proper) surrounds the spinal cord, spinal nerve roots and spinal subarachnoid space. The external lamina is represented by the periosteum of the vertebral canal and is closely attached to the posterior longitudinal vertebral ligament, the ligamenta flava. As a result, the space commonly referred to as the spinal epidural space (17) actually lies intradurally (4). In infants up to the age of 1½ years both lamina are well defined, but in adults the external lamina can no longer be identified as a separate layer (4,13). Close to the foramen magnum both lamina fuse and form the double-layered intracranial dura mater. Caudally the epidural space is limited at the sacral hiatus (13).

5.2.2 The spinal epidural venous structures.

The intracranial venous sinuses lie between the two dural lamina. Resembling these sinuses, and connected with them, lies a spinal venous plexus, which is called the internal vertebral venous plexus. Unlike many veins, this spinal venous system is supposed to be valveless (5). It is formed by two pairs of longitudinal veins, one pair lying anteriorly and the other pair posteriorly. Each pair is joined by transversely running anastomoses, forming the anterior and posterior internal vertebral venous plexuses, each of which resembles a rope ladder. The anterior internal plexus connects with the intracranial basilar venous plexus, which joins both cavernous sinuses and the petrosal sinus. The posterior internal plexus communicates with the occipital sinus, the marginal sinus and thereby the confluens sinuum. The internal vertebral venous plexus also connects, via the segmental spinal veins, with the inferior vena cava and the azygos and hemiazygos veins. Microscopically, the walls of the spinal venous plexus contain a small amount of elastic and a large amount of collagen fibers. The presence of smooth muscle cells enables this venous system to change its volume. As a result, the internal vertebral venous plexus is an important cerebral venous outflow tract and also a volume/pressure regulating system between intrathoracic, intra-abdominal, intracranial and spinal venous channels (4,5).

In the past, the vertebral venous system has been largely ignored by clinicians and anatomists. Breschet, in 1828-1832 (3), established that the vertebral venous system formes a separate and discrete group of vessels, paralleling, joining and at the same time by-passing, the longitudinal veins of the thoraco-abdominal cavity. On the one hand, like the azygos veins it unites the superior and inferior vena cava but, on the other hand, it is protected from thoraco-abdominal pressures because of its course in the rigid spinal canal (1). Batson (2) demonstrated, by the injection of radiopaque material into the deep dorsal vein of the

penis, the connection of the pelvic venous plexus with the vertebral vennous plexus. This finding provided a ready explanation for the observations that metastases from cancers of the urogenital system, in particular prostatic metastases, distibute to the vertebral skeleton and spinal epidural space.

Clemens (5) noted, in a study of 30 human cadavers (aged between 24 and 92 years), that the posterior internal vertebral venous plexus is the largest component of the internal vertebral venous system. He stated that the posterior internal vertebral venous plexus is a typical continuous venous network, particularly at the levels C6-T3. A similar concentration of veins was found at the lumbar spine; however, in this region the networks seemed restricted to the level of each vertebral body. In contrast to the posterior part of the internal vertebral venous plexus, which lies free in the peridural fat, a significant part of the anterior internal venous plexus is covered by the posterior longitudinal vertebral ligament (*Figure 5.1*).

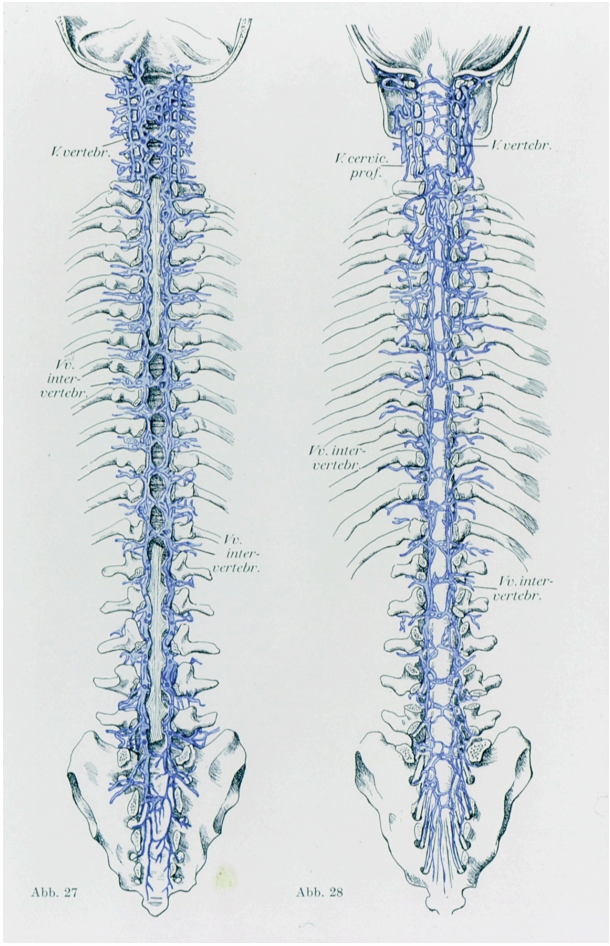


Figure 5.1. Schematic reconstruction of the anterior (left) and the posterior (right) internal vertebral venous plexus. (Reproduced from Clemens (1961) by permission of Walter de Gruyter & Co., Publishers.)

5.2.3 The spinal epidural arterial structures.

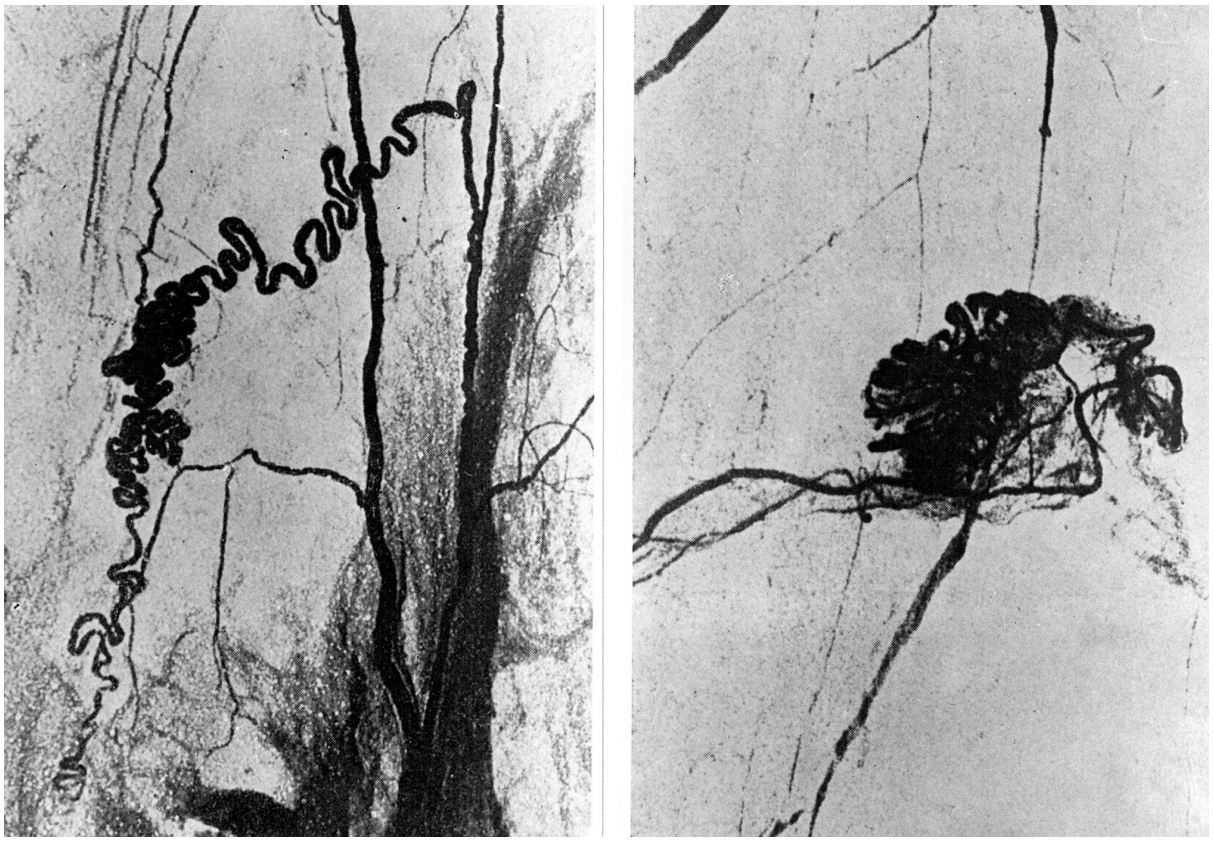
The anatomy of the arteries in the cervical epidural space is different from that in the thoracic and lumbar region. The arterial supply of the meninges and the epidural space in the thoracic, lumbar and sacral region originates from branches of the segmental intercostal, lumbar and sacral arteries (6). These so-called lateral spinal arteries enter the intervertebral foramen and divide into: [1] the neural ramus and its posterior and anterior radicular arteries which penetrate the dural sheath to supply spinal nerve roots, arachnoid and spinal cord; [2] the posterior central ramus supplying the ventral part of epidural space and dura; and [3] the preliminary ramus which supplies the dorsal half of the epidural space (4).

The supply for the cervical meninges originates from branches of the ascending and the deep cervical arteries which, in turn, arise from the thyreocervical trunks and from the subclavian arteries. Inside the spinal canal the ascending and deep cervical arteries divide so as to form a longitudinal arterial channel. Originating from this longitudinal channel, and midway between adjacent nerve roots, the so-called bridging vessels run transversely towards the midline of the dural sac, where they anastomose with bridging arteries from the opposite side. These bridging vessels and longitudinal channels are restricted to the cervical region.

Lazorthes and Manelfe (11), in a study on 18 subjects [15 adults and 3 fetuses], described two types of small spinal dural arteries. The spiral type arteries (”artère spiralée” or ”artère en méandre”) (*Figure 5.2*), between 1 and 10 mm long, run longitudinally, mainly on the posterior surface of the spinal dura (the internal lamina) and on the root sheaths. The number of spiral arteries, most numerous over the first five cervical segments, gradually decreases caudally with an increase at T2 and the lower thoracic levels. Microscopically the spiral artery is composed of endothelium, a thick basal membrane, a thin media, and a covering of muscular fibers. Lazorthes and Manelfe (11) note the existence of spiral arteries in the uterus, spermatic cord and the kidney. Despite many theories, the exact function of these arteries remains unknown.

The second type is the cluster-type artery (”les pelotons vasculaires”) (*Figure 5.2*), which is a complex of arteries with centrally and peripherally localized veins (”une véritable pelote vasculaire”) (11). The ”pelotons vasculaires” are actually small arterio-venous anastomoses. These clusters are most common in the thoracic region, especially at the level of T2. Generally, they are situated at the posterior surface of the spinal dura, an anterior location being exceptional. Microscopically, the arterial component resembles the spiral artery and the veins are rather similar to ordinary veins. Compared to the general type of veins, cluster veins have a rather thick fibromuscular media. Like the spiral arteries, the significance of the cluster arteries is unknown.

Figure 5.2. Spiral artery at the level T4 (left) and Cluster artery at the level T2 (right). (Reproduced from Lazorthes and Manelfe (1970) by permission of Masson éditeur.)



5.3 Discussion.

As stated in the introduction, the study of the etiology of the SSEH requires an understanding of the anatomy of the spinal epidural space, specially with regard to its vascular structures. Based on current knowledge of the anatomy of the spinal epidural space and the findings after study of a large series of cases, the theories about the etiology of SSEH can be discussed.

5.3.1 The arterial theory.

Some authors propose an arterial origin of the SSEH. Beatty and Winston (2) suggest a rupture of one of the free bridging cervical epidural arteries as a cause for the acute spontaneous cervical epidural hemorrhage, as these arteries are thought to be extremely vulnerable to mechanical disruption. The authors advocate that this theory can apply to the entire spinal epidural space; this is doubtful because no anatomical evidence is found in the

literature for the existence of "free bridging epidural arteries" at levels other than the cervical spine. Furthermore, the assumption of mechanical disruption of these vessels seems unlikely, since most SSEHs appeared to occur at rest or under tranquil conditions.

5.3.2 The vascular malformation theory.

A significant number of authors stress the role of spinal epidural vascular malformations in the etiology of the SSEH (7-10,15). In the previous chapter (*Chapter 4*), 18 hematomas are reported to have resulted from a ruptured epidural angioma or vascular malformation (see *Table 4.3*). According to the histological accounts in these cases, some marginal notes need to be made. Dawson (8), Foo and Rossier (9) and Müller et al. (15) detected structures with "intimate union of arteries and veins" in their histological preparations. Their reports of small "vascular anomalies" resemble the definition of the cluster-type artery, as reported by Lazorthes and Manelfe (11). These structures have never been mentioned in discussions about the etiology of the SSEH. Because of the unacquaintedness with these vascular clews, the cluster-type artery can easily be mistaken for a vascular malformation.

In some reports (8,10,12,14-16) the authors concluded to a vascular anomaly after meeting with venous clusters in an epidural hematoma (*Table 4.3*). It should be taken into consideration that in these cases the hematomas enclosed congested or distended vessels of the posterior internal vertebral venous plexus.

5.3.3 The venous theory.

The studies of Breschet (3), Batson (1) and Clemens (5) on the venous system of the human vertebral canal demonstrated the significance of the internal vertebral venous plexus. Because of the assumption of the absence of valves, the plexus is thought to be vulnerable to increases of the intra-abdominal pressure (4). There are several arguments that seem in favor of a "venous cause" of SSEH.

[1] Most SSEHs are situated in the posterior and postero-lateral part of the epidural space (see *Chapter 3*). The posterior internal vertebral venous plexus lies freely in the epidural space, while a significant part of the anterior internal vertebral venous plexus is covered by the posterior longitudinal vertebral ligament (5). Due to this anatomical situation, part of the anterior internal vertebral venous plexus is sheltered from the epidural space. If a bleeding occurs in this area, the tight posterior longitudinal vertebral ligament might prevent the hematoma from expansion and compressing the spinal cord. Possibly this is the explanation for the fact that symptomatic SSEHs are predominantly situated in the posterior part of the spinal epidural space.

[2] Clemens has described a concentration of veins in the cervico-thoracic part of the posterior internal vertebral venous plexus (5). He also noticed a (much smaller) increase of veins in the lumbar posterior internal vertebral venous plexus (5). This seems to fit with the

segmental distribution of SSEH. Among the group of younger patients with SSEH (0-40 years), most hematomas were located in the lower cervical and upper thoracic epidural space (C6-T3) (see *Chapter 4, Figure 4.4*). Spontaneous epidural bleeding at the lower thoracic and upper lumbar levels occurred almost exclusively in older patients (40-80 years). According to what is currently known about the anatomy of the posterior internal vertebral plexus (3,5), a rupture of the compact continuous venous network at the levels C6-T3 seems a very likely cause of the SSEH. However, this does not explain the large number of hematomas occurring at the lower thoracic epidural space, neither the remarkable age distribution in SSEH. It could very well be that degenerative changes of the spinal column and/or the spinal epidural veins are responsible for this phenomenon.

5.4 Conclusions.

(1) In general, it may be concluded that a lack of familiarity with the normal vascular structures of the spinal epidural space may have resulted in erroneous conclusions about the etiology of the SSEH.

(2) The cluster-type artery can easily be mistaken for a vascular anomaly, because of its remarkable morphology and the unacquaintedness of the clinician with this structure.

(3) The rupture of a spinal epidural artery (cluster- or spiral-type) might be responsible for some cases of SSEH.

(4) The anatomical dimensions of the posterior internal vertebral venous plexus (as deduced from the literature) and the distribution of SSEHs over the spinal levels in 333 cases, suggest that the majority of SSEHs result from a rupture in this particular vascular network.

5.4 References.

1. Batson O.V. The vertebral vein system. Am J Roentg, Radium Ther and Nucl Med (1957) 78:195-212.
2. Beatty R.M. and Winston K.R. Spontaneous cervical epidural haematoma. A consideration of etiology. J Neurosurg (1984) 61:143-148.
3. Breschet G. Recherches anatomiques, psychologiques et pathologiques sur le système veineux et spécialement sur les caveaux veineux des os. (1828-1832) Paris: Villaret et Cie.
4. Bruyn G.W. and Bosma N.J. Spinal extradural haematoma. In: Vinken en Bruyn, eds. Handbook of Clinical Neurology. (1976) Amsterdam: North-Holland publishing company. pp. 1-30.

5. Clemens H.J. Die Venensysteme der menschlichen Wirbelsäule. Morphologie und functionelle Bedeutung. (1961) Berlin: Walter de Gruyter & co.
6. Crock H.V. and Yoshizawa H. The blood supply of the vertebral column and spinal cord in man. (1977) Berlin/Heidelberg/New York: Springer Verlag.
7. Cube H.M. Spinal extradural hemorrhage. J Neurosurg (1962) 19:171-172.
8. Dawson B.H. Paraplegia due to spinal epidural haematoma. J Neurol, Neurosurg and Psych (1963) 26:171-173.
9. Foo D. and Rossier A.B. Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas. Surg Neurol (1981) 15:398-401.
10. Koyama T., Igarashi S., Hanakita J. and Handa J. Das spinale epidurale Hämatom. Neurochirurgia (1982) 25:11-13.
11. Lazorthes G. and Manelfe C. La vascularisation artérielle de la dure-mère rachidienne. Les artères spiralées et les pelotons vasculaires. CR Acad Sc Paris (1970) 271:88-91.
12. Lougheed W.M. and Hoffman H.J. Spontaneous spinal extradural hematoma. Neurology (1960) 10:1059-1063.
13. Luyendijk W. Canalografie. Thesis. (1962) Leiden: Groen v/h & Zoon. pp. 23-32.
14. Mayer J.A. Extradural spinal hemorrhage. Case report. Can Med Ass J (1963) 89:1034-1037.
15. Müller H., Schramm J., Roggendorf W. and Brock M. Vascular malformations as a cause of spontaneous spinal epidural haematoma. Acta Neurochir (Wien) (1982) 62:297-305.
16. Solero C.L., Fornari M. and Savoiardo M. Spontaneous spinal epidural haematoma arising from ruptured vascular malformation. Case report. Acta Neurochir (Wien) (1980) 53:169-174.
17. Williams P.L., Warwick R., Dyson M. and Bannister L.H. (eds.) Gray's Anatomy (1989) Edinburgh: Churchill Livingstone. pp. 1088-1089.

Chapter 6

THE MORPHOLOGY OF THE INTERNAL VERTEBRAL VENOUS PLEXUS.

A cadaver study after intravenous Araldite CY 221 injection.

6.1 Introduction.

In the previous chapters (*Chapter 4 and 5*), it has been concluded that a rupture in the posterior part of the internal vertebral venous plexus seems the most likely cause of SSEH. The predominance of SSEH in the posterior/posterolateral area of the cervicothoracic and thoracolumbar segments of the spinal canal, might be related to regional differences that are reported in the morphology of the posterior internal vertebral venous plexus (4). Since details about the morphology of the internal vertebral venous plexus are not available from the literature, it was decided to perform a human cadaver study with emphasis on the segmental and interindividual variations of this vascular system.

6.2 Materials and methods.

6.2.1 Injection material.

Araldite CY 221 in combination with hardener HY 2967 and dilutioner DY 026 SP was used in this study. This polymere is preferable to other injection materials because of its low viscosity, low surface tension, long polymerization-time and the solidity of the cast (29). We used a mixture of 20 units of volume Araldite CY 221 with 7.5% Microlith T blue (kindly provided by CIBA-GEIGY (Arnhem, The Netherlands)), 45 units of volume of hardener HY 2967 and 60 units of volume of dilutioner DY 026 SP.

Table 6.1. Relevant data of the 10 cadavers.

Number	Sex	Age	Cause of death
533	_	80	Brain tumor
537	_	80	Cardiac failure
546	_	89	Cardiac failure
550	_	93	Cardiac failure
554	_	83	Cardiac failure
555	_	93	Cardiac failure
556	_	78	Lung cancer
557	_	64	Oesophageal cancer
573	_	68	Intracerebral hemorrhage
587	_	77	Pancreatic cancer

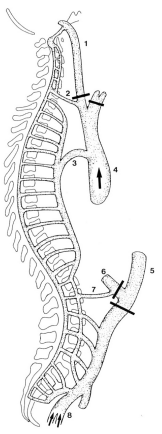
6.2.2 Specimens and injection procedure.

Injection of Araldite was performed in ten fresh, unembalmed human cadavers (*Table 6.1*), within 48 hours after death. Initially (analogue to the technique as described by Clemens (4)), the proximal part of both clavicles and the sternal manubrium were removed. Thereafter the following veins were dissected and ligated carefully: the external and internal jugular veins, the subclavian veins (proximal to the costocervical truncus), the inferior and imal thyreoid veins, the internal thoracic veins and the superior vena cava (proximal to the azygos vein). The vertebral veins, the deep cervical veins and the azygos/hemiazygos veins were not ligated, enabling injection-material to flow via the superior vena cava (in a retrograde direction) into the vertebral venous system. Both femoral veins were ligated through separate incisions in the inguinal region. Cannulas were inserted in the superior vena cava

and both femoral veins. After removal of the excess of blood clots by infusion of 500 ml of saline (room temperature) under hydrostatic pressure (50 cm above the heart of the cadaver), 100,000 units of Streptokinase or Urokinase (kindly provided respectively by Pharmacia & Upjohn B.V. (Woerden, The Netherlands) and Lamepro B.V. (Raamsdonkveer, The Netherlands)) in 500 ml of saline were injected into the venous system and left in place for at least two hours to dissolve remaining blood clots. Afterwards, the vertebral venous system was reflushed with saline in retrograde (via the superior vena cava) and in anterograde (via the femoral veins) fashion, both with the infusion reservoir 50 cm above the heart. Tricuspic valves were encountered in both vertebral veins of one cadaver (Nº 554), just proximal to their entrance into the subclavian veins. Those valves were excised and silicone tubes were inserted to enable perfusion of the cervical part of the vertebral venous plexus. After reflushing with saline, 500 ml of Araldite (room temperature) was injected manually into the superior vena cava (retrograde injection) and/or the femoral veins (anterograde injection) (*Figure 6.1*). Twelve hours later, after polymerization was completed, the vertebral column including a small part of the occiput was taken out and was immersed in a fixative consisting of 4% formaldehyde for at least two weeks. In order to visualize the cast of the posterior part of internal vertebral venous plexus, the perivertebral tissues, spinous processes, vertebral arches, apophyseal joints and spinal epidural fat tissue were removed carefully. Cadavers with destructive and/or metastatic diseases of the vertebral colum and its related structures, were excluded from the study. The preparations were recorded on photographs. After excision of the posterior part of the internal vertebral venous plexus, the spinal cord was removed and the anterior part of the internal vertebral venous plexus was visualized and recorded photographically.

It was noticed that Araldite injection according to the procedure described by Clemens (4), irrespective of the total volume injected, resulted in absent or incomplete perfusion of the lumbar and sacral parts of the anterior and posterior internal vertebral venous plexuses in the first four cadavers (see *Table 6.2*, Group A). Subsequently, a modified procedure was adopted by means of an additional supra-umbilical midline laparotomy, in order to ligate the (infrarenal) inferior vena cava (to prevent back-flow from the sacral venous plexus and the femoral veins) and the left renal vein (to prevent back-flow into the (suprarenal) inferior vena cava via the ascending lumbar vein) (*Figure 6.1*). This included the remaining six cadavers (see *Table 6.2*, Group B). In this group, injection of Araldite resulted in complete filling of both the anterior and posterior internal vertebral venous plexuses.

Figure 6.1 Schematic representation of the venous drainage of the spine, showing the sites of venous ligation (stripes) and Araldite injection (arrows).
1, jugular vein; 2, vertebral vein; 3, azygos vein; 4, sup. vena cava; 5, inf. vena cava; 6, left renal vein; 7, ascending lumbar vein; 8, femoral veins.
(Modified after Lasjaunias and Berenstein (14))



6.3 Results.

As has been described above, the vertebral venous system was flushed with saline prior to Araldite injection. During this procedure, patency of the venous system was confirmed through the "distal" canula (in the femoral veins or the superior vena cava, depending on the site of injection). Shortly after flushing had started, saline dropped out of the distal canula and this stopped within one minute after the infusion was interrupted. After the initial "filling phase", the inflow rate into the proximal canula and outflow rate from the distal canula were equal, indicating a steady state of flow without (significant) leakage. Interestingly, the flow rate during flushing via the superior vena cava (i.e. "retrograde venous infusion" via both vertebral veins and the azygos vein) was significantly lower in comparison with the flow rate while flushing via the femoral veins (i.e. "anterograde venous infusion"). As a consequence, "retrograde" flushing of the same volume of saline lasted approximately 15 minutes longer (which was about one third of the all-over infusion time) than "anterograde" flushing in the same cadaver (infusion reservoir 50 cm above the heart).

During dissection of the cadavers, Araldite was encountered in the cranial sinusses (superior sagittal sinus, confluens sinuum, sigmoid sinus, cavernous sinus, plexus basilaris), the major cerebral and cerebellar cortical veins, the subcutaneous cranial veins, the jugular veins, the entire external vertebral venous plexus, the intercostal veins and the plexus venosus sacralis (the latter only after intra-abdominal ligation of the inferior vena cava and the left renal vein). This confirms the existence of a wide communication of the vertebral venous system with the intracranial, intra-thoracic and intra-abdominal veins.

6.3.1 The posterior internal vertebral venous plexus.

The cast of the posterior part of the internal vertebral venous plexus was incomplete in cadavers 533, 537, 546 and 550 (Group A, *Table 6.2*). As indicated above, this appears to be related to the omission of an additional ligation of the left renal vein and the inferior vena cava (infrarenally). In the six cadavers in which these additional intra-abdominal ligations were performed (Group B, *Table 6.2*), a complete cast of the posterior internal vertebral venous plexus was obtained.

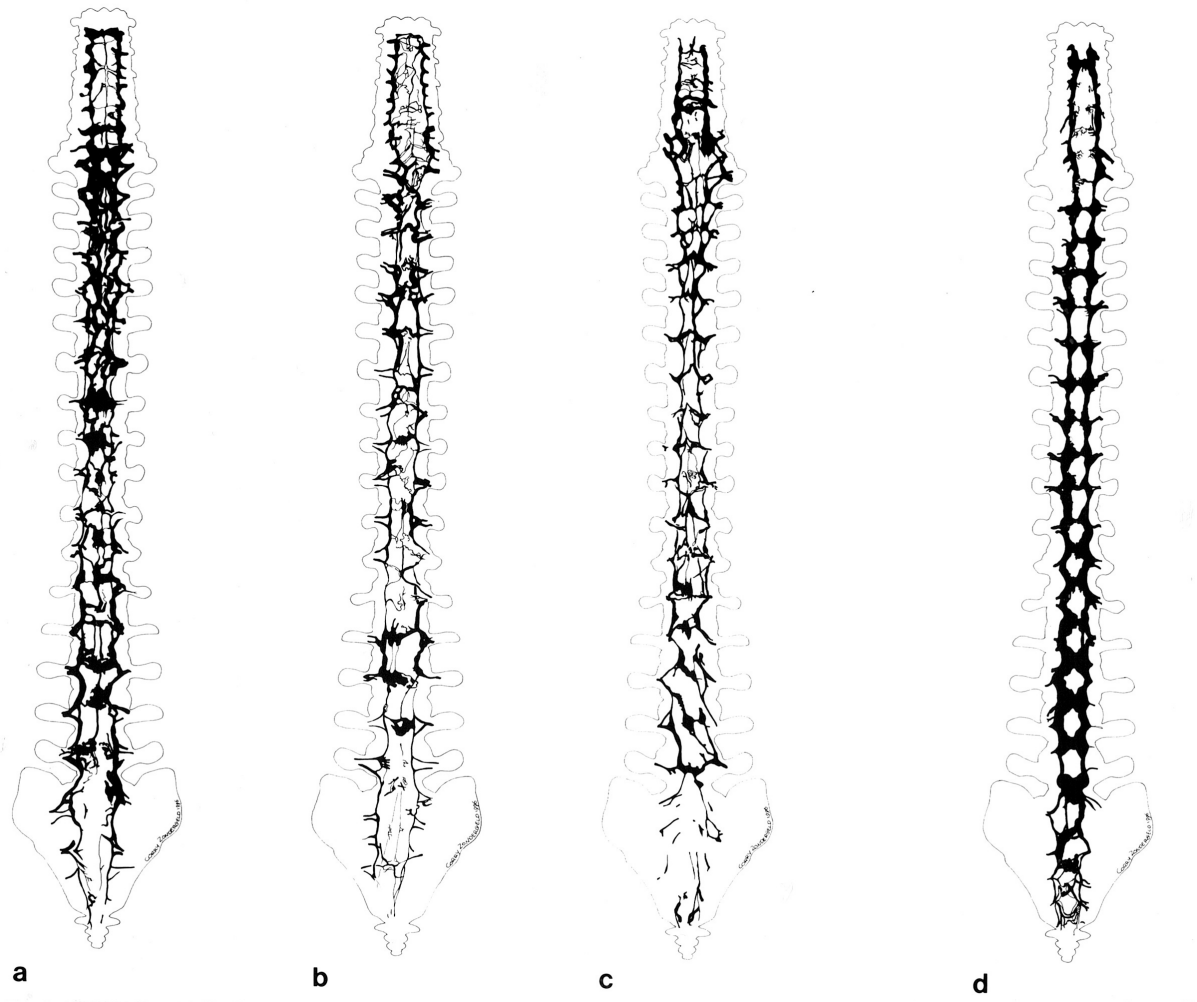
Table 6.2 Technique and results of vertebral venous system injection (Araldite CY) in 10 human cadavers.

		INJECTION-TECHNIQUE			INJECTION-RESULTS (venous filling)							
Number		v.ren. / v.c.inf. ligation	Injection-site		Posterior epidural plexus				Anterior epidural plexus			
			v.c.sup.	v.fem.	C	Th	L	S	C	Th	L	S
533	Group A	no	yes	no	-	+	±	-	+	+	+	-
537		no	yes	no	±	+	-	-	+	+	±	-
546		no	yes	no	±	±	±	-	+	+	+	±
550		no	yes	no	+	+	±	-	+	+	+	±
554	Group B	yes	yes	no	+	+	+	+	+	+	+	+
555		yes	yes	yes	+	+	+	+	+	+	+	+
556		yes	yes	no	+	+	+	+	+	+	+	+
557		yes	no	yes	+	+	+	+	+	+	+	+
573		yes	yes	yes	+	+	+	+	+	+	+	+
587		yes	yes	yes	+	+	+	+	+	+	+	+

+ = complete filling; ± = incomplete filling; - = absent filling

The posterior internal vertebral venous plexus lies freely within the spinal epidural fat and consists of one pair of longitudinal venous channels, each channel situated posterolaterally in the spinal epidural space. This pair of longitudinal veins is interconnected by means of segmental transverse bridging anastomoses that are usually situated underneath the arch of each vertebra, resulting in a ladder-like appearance. Three examples of the posterior internal vertebral venous plexus are shown in (*Figure 6.2*[a-c]).

Figure 6.2 Schematized representation of the posterior (a-c) and anterior (d) internal vertebral venous plexus (artist's impression, C. Zondergeld).



Although there appears to be a significant interindividual variability in the morphology of the posterior part of the internal vertebral venous plexus, the following general pattern can be discerned:
The ***cervical*** posterior internal vertebral venous plexus consists of a limited number of small horizontal veins bridging the longitudinally oriented left and right posterolateral epidural venous channels. At C1 (suboccipital plexus) and C6-C7 (*Figure 6.3*[a]) the plexus is more pronounced and consists of a more dense traversing venous network. In contrast to the rest of the posterior internal vertebral venous plexus, the suboccipital plexus is covered with a large amount of connective tissue. In most cases a thin longitudinal venous channel was found in the midline.

The ***thoracic*** posterior internal vertebral venous plexus is more extensive when compared with the cervical part. In all cadavers the upper part of the thoracic venous plexus was more extensive and voluminous than the lower part. There are large posterolateral channels that are interconnected by segmental bundles of traversing veins. We noticed two different types of traversing thoracic veins. In some cadavers the thoracic posterior internal vertebral venous plexus was very dense with many interconnecting bridging veins (*Figure 6.2[a] and 6.3[b]*). In other cadavers the veins were rather small and far less numerous (*Figure 6.2[c] and 6.3[c]*). In general, the configuration of the transversing thoracic veins resembles a (more or less symmetrical) inversed "V", with the wedge pointing rostrally (*Figure 6.3[c]*). As in the cervical area, the transversing thoracic posterior internal vertebral veins are interconnected by a small longitudinal channel in the midline.

The ***lumbar*** posterior internal vertebral venous plexus has broad horizontal segmental bridging anastomoses that connect both posterolateral longitudinal channels (*Figure 6.3[d]*). Longitudinal (para)median venous channels are also present in this area.

The ***sacral*** part of the posterior internal vertebral venous plexus consists of two lateral longitudinal channels. Bridging veins and longitudinal (para)median veins are absent (*Figure 6.3[e]*). A schematized reconstruction of the posterior internal vertebral venous plexus is reproduced in *Figure 6.4*.

Figure 6.3 Results after Araldite injection of the cervical (a), thoracic (b and c), lumbar (d) and sacral (e) parts of the posterior internal vertebral venous plexus and the anterior internal vertebral venous plexus (f) (detail of the lumbar area).

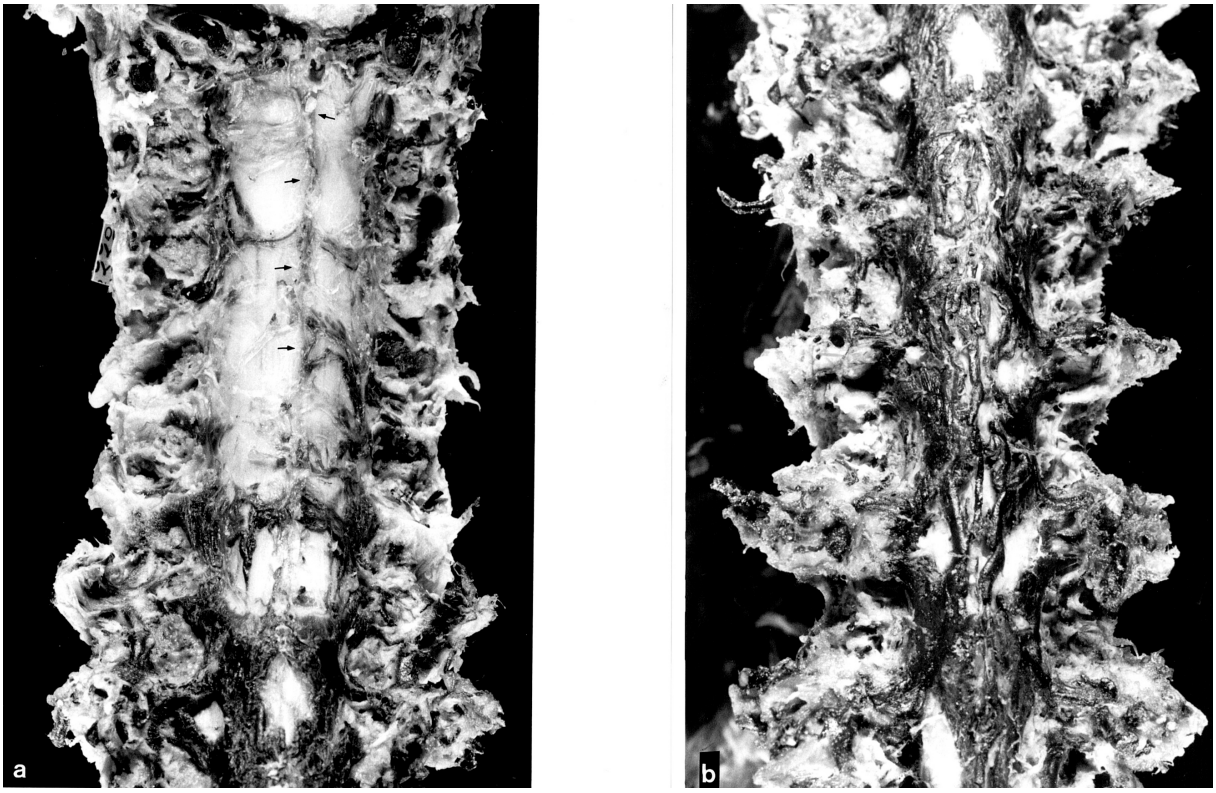
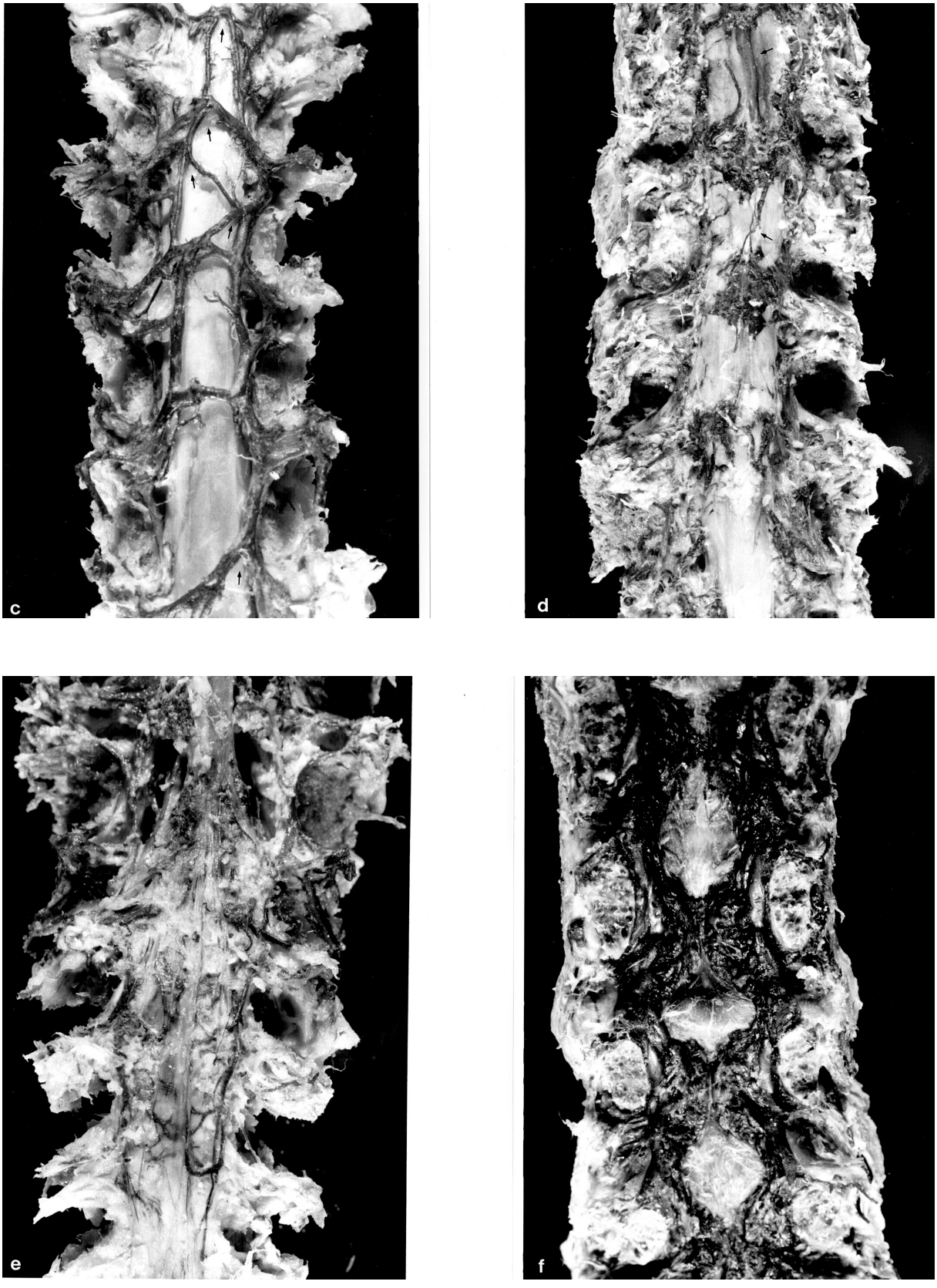


Figure 6.3(continued).

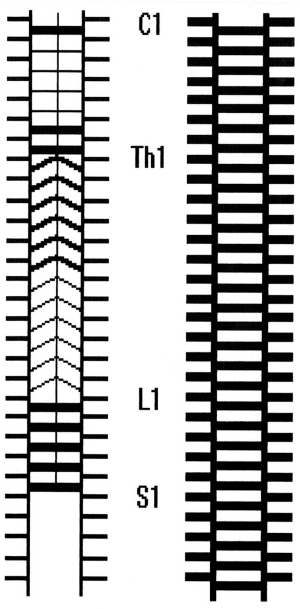


6.3.2 The anterior internal vertebral venous plexus.

This plexus runs in the anterior spinal epidural space, dorsal to the posterior aspect of the vertebral bodies and intervertebral discs. Epidural fat is almost absent in this area of the spinal canal. The posterior vertebral longitudinal ligament (PLL) is partially attached to the vertebral bodies. At the level of the intervertebral discs, the anterior internal vertebral venous plexus is situated *posterior* to the PLL. Cranial and caudal from the intervertebral discus, the plexus is lying *anterior* to the PLL. The Araldite cast of the anterior internal vertebral venous plexus was incomplete in the first four cadavers (Group A, *Table 6.2*). In cadavers in which an additional ligation of the intra-abdominal veins was performed (see above), Araldite injection (via the superior vena cava and/or the femoral veins) resulted in excellent perfusion of the entire anterior internal vertebral venous plexus (Group B, *Table 6.2*).

In contrast with the posterior internal vertebral venous plexus, the anterior plexus has a very constant morphologic pattern. Similar to the posterior internal vertebral venous plexus, the anterior system consists of a pair of longitudinal venous trunks that are located anterolaterally in the spinal canal. The cranial part of this plexus continues as the basilar venous plexus. Both longitudinal channels deviate laterally dorsal to each intervertebral disc and converge medially half way each vertebral body where they unite with the basivertebral vein. In this way they form a dense retrocorporeal network in close contact with the medial side of the vertebral pedicles (*Figure 6.3*[b]). The cervical part of this plexus is the smallest. While descending the spinal canal, the anterior internal vertebral venous plexus becomes more voluminous and more pronounced (with a maximum at L4 and L5). An abrupt decrease is noticed in the sacral canal. This pattern appeared to be identical in all ten cadavers (*Figure 6.2*[a]). A schematized reconstruction of the anterior internal vertebral venous plexus is reproduced in *Figure 6.4*.

Figure 6.4 Schematized reconstruction of the posterior (left) and anterior (right) internal vertebral venous plexuses.



6.4 Discussion.

6.4.1 Historical note.

The first anatomical study of the vertebral veins has been presented by Breschet (1828-1832). In that same period, Cruveilhier (1834-1836) underlined the importance of the internal vertebral venous plexus as a collateral system, after occlusion of the inferior vena cava. During a long period following these early accounts, the knowledge of these veins was hardly extended. This may be due to the fact that special methods have to be used to display this plexus and also because the clinical relevance of this system was not evident. Batson (1) was responsible for the revival of interest in the vertebral venous system. He demonstrated, by injecting the dorsal vein of the penis, that there are connections between the pelvic veins and the cranial sinuses via the vertebral venous plexus, bypassing the inferior and superior caval venous system. This provided a ready explanation for the distribution of prostatic cancer metastases to the vertebral skeleton and the spinal epidural space (1,2). However, even the role of the vertebral venous system as a vehicle for metastasis was neglected, as has been emphasized by Hussey (12).

In 1961, Clemens published a study on the morphological and histological aspects of the human vertebral venous system (4,5). In the years that followed, many publications appeared about the anatomico-radiological characteristics of the internal vertebral venous plexus (13,16,19,20,23,25) due to the introduction of intraosseous and transfemoral venography for diagnosis of lumbar (or less frequently cervical) disc herniation. These studies were focussed on the anterior part of the internal vertebral venous plexus. Recently, Plaisant et al. (17,18) described the anterior spinal epidural space, using magnetic resonance imaging (MRI) visualization after Gelatin/Gadolinium injection of the vertebral venous plexus. However, these studies were aimed at the lumbar posterior longitudinal vrtebral ligament. In the literature, little attention has been paid to the posterior part, or its existence has even been denied (23), most probably because of the difficulties with imaging of these vessels. Nowadays, the vertebral venous plexus is reproduced in most anatomical atlasses, but frequently the details are inaccurate and no attention is paid to the segmental and interindividual variability of the morphology of this venous system (*Figure 6.5*). Only Breschet (3) and Clemens (4) have studied both the anterior and the posterior part of the internal vertebral venous plexus. As a consequence, current knowledge of the morphology of the internal vertebral venous plexus is limited.

6.4.2 Technical considerations.

6.4.2.1 Cadaver-injection-study.

Visualization of the entire internal vertebral venous plexus appeared to be difficult. From the era of spinal venography/phlebography, it has become clear that this technique is inadequate for imaging the posterior part of the internal vertebral venous plexus (8,16,19,20,22,23,25,26). This can be explained by the fact that the vertebral venous system is a wide network that consists of several intercommunicating divisions. The direction of

blood flow within the venous plexus depends on the position of the body (hydrostatic factors) and on changes of the intra-thoracic and intra-abdominal pressure. These conditions appeared to be very difficult to controle during phlebography (23,25). Balloon occlusion of the inferior vena cava facilitates visualization of the vertebral venous plexus, but this technique is very time-consuming and requires great skill (25). Moreover, interpretation of phlebograms of the vertebral venous system is very problematic, since the morphological characteristics of this system (in particular the internal vertebral venous plexus) have not yet been established. As a consequence, post mortem injection of a solidifying polymere into the vertebral venous system and subsequent dissection of the spinal collumn seems the only reliable way to study the morphology of the human posterior internal vertebral venous plexus. In cadavers, the hydrostatic factors are stable and intra-thoracic and intra-abdominal pressure-waves are absent. During injection, flow will be uni-directional and, if the caval venous system has been trapped sufficiently, complete perfusion of the internal vertebral venous plexus will be achieved. This has been confirmed in the present study.

6.4.2.2 Injection procedure.

After death, blood tends to accumulate in the venous system. When the body is in the supine position, blood is pooled, especially in the dorsal parts of the trunk. Therefore, flushing of the vertebral venous system with saline and Streptokinase/Urokinase, prior to Araldite injection, is essential to remove obstructing blood clots from the areas of interest and to obtain perfusion of the internal vertebral venous plexus. Initially, Araldite was injected according to the descriptions of Clemens (4), which resulted in the absence of Araldite in the lumbar and sacral parts of the (posterior) internal vertebral venous plexus (see Group A, *Table 6.2*). Large amounts of Araldite were found in the inferior vena cava, renal veins, the heart and the pulmonary arteries. In the remaining cadavers (Group B, *Table 6.2*) an additional ligation of the infrarenal inferior vena cava and the left renal vein (proximal to the ascending lumbar vein) was performed, which prevented Araldite from escaping into the caval system and resulted in perfusion of the complete internal vertebral venous plexus. It may thus be concluded that the application of thrombolytics, the use of a modern polymere as injection material and the ligations of the inferior infrarenal vena cava and the left renal vein (as a modification of the technique described by Clemens (4)), are essential in perfusion studies of the vertebral venous system.

6.4.2.3 Quantitative analysis.

In order to get quantitative data of the internal vertebral venous plexus, several attempts have been made to calculate the segmental volume of these veins. This requires systematic equidistant transverse sectioning of the specimen (24). Different techniques were considered to obtain transverse sections of the areas of interest:

[1] Sawing of the spine. This was not carried out, because this results in the unevitable loss of tissue and leads to destruction of the cast before the morphological pattern of the venous plexus can be studied.

[2] Computerized tomography scanning was studied. Unfortunately, imaging of the plexus was poor, because discrimination of the Araldite cast nearby the bony structures was hardly possible. Another drawback was the poor resolution of small vessels. The addition of

radiopaque contrast medium (containing ionized iodine) to the Araldite mixture (in order to enhance contrast of the venous plexus) was impracticable, because the catalyst effect of the ionized iodine did reduce the polymerization-time of Araldite to an inacceptable level. Consequently, computerized tomography techniques had to be rejected.

[3] Magnetic resonance imaging likewise was abandoned, because of problems with resolution and discrimination of the venous structures. Solidified Araldite, like cortical bone, can not be visualized with MR-techniques, because of the absence of free protons. The application of Gadolinium appeared to be useless, because these molecules become trapped within the Araldite-mixture after polymerization has been completed.

In conclusion, besides from the numerical problems (only ten cadavers were dissected), volumetric analysis of the internal vertebral venous plexus had to be given up on practical grounds.

6.4.3 Morphological findings.

The size of the anterior internal vertebral venous plexus gradually increases from C1 down to L5, and rapidly decreases in the sacral canal. This may be related to the size of the vertebral bodies. The main factor in this perspective seems to be the volume of the vertebral body and its venous drainage via the basivertebral vein. A large vertebral body needs a large basivertebral vein for adequate venous drainage. As a result, the corresponding part of the anterior internal vertebral venous plexus also has to be large. This correlation is not applicable to the posterior internal vertebral venous plexus, as can be deduced from its typical shape.

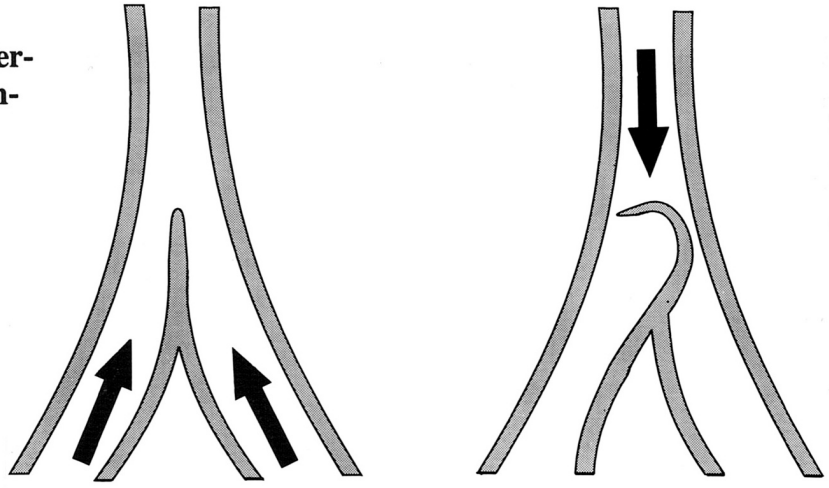
Although it seems reasonable to assume that the least extensive part of the posterior internal vertebral venous plexus is situated in the narrowest segments of the spinal canal, this apparently is not the case: in adults, the anteroposterior diameter of the spinal canal does not significantly change within the thoracic area (7). On the contrary, the sagittal diameter of the spinal cord (15) and the interpedicular diameter of the spinal canal (11) are smallest between the third and the ninth thoracic vertebrae. As the reduction of the interpedicular diameter in this area is more prominent than the reduction of the sagittal diameter of the spinal cord (11,15), the T3-T9 spinal epidural space is being narrowed proportionally. However, the posterior internal vertebral venous plexus appears to be very prominent in this area. Therefore, the relationship between the dimensions of the vertebral canal and the posterior internal vertebral venous plexus can not be used as a clear explanation for the typical configuration of the posterior internal vertebral veins.

6.4.4 Functional considerations.

The vertebral venous system is considered to constitute a valveless anastomotic system. As a result, the blood is supposed to flow in either direction (4,23,25), depending on changes of the intra-thoracic and intra-abdominal pressure (respiration, coughing) and hydrostatic factors (changes of posture), (9,10,23,25). We did not find anatomical valves in

the vertebral venous system, except the tricuspic valves that were encountered in both vertebral veins of cadaver N° 554. However, while flushing the vertebral venous system (prior to Araldite injection), is was noticed that, after reaching the "steady state", the flow rate during anterograde infusion (500 ml of saline, 50 cm above the heart) was much higher than the flow rate during retrograde infusion (under similar conditions). This suggests that the internal resistance of the vertebral venous plexus is correlated to the direction of the flow. Suh and Alexander (21) have observed monocuspidal valves at the bifurcations of the spinal cord veins. Those valves are held responsible for preventing retrograde flow into the second- order veins and the capillary network of the spinal cord. Possibly a similar situation may occur within the posterior internal vertebral venous plexus, especially when we take into account the "inversed V" configuration of the traversing veins in the thoracic segments of the plexus. The bifurcation of these venous channels may serve as a functional valve when blood is forced caudally. Under circumstances of flow in caudal direction, the veins may be (partially) closed by the "valve", which leads to an increase of the intravascular resistance and a slow-down of bloodflow in this part of the internal vertebral venous plexus (*Figure 6.6*). The functional significance of this anatomical configuration is unclear, but the presence of a functional valve in the posterior internal vertebral venous plexus might well explain the difficulties with imaging of these veins in vivo (vertebral venography and phlebography) and in vitro (injection studies in cadavers).

Figure 6.5 Supposed valve-mechanism, related to the direction of blood flow (arrows) within the traversing posterior thoracic internal vertebral veins.



The volume of the entire vertebral venous system is much larger than that of the contributing arteries (4,25). The excess of venous channels in relation to the arterial supply of the spinal epidural area has lead to many speculations about its function (4,9,25,27,28), but to date this disproportion has not been explained in functional terms. Herlihy (9) stated that the vertebral venous system has to be considered as a provision of nature to equalize venous pressure, to redistribute blood, and, in pathological conditions of the vena cava, to act as an alternate path for the continuation of the circulation (9). Others have stressed the role of the internal vertebral venous plexus in cerebrospinal fluid reabsorption (27).

Recently, the vertebral venous system has been implied in relation to evaporation-induced "selective brain cooling" via its connections with the external vertebral venous system (28).

6.5 Conclusions.

The present anatomical study offers further clarification concerning the structure of the posterior internal vertebral venous plexus. Although a wide segmental and interindividual variability was noticed in the morphology of this part of the vertebral venous system, a general pattern can be recognized in the configuration of the posterior internal vertebral venous plexus in aged cadavers. This pattern seems to correlate with the segmental distribution of hematomas in older patients that suffered SSEH. From the clinical point of view it is of interest to find out whether the morphology of the internal vertebral venous system is essentially different in younger people. In that catagory of patients, SSEH almost exclusively occurs in the cervicothoracic area (*Chapter 4*). However, this question remains unanswered, since "young" human bodies were not available for this anatomical study.

6.6 References.

1. Batson O.V. The function of the vertebral veins and their role in the spread of metastases. Ann Surg (1940) 112:138-149.
2. Batson O.V. The vertebral vein system. Am J Roentgen (1957) 78:195-212.
3. Breschet M.G. Recherches anatomiques, physiologiques et pathologiques sur le système veineux des os (1828-1832). Paris: Villaret.
4. Clemens H.J. Die Venensysteme der menschlichen Wirbelsäule. Morphologie und functionelle Bedeutung (1961). Berlin: Walter de Gruyter & Co.
5. Clemens H.J. Beitrag zur Histologie der Plexus venosi vertebrales interni. Z Mikrosk Anat Forsch (1961) 67:183-189.
6. Cruveilhier J. Traité d'Anatomie Descriptive (1834-1836). Tome 3-ième, Béchet Jeune, Paris: Veines du rachis.
7. Domisse G.F. The arteries, arterioles and capillaries of the spinal cord. Surgical guidlines in the prevention of postoperative paraplegia. Ann R Coll Surg Engl (1980) 62:369.
8. Greitz T., Lilliequist B. and Müller R. Cervical vertebral phlebography. Acta Radiol (1963) 57:353-365.
9. Herlihy W.F. Revision of the venous system: the role of the vertebral veins. Med J Aus (1947) 1:661-672.
10. Herlihy W.F. Experimental studies on the internal vertebral venous plexus. Essays Biol (1948) 1:151-163.
11. Hinck V.C., Clark W.M. and Hopkins C.E. Normal interpediculate distances (minimum and maximun) in children and adults. Am J Roentgen (1966) 97:141-153.
12. Hussey H.H. The vertebral venous system. JAMA (1976) 235:2524.

13. Lardé D. Les veines epidurales lombo-sacrées. Etude anatomique et radiologique. Intérêt de la phlébographie lombaire dans le diagnostic des discopathies. Thèse (1977) Nancy: P. Oudart.
14. Lasjaunias P. and Berenstein A. Surgical Neuroangiography. Part 3. Functional vascular anatomy of brain, spinal cord and spine (1990). Berlin: Springer-Verlag. p.85.
15. Lusted L.B. and Keats T.E. The Spine. In: Atlas of roentgenographic measurement. (1978) Year Book Medical Publishers, Chicago, pp. 113-134.
16. Meijenhorst G.C.H. Transfemoral epidural double-catheter venography in the diagnosis of lumbar disc herniation. Thesis (1979). Deventer: N.B. de Bruijn B.V.
17. Plaisant O., Cosnard G., Gillot C., Schill H. and Lassau J.P. MRI of the epidural space after Gelatin/Gadolinium venous injection. Surg Radiol Anat (1994) 16:71-75.
18. Plaisant O., Sarrazin J.L., Cosnard G., Schill H. and Gillot C. The lumbar anterior epidural cavity: the posterior longitudinal ligament, the anterior ligaments of the dura mater and the anterior internal vertebral venous plexus. Acta Anat (1996) 155:274-281.
19. Renard M., Masson J.P. and Lardé D. Contribution a l'etude anatomoradiologique du systeme veineux rachidien de la jonction lombo sacree. Bull Assocc Anat (Fr) (1979) 59(166):725-735.
20. Renard M., Masson J.P. and Lardé D. Les veines epidurales lombaires (Etude anatomique à propos de 31 cas). Bull Assocc Anat (Fr) (1979) 60:787-800.
21. Suh T.H. and Alexander L. Vascular system of the human spinal cord. Arch Neurol Psych (Chicago) (1939) 41:659-671.
22. Théron J. and Djindjian R. Cervicovertebral phlebography using catheterization. A preliminary report. Radiology (1973) 108:325-331.
23. Théron J. and Moret J. Spinal phlebography. Lumbar and cervical techniques (1978). Berlin: Springer-Verlag.
24. Uylings H.B.M., Van Eden C.G. and Hofman M.A. Morphometry of size/volume variables and comparison of their bivariate relations in the nervous system under different conditions. J Neurosci Meth (1986) 18:19-37.
25. Vogelsang H. Intraosseous spinal venography (1970). Amsterdam: Exerpta medica.
26. Wilkie R. and Beetham R. Transfemoral lumbar epidural venography. Spine (1980) 5:424-431.
27. Zenker W., Bankoul S., and Braun J.S. Morphological indications for considerable diffuse reabsorption of cerebro-spinal fluid in spinal meninges particularly in the areas of meningeal funnels. An electromicroscopical study including tracing experiments in rats. Anat Embryol (1994) 189:243-258.
28. Zenker W. and Kubik S. Brain cooling in humans - anatomical considerations. Anat Embryol (1996) 193:1-13.
29. Zwan van der A. and Hillen B. Araldite F as injection material for quantitative morphology of cerebral vascularization. Anat Rec (1990) 228:230-236.

Chapter 7

GENERAL DISCUSSION.

Since SSEH is a very rare disorder, the number of patients that are encountered within a single institution is very small. An extensive review of the literature appeared to be the most effective method to collect a significant number of patients. This implicated certain drawbacks, as there is the retrospective character of the study and the incomplete documentation of cases. Nevertheless, a large number of cases were collected that were reported adequately and confirmed by surgery or autopsy.

7.1 The clinical findings.

The major factors that are determining the prognosis after SSEH appeared to be [1] the localization of the hematoma, [2] the preoperative neurological condition, and [3] the operative interval (*Chapter 3*).

Mortality correlated highly with a cervical and cervico-thoracic localization of the hematoma, especially in patients with hypertension and/or coagulopathies. Cardiovascular complications (myocardial infarction and pulmonary embolism) were the main causes of death in this category of patients.

In patients with complete preoperative sensorimotor deficits, surgery in ≤36 hours correlated with favourable outcome. In patients with incomplete preoperative sensorimotor loss, favourable outcome correlated with operative decompression in ≤48 hours. It should be realized that these intervals are not absolute. They only show that the time is limited to establish the diagnosis and to initiate operative treatment in patients with signs and symptoms of (severe) spinal cord compression.

7.2 The pathophysiology of neural compression.

The pathophysiological mechanisms that are responsible for neurological deterioration in SSEH most probably are multifactorial. Both mechanical and circulatory factors (venous and arterial), should be considered in the pathogenesis of compression injuries of the spinal cord.

7.2.1 Mechanical factors.

Direct mechanical compression of the neural structures seems to be responsible for the early neurological signs and symptoms after hemorrhage. Conduction-blocks and damage to the myelin have been observed in peripheral nerves (24,26,31) and spinal cord (3,4) after focal compression. From experimental animal studies employing spinal cord compression, it has become clear that two pathological processes may affect central nerve fibers: [1] Wallerian-type degeneration, in which both axon and myelin desintegrate, and [2] demyelination in which the myelin is destroyed, leaving the axon in continuity through the lesion. Large experimental demyelinating lesions produce complete conduction-blocks. Smaller lesions allow conduction to continue, but conduction-velocity will be reduced, and the ability of these fibers to carry long trains of impulses faithfully is impaired. All these conduction-defects contribute to functional loss (25). There is evidence for remyelinization after transient spinal cord compression (15), which might explain late functional recovery following surgical relief of spinal cord compression.

7.2.2 Circulatory factors.

The pathophysiology of circulatory disturbances secondary to epidural spinal cord compression is suggested to follow a step-wise progression: venous congestion may appear, due to the impairment of the venous drainage of the spinal cord. This causes white matter edema and axonal swelling and is associated with early myelopathy. At this early stage of cord compression, spinal cord blood flow is not diminished. However, when the edema progresses, spinal cord blood flow becomes altered. If compression persists, irreversible ischemic damage will ensue (6). It has been suggested that compression of the spinal cord may interfere with the circulation of the main spinal arteries (22). This mechanism might cause ischemic infarctions of spinal cord segments remote from the area of compression and lead to irreversible neurological deficits. Based on the osseous peculiarities of the spinal canal and the vascularization of the spinal cord (see *Chapter 3*), the T3 to T7 spinal cord segments are suspected to be highly vulnerable and susceptable for this type of lesion. However, such a correlation did not appear in the present study. Therefore, it seems unlikely that obstruction of the main spinal arteries is a major factor in spinal cord compression.

7.3 Therapeutic window in SSEH.

The therapeutic window for successful treatment in SSEH is determined by the severity of neural compression and the rate of the progression of the pathophysiological mechanisms that are described above. It is well known from neurosurgical practice that slowly progressive epidural and intradural tumors cause severe distortion and impression of the spinal cord, without producing serious neurological deficits. This implicates that, within certain limits, the spinal cord is capable to adjust itself to conditions of (gradual and/or chronic) compression. It is also known that those patients usually do very well after operative decompression. Analogous to these experiences, it may be expected that slowly progresssive sensorimotor deficits in SSEH are caused by gradually progressive compression, and rapid neurological deterioration is caused by acute severe compression. Consequently, it has been suggested that a rapid neurological deterioration correlates with unfavorable outcome (12,13,37). In the present study we were not able to confirm this statement, most probably because of the long operative intervals in both groups (mean interval >43 hours), which in itself does result in unfavorable outcome. Only careful documentation and prompt surgical treatment (if necessary) of future patients with SSEH may lead to clinical evidence for a correlation between the compression speed of the spinal cord and neurological outcome after operative decompression.

7.4 The source of hemorrhage.

The majority of SSEHs appeared to be situated posteriorly or posterolaterally in the spinal epidural space. In young patients (0-40 years) SSEHs allmost exclusively occured in the cervicothoracic area, but in older patients (41-80 years) both the cervicothoracic and the thoracolumbar vertebral segments were preferably affected (*Chapter 4*). Histopathological findings that support the suspicion of a vascular malformation as the source of hemorrhage were lacking in most of the cases. Therefore, the most prominent question that remained, concerned the etiology of the SSEH. This incited to search for a morphological explanation for the hemorrhage within the spinal epidural space.

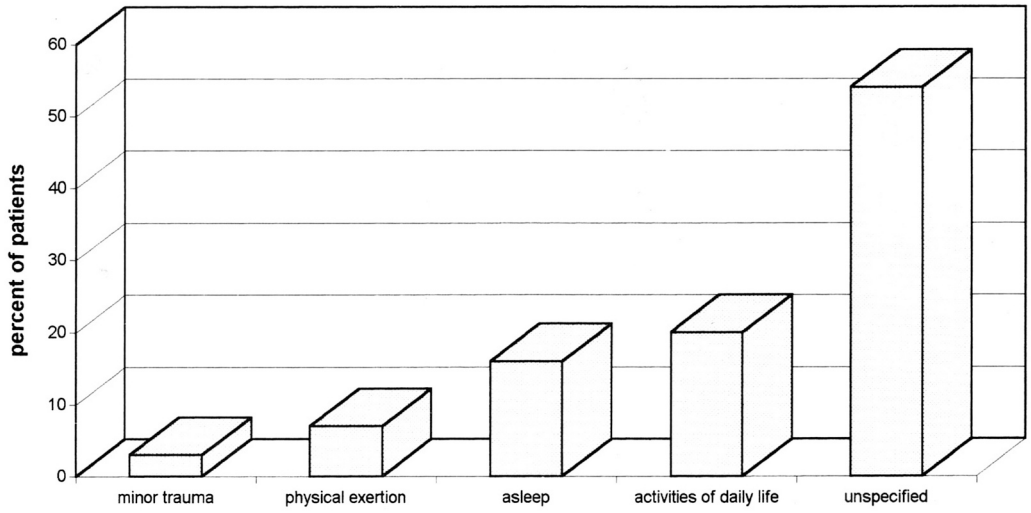
Reviewing the vascular anatomy of the spinal epidural space (*Chapter 5*), a rupture in the posterior part of the internal vertebral venous plexus seems a very likely cause of SSEH. According to the descriptions by Breschet (5) and Clemens (7), this vascular network has a dense concentration of veins at the C6 to T3 vertebral segments and in the lumbar area. These findings might be the explanation for the posterior and posterolateral localization and the segmental distribution of SSEH that have been observed in a series of 333 cases (*Chapter 4*). However, based on the literature, no anatomical explanation could be offered for the the differences regarding the preferential localization of SSEH that we observed between the two age-groups (0-40 years and 41-80 years).

Since details about the segmental and interindividual variability of the internal vertebral venous plexus were not available from the literature, a human cadaver study was performed aiming at describing the anatomy of this venous network following injection with a modern polymere (*Chapter 6*). From this study, it appeared that the anterior internal vertebral venous plexus has a very constant pattern, both segmentally and interindividually. Part of this plexus is lying anterior to the posterior longitudinal vertebral ligament. While descending the spinal canal, the anterior internal vertebral veins become more voluminous and more pronounce, down to and including the L5 segment. An abrupt decrease in volume is noticed in the sacral area. On the contrary, the anatomical variation of the posterior internal vertebral venous plexus is wide. The description of the posterior internal vertebral venous plexus, as displayed by Breschet (5) and Clemens (7), is a simplification. In the present study, the plexus is found to be most extensive in the cervicothoracic and the lumbar areas. This is in agreement with the findings of Breschet (5) and Clemens (7). Moreover, in most cadavers the suboccipital plexus (at C1) was also quite massive. In contrast to the rest of the posterior plexus (which lies freely within the epidural fat), this part of the plexus is surrounded by connective tissue. Two types of traversing venous channels have been noticed in the thoracic area: [1] in some cadavers these veins were very small in size and in number [2] in other cadavers the traversing veins were wide and numerous. Based on the pattern of the thoracic part of the plexus, (at least) two different types of the posterior internal vertebral venous plexuses can be discerned: [1] a rather thin plexus, with predominantly small, single traversing thoracic venous channels, and [2] a voluminous plexus, with wide, numerous traversing thoracic venous channels. In both types, an increase of the number of veins was observed in the cervicothoracic and the lumbar parts of the posterior internal vertebral venous plexus. These findings support the idea that the posterior internal vertebral venous plexus is the most likely source of spontaneous spinal epidural hemorrhage.

7.5 The pathophysiology of hemorrhage.

The pathophysiological mechanisms that result in a rupture of the spinal epidural vessels are unknown. Vasculitis was found in only one patient with SSEH (12). Maybe anticoagulant therapy or coagulation disorders facilitate the occurrence of SSEH. However, there are no data to support this idea. Physical exertion (9,14,23,29), coughing (20), sneezing (27,32), voiding (1), vomiting (21), and minor trauma (8,10,17,33,35) are suggested to play a role, all assumed to cause a sudden increase of pressure within the vertebral venous plexus with subsequent venous rupture. The activities preceding a SSEH are listed in *Figure 7.1.*

Figure 7.1 Activities preceding SSEH (N = 333).



It is very unlikely that those factors play a decisive role in the pathogenesis of SSEH. Most probably, bleeding occurs because of the rupture of a vein that has become vulnerable, due to pathological changes of the venous wall. When we suppose the incidence of those pathological changes to be equally distributed over the entire internal vertebral venous plexus, it may be concluded that the distribution of hematomas along the spinal canal is proportional to the quantity of veins within this venous system. In this perspective, the segmental distribution of SSEH that we observed in the series of 333 cases (see *Chapter 3, Figure 3.1*) can be explained by the regional differences in the morphology of the posterior internal vertebral venous plexus.

Due to the erectness of man, the internal vertebral venous plexus is subject to hydrostatic forces. This may cause spinal epidural venous distension and varicosis, which may be aggravated by age-related degenerative changes that occur within the venous wall (loss of tissue elasticity). The lumbar part of the internal vertebral venous plexus seems to be accident-prone to this condition, because of its caudal position. This might explain the thoracolumbar SSEH-peak that has been observed in the category of patients between 41 and 80 years. In this context, it is of interest to mention that several cases of symptomatic "spinal varices" have been reported (11,16,38). The majority of those patients suffered lumbar radicular symptoms due to root compression by thrombosed lumbar epidural varicose veins. Dickman et al. (11) described a patient with posterior spinal cord compression caused by thrombosed epidural varices at the cervicothoracic junction. This illustrates that spinal epidural varicosis is a reality and that thrombosis within those veins may occur.

The physiological oscillation of body-length has been studied by several authors (28,30,34). It has become clear that there is a circadian variation, approximating 1.1% of stature, which is mainly caused by the decrease of the water-content of the intervertebral disc in the erect position, and an increase in the supine position (i.e. during sleep) (30,34). This means that the average oscillation of length will be between 1 to 2 cm every 24 hours in most individuals (34). Due to this, the intrinsic structures of the spinal column (i.e. the spinal cord, the dural sack and the epidural stuctures) are subject to repeatedly shrinking and stretching. Besides from this process, osteoporosis and intervertebral disc degeneration are responsible for a progressive shortening of the spinal column (due to aging), which causes descensus of the spinal cord and dural sack within the spinal canal. The combination

of those conditions may result in compression and distortion of the spinal epidural structures and may also be responsible for weakening of the internal vertebral venous plexus.

The direction of blood flow within the vertebral venous system depends on the position of the body and the relationship between the intra-thoracic and intra-abdominal pressure (2,18,19,36). It can be imagined that incidently blood flow is be very slow or even absent anywere in this plexus. Under these conditions, venous thrombosis may occur in a previously weakened vein. Thrombosis may lead to engorgement and distension, and this may cause a rupture of the venous wall and the development of a spinal epidural hematoma. The use of anticoagulants or the presence of a coagulopathy not necessarily rules out a similar mechanism in this category of patients, because even under these conditions thrombosis may occur. However, a simple rupture or leak of a weakened vessel within the internal vertebral venous plexus, without preceding venous thrombosis, is another possible cause of SSEH.

Obviously, the pathophysiological events that precede spontaneous spinal epidural hemorrhage remain speculative. Further study of the morphology and the physiology of the vertebral venous system may result in a better understanding of the function of the internal vertebral venous plexus and its pathological conditions.

7.6 References.

1. Bain W. A case of haematorrachis. Br Med J (1897) 2:455.
2. Batson O.V. The function of the vertebral veins and their role in the spread of metastases. Ann Surg (1940) 112:138-149.
3. Blight A. Delayed myelination and macrophage invasion. A candidate for secundary damage in spinal cord injury. Centr Nerv Syst Trauma (1985) 2:299-315.
4. Blight A. Morphometric analysis of experimental spinal cord injury in the cat: relation of injury intensity to survival of myelinated axons. Neurosci (1986) 19:321-341.
5. Breschet G. Recherches anatomiques, psychologiques et pathologiques sur le système veineux et spécialement sur les caveaux veineux des os. Paris, Villaret et Cie. (1828-1832).
6. Byrne T.N. and Waxman S.G. Spinal cord compression: diagnosis and principles of management. (1990) Philadelphia: F.A. Davis Company. pp. 151-154.
7. Clemens H.J. Die Venensysteme der menschlichen Wirbelsäule. Morphologie und functionelle Bedeutung. (1961) Berlin: Walter de Gruyter & co..
8. Cooper D.W. Spontaneous spinal epidural hematoma. Case report. J Neurosurg (1967) 26:343-345.
9. Correa A.V. and Breasley B.A.L. Spontaneous cervical epidural hematoma with complete recovery. Surg Neurol (1978) 10:227-228.
10. Devadiga K.V. and Gass H.H. Chronic lumbar extradural hematoma simulating disc syndrome. J Neurol Neurosurg Psychiat (1973) 36:255-259.
11. Dickman C.A., Zabramski J.M., Sonntag V.K.H. and Coons S. Muelopathy due to epidural varicose veins of the cervicothoracic junction. Case report. J Neurosurg (1988) 69:940-941.
12. Flaschka G., Sutter B., Ebner F., Klein G.E. and Tilz G. Das spinale Epiduralhämatom. Langzeitergebnisse von 4 eigenen Fällen. Nervenarzt (1990) 61:629-633.

13. Foo D. and Rossier A.B. Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas. Surg Neurol (1981) 15:398-401.
14. Ghanem M.B. and Ivan L.P. Spontaneous spinal epidural hematome in an eight year old boy. Neurology (Minneap) (1978) 28:829-832.
15. Gledhill R.F. and McDonald W.I. Morphological characteristics of central demyelinisation and remyelinisation: a single fiber study. Ann Neurol (1977) 1:552-560.
16. Gümbel U., Pia H.W. and Vogelsang H. Lumbosacrale Gefäß-anomalien als Ursache von Ischialgien. Acta Neurochir (1969) 20:131-151.
17. Hehman K. and Norrel H. Massive chronic spinal epidural hematoma in a child. Am J Dis Child (1968) 116:308-310.
18. Herlihy W.F. Revision of the venous system: the role of the vertebral veins. Med J Aus (1947) 1:661-672.
19. Herlihy W.F. Experimental studies on the internal vertebral venous plexus. Essays Biol (1948) 1:151-163.
20. Jackson R. Case of spinal apoplexy. Lancet (1869) 2:5-6.
21. Kaplan L.I. and Denker P.G. Acute non-traumatic spinal epidural hemorrhage. Am J Surg (1949) 78:356-361.
22. Lazorthes G., Gouaze A., Zadeh J.O., Santini J.J., Lazorthes Y. and Burdin P. Arterial vascularization of the spinal cord. Recent studies of the anastomotic substitution pathways. J Neurosurg (1971) 35:253-262.
23. Lepoire J., Tridon P., Montaut J. and Germain F. L'hématome extra dural rachidien spontané. Neurochirurgie (1959) 7:298-313.
24. MacGregor R.J., Sharpless S.K. and Luttges M. A pressure vessel model of nerve compression. J Neurol Sci (1975) 24:295-304.
25. McDonald W.I. Mechanisms of functional loss and recovery in spinal cord damage. Ciba Found (1975) 34:23-33.
26. Ochoa J., Fowler T.J. and Gilliatt R.W. Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. J Anat (Lond) (1972) 113:433-435.
27. Pear B.L. Spinal epidural hematoma. Am J Roentgenol Radium Ther Nucl Med (1972) 115:155-164.
28. Puky de P. The physiological oscillation of the lenght of the body. Acta Orthop Scand (1935) 6:338-347.
29. Rao B.D., Rao K.S., Subrahmanian M.V. and Raghava Reddy M.V. Spinal epidural hemorrhage. Br J Surg (1966) 53:649-650.
30. Reilly T., Tyrrell A and Troup J.D.G. Circadian variation in human stature. Chronobiol Intern (1984) 1:121-126.
31. Rudge P., Ochoa J. and Gilliatt R.W. Acute peripheral nerve compression in the baboon. J Neurol Sci (1974) 23:403-420.
32. Scott B.B., Quisling R.G., Miller C.A. and Kindt G.W. Spinal epidural hematoma. JAMA (1976) 235:513-515.
33. Svien H.J., Adson A.W. and Dodge H.W. Lumbar extradural hematoma. Report of a case simulating protruded disc syndrome. J Neurosurg (1950) 7:587-588.
34. Tyrrell A.R., Reilly T. and Troup J.D.G. Circadian variation in stature and the effect of spinal loading. Spine (1985) 10:161-164.
35. VerBrugghen A. Extradural spinal hemorrhage. Ann Surg (1946) 123:154-159.
36. Vogelsang H. Intraosseous spinal venography (1970) Amsterdam: Exerpta medica.
37. Von Klossek H. and Hüller E. Zur problematiek der spontanen spinalen Epiduralhämatome. Zeitbl Neurochirurgie (1984) 45:116-123.

38. Zimmerman G.A., Weingarten K. and Lavyne M.H. Symptomatic lumbar epidural varices. J Neurosurg (1994) 80:914-918.

Summary.

This thesis concerns a clinical study of the spontaneous spinal epidural hematoma (SSEH) and a study of the vascu¨lar anatomy of the spinal epidural space. In particular the morphology of the internal vertebral venous plexus is studied, in an attempt to find an explanati¨on for the etiology of this disorder.

In ***Chapter 1*** the background of this study is described. After meeting with two patients with SSEH in neurosurgical practice, it was decided to study the literature on this item. It appeared that the etiology of SSEH remained obscure in most cases. Despite of the large number of cases that have been reported in the medical literature after the first publication by Jackson (1869), little is known about the etiology, the clinical course and the factors that are determining postoperative recovery. Since the number of patients that are treated within one single institution is very limited, it was decided to collect case-reports from the literatu¨re to obtain a significant body of data for further study on this subject.

In ***Chapter 2*** three patients are presented, as to illustrate the clinical picture, the diagnostic procedures, the therapeutic approaches and the postoperative course of SSEH. Since the introduction of MRI, the number of reported cases that recover spontaneously is rising. This can be explained by the fact that the diagnosis of SSEH is greatly facilitated by this new technique, enabling more cases with a benign natural course to be detected. In general, SSEHs present as an acute syndrome with symptoms of progressive spinal cord and/or nerve root compression that requires urgent operative decompression.

The factors that affect postoperative outcome in patients who have suffered SSEH are described in ***Chapter 3***. Threehunderd and thirty cases of SSEH from the literature and the three cases from chapter 2 are reviewed. Attention was focussed on sex, age, medical history, mortality, size and position of the hematoma, vertebral level of the hematoma, preoperative neurological condition, operative interval and postoperative result. It appeared that sex, age and size and position of the hematoma did not correlate with postoperative outcome. Mortality correlated highly with cervical or cervicothoracic hematomas, especially in patients with cardiovascular disease and those undergoing anticoagulant therapy. Incomplete preoperative sensorimotor deficit correlated highly with favorable outcomes ($P < 0.0005$). Recovery was significantly better when decompression was performed in ≤ 36 hours in patients with complete sensorimotor loss ($P < 0.05$) and in ≤ 48 hours in patients with incomplete sensorimotor deficit ($P < 0.005$). It is concluded that the critical factors for recovery after SSEH are the severity of preoperative neurological deficit and the operative interval. The vertebral level of the hematoma did not correlate with postoperative results, which suggests that local compression, rather than direct arterial obstruction is the main factor in producing neurological deficits.

In ***Chapter 4*** a study of the etiology of the spontaneous spinal epidural hematoma is described, based on the series of patients as listed in the previous chapter. One hundred and eighty two patients (54.6%) had no medical history. Coagulopathies (disease or medication) were found in 69 patients (20.7%), hypertension was present in 39 patients (11.7%), and 20 patients (6%) were both hypertensive and adjusted on oral anti-coagulants. Although many authors have advocated the causal relationship between

hypertension and the occurrence of SSEH, this was falcified in the present study. The segmental distribution in the whole series of SSEH shows a peak at the levels C5-T2 and a second peak at T10-L1. It appeared that the segmental distribution strongly depends on the age. Below the age of 40 years, a lower thoracic or lumbosacral localization of SSEH is exceptional. The second peak that was observed in the total series of 333 patients almost exclusively resulted from the patients between 41-80 years of age. This suggests the existence of two separate groups among the patients with SSEH. Histopathological investigation was performed in 176 cases (52.8%). Only in 18 patients (5.4%) this showed a more or less defined "vascular anomaly". Many theories have been advocated about the etiology of SSEH. Despite suggestions about the rupture of epidural veins, arteries, cryptic angiomas, vascular malformations or hemangiomas as the possible cause of SSEH, none of the authors managed to give supportive evidence for their theory, neither statistically nor on anatomical basis.

This incited to perform a review of the vascular anatomy of the spinal epidural space, which is presented in ***Chapter 5***. Based on this review, the different theories about the etiology of SSEH are discussed. It seems that a lack of familiarity with the normal vascular structures of the spinal epidural space has resulted in erroneous conclusions about the etiology of SSEH. In some reports the authors suggested that a vascular anomaly is the cause, based on the observation of venous clusters in an epidural hematoma. It should be taken into consideration that in these cases the hematomas enclosed congested or distended vessels of the internal vertebral venous plexus. Secondly, the poorly recognised "cluster-type" arteries might be responsible for some cases of SSEH; these structures can easily be mistaken for a vascular anomaly. The anatomical dimensions of the posterior internal vertebral venous plexus (as deduced from the literature), and the segmental distribution of SSEH in 333 cases, suggest that the majority of these hematomas result from a rupture of this particular vascular network. However, no data are available on the segmental and interindividual variability of the internal vertebral venous plexus from the (very scarce) literature. Therefore, a human cadaver study of the anatomy of the spinal venous system was performed.

In ***Chapter 6*** the morphological study of the human internal vertebral venous system is described. The vertebral venous systems of ten fresh human cadavers, between 64 and 93 years of age, were injected with Araldite CY 221 mixture. A specific injection procedure was developed to ensure complete filling of the spinal venous plexus. All cadavers were dissected and the posterior and anterior internal vertebral venous plexuses were studied. The anterior part of the internal vertebral venous plexus was fairly constant in all specimens. On the contrary, the posterior internal vertebral venous plexus showed a striking segmental and interindividual variability. Nevertheless, a general pattern could be recognized in the configuration of the posterior internal vertebral venous plexus in these old-aged cadavers. This pattern seems to correlate with the segmental distribution of SSEH in the category of patients aging 41-80 years. In the thoracic area, two types of traversing veins are observed. Both types show a more or less symmetrical "inversed V" configuration. No anatomical valves were observed. Nevertheless, antegrade infusion the femoral veins) of the vertebral venous system appeared to proceed much faster than retrograde infusion (via the superior caval vein). It is concluded that the classical picture of the internal vertebral venous plexus as described by Breschet and Clemens is a simplification of the actual anatomy. Especially in the posterior part, segmental and interindividual differences are prominent. The preferential direction of flow during

flushing suggests the presence of functional valves, which are probably located in the thoracic part of the posterior internal vertebral venous plexus, resulting from the typical shape of the veins in this area. This explains the difficulties with imaging of the posterior part of the internal verte¨bral venous plexus in vivo as well as in post mortem studies.

In ***Chapter 7*** the clinical and anatomical studies are evaluated. The pathophysiological mechanisms that might be responsible for neurological deterioration in SSEH are discussed. It seems that both mechanical and circulatory factors are responsible. The therapeutic window for treatment in SSEH is determined by the severity of neural compression and the evolution of the mechanical and circulatory disturbances. Furthermore, it is concluded that the data of 333 cases of SSEH, and the findings after dissection of ten human cadavers, are highly suggestive for a correlation between the SSEH and the posterior internal vertebral venous plexus. The pathophysiology of hemorrhage remains speculative, but it is suggested that the process is initiated by pathological changes that occur within the venous wall of this plexus. When the incidence of those pathological changes is supposed to be equally distributed over the individual veins composing the entire internal vertebral venous plexus, it may be concluded that the distribution of hematomas along the spinal canal is proportional to the quantity of veins within this venous system. In that perspective, the segmental distribution of SSEH seems to be explained by the regional differences in the morphology of the posterior internal vertebral venous plexus. The combination of hydrostatic forces (due to the erectness of man) and age-related degenerative changes (loss of elasticity of the venous walls) may cause distension and varicosis of the (valveless) spinal epidural veins. The possible role of venous thrombosis in the etiology of SSEH is suggested, based on the above mechanism and reports in the literature of symptomatic spinal epidural varicosis. Besides, the physiological oscillation of body-length and the degenerative shortening of the spinal column may result in shrinking and stretching of the spinal epidural structures, and may lead to weakening of the internal vertebral venous plexus. Further study of the morphology and physiology of the vertebral venous system may result in a better understanding of the function of this system and possibly may elucidate its pathological conditions.

Samenvatting.

Dit proefschrift heeft als onderwerp het spontane spinale epidurale hematoom (SSEH). Het betreft een klinische studie van een grote serie patiënten die zijn verzameld uit de medische literatuur, en een anatomische studie naar het bloedvaatstelsel van de spinale epidurale ruimte (het buiten het ruggenmergsvlies gelegen deel van het wervelkanaal). In het bijzonder gaat de aandacht uit naar de interne vertebrale veneuze plexus (een netwerk van aderen dat als een soort vlechtwerk rondom het ruggenmergsvlies ligt), omdat hierin mogelijk de verklaring ligt voor de oorsprong van het SSEH.

In ***Hoofdstuk 1*** wordt de achtergrond van de studie beschreven. In 1869 werd door Jackson voor het eerst verslag gedaan van een patiënt die was overleden aan de gevolgen van een bloeding in de epidurale ruimte van het halswervelkanaal. De verschijnselen bestonden uit toenemende verlammingen van de armen en de ademhalingsspieren, zodanig dat de patiënt uiteindelijk ten gevolge van ademhalingsstoornissen overleed. Bij sectie werd een grote bloeding gevonden in het wervelkanaal, buiten het ruggenmergsvlies, die aanleiding was voor ernstige beknelling van het ruggenmerg ter plaatse. Een oorzaak voor de bloeding werd niet gevonden. De diagnose werd gesteld op een spontane spinale epidurale bloeding (SSEH). Sedertdien zijn er enkele honderden ziektegeschiedenissen gepubliceerd over patiënten met een SSEH. Opmerkelijk is dat de oorzaak van de bloeding in het overgrote deel van de gevallen niet kon worden vastgesteld. Dit heeft geresulteerd in een groot aantal theorieën over de mogelijke oorzaak. De verscheuring van een slagadertje, van een adertje, van een vaatmisvorming of van een vaattumortje zijn alle genoemd als een mogelijke oorzaak. Tevens is er een verband gesuggereerd met hoge bloeddruk, hart- en vaatziekten, bloedstollingsstoornissen (o.i.v. bloedverdunners of tgv. bloedstollingsziekten (o.a. leukemie, leverziekten)) en zwangerschap. Andere veronderstellen mechanische factoren (bewegingen van de wervelkolom) of situaties die aanleiding geven tot een acute drukverhoging binnen het wervelkanaal (hoesten, niezen, tillen, persen, braken, etc.) als de mogelijke oorzaak van het SSEH. Geen van deze theorieën wordt echter gesteund door anatomische, histologische of statistische bewijsvoering.

Omdat het SSEH zeldzaam is, bestaat er weinig ervaring met dit ziektebeeld. Snelle herkenning van het ziektebeeld is noodzakelijk, omdat bij de meeste patiënten een met spoed uitgevoerde operatie, waarbij de beknelling van het ruggenmerg wordt opgeheven, de enige kans is op het terugkeren van de neurologische functies (vaak hebben deze patiënten een [gedeeltelijke of complete] dwarslaesie).

Het doel van de huidige studie is een beschrijving te geven van het ziektebeeld, om inzicht te verstrekken in de klinische verschijnselen, en om de factoren te bepalen die van invloed zijn op het postoperatieve neurologische herstel. Tevens zal aan de hand van een studie van de anatomie van de binnen de epidurale ruimte gelegen bloedvaten getracht worden een mogelijk verband te leggen met de oorzaak van deze bloedingen. In

In ***Hoofdstuk 2*** wordt een beschrijving gegevens van drie patiënten, aan de hand waarvan het klinische beeld, de diagnostiek, de behandeling en het postoperatieve beloop van het SSEH worden geïllustreerd.

De factoren die van invloed zijn op het herstel ná operatieve behandeling (het postoperatieve herstel) zijn beschreven in ***Hoofdstuk 3***. Driehonderd en dertig

patiënten die zijn verzameld uit de literatuur, én de drie patiënten die zijn beschreven in het vorige hoofdstuk, werden bestudeerd. Daarbij is gekeken naar de invloeden van de leeftijd, het geslacht, de ziektegeschiedenis (zoals hoge bloeddruk, bloedstollings-stoornissen, etc.), de grootte van de bloeding, de positie van de bloeding ten opzicht van het ruggenmerg (ervóór, erachter of ernaast), de plaats van de bloeding ten opzichte van de wervelkolom (ter hoogte van nekwervels, borstwervels, lendenwervels en/of heiligbeen), de neurologische toestand vóór operatie, de tijd die verstreek vanaf het begin van de bloeding tot het moment van operatie (operatie-interval) en het postoperatieve resultaat (onveranderd, incompleet herstel, compleet herstel).

Het bleek dat de leeftijd, het geslacht, de grootte van de bloeding en de positie van de bloeding geen significante invloed hadden op het postoperatieve herstel. Een statistisch significant verband werd gevonden tussen overlijden en de aanwezigheid van een SSEH ter hoogte van de halswervelkolom (cervicale wervelkolom) en ter hoogte van de overgang van de halswervelkolom naar de borstwervelkolom (cervico-thoracale overgang), met name bij patiënten met hart- en vaatziekten en bij gebruikers van bloedverdunners. Bij patiënten die vóór de operatie een gedeeltelijke uitval van motorische en/of sensibele functies hadden (incomplete dwarslaesie) was het postoperatieve herstel statistisch significant beter dan bij patiënten met volledige uitval (complete dwarslaesie) ($P < 0.0005$). Bij patiënten met een incomplete dwarslaesie was het postoperatieve herstel statistisch significant beter indien de operatie binnen 48 uur was verricht, gerekend vanaf het optreden van de eerste uitvalsverschijnselen. Bij patiënten met een complete dwarslaesie was het postoperatieve herstel statistisch significant beter indien de operatie binnen 36 uur was verricht. Hieruit kon worden geconcludeerd dat het preoperatieve neurologische beeld en het operatie interval de belangrijkste factoren zijn voor postoperatief herstel na een SSEH.

In ***Hoofdstuk 4*** wordt een beschrijving gegeven van de ontstaanswijze van het SSEH, aan de hand van het patiëntmateriaal uit het voorgaande hoofdstuk. Van de gehele groep patiënten waren er 182 voordien gezond (54.6%). Stollingsstoornissen kwamen voor bij 69 patiënten (20.7%), hoge bloeddruk bij 39 patiënten (11.7%), en bij 20 patiënten (6%) werden zowel stollingsstoornissen als een hoge bloeddruk vastgesteld. Hoewel door vele auteurs een oorzakelijk verband werd gesuggereerd tussen de aanwezigheid van een hoge bloeddruk en het SSEH, kon uit de gegevens van deze studie geen significant verband worden aangetoond. Uit onderzoek naar de segmentale verdeling van de bloedingen binnen het wervelkanaal bleek dat het SSEH bij voorkeur voorkomt tussen de 5^e halswervel en de 2^e borstwervel (C5-T2) en tussen de 10^e borstwervel en de 2^e lendenwervel (T10-L2). Deze verdeling bleek een opmerkelijk relatie te vertonen met de leeftijd: [1] Bij patiënten tussen de 0-40 jaar bevonden de bloedingen zich vrijwel uitsluitend in het cervico-thoracale traject, terwijl een bloeding in het thoracolumbale en sacrale traject hoogst uitzonderlijk was. [2] Bij patiënten tussen de 41-80 jaar bleken de gebieden C5-T2 en T10-L2 bij voorkeur te zijn aangedaan. Deze waarneming suggereert de aanwezigheid van twee aparte groepen binnen de serie patiënten met een SSEH.

Bij 176 patiënten (52.8%) werd microscopisch (histopathologisch) onderzoek verricht van het bloedings-materiaal dat tijdens de operatie of bij sectie was verwijderd. Slechts in 18 gevallen (5.4%) leverde dit aanwijzingen voor een al dan niet omschreven vaatafwijking, die door de auteurs verantwoordelijk werd gesteld voor het ontstaan van de bloeding. In alle andere gevallen werden geen afwijkingen gevonden.

Teneinde de mogelijkheid te onderzoeken van een meer "anatomische" verklaring van het SSEH, waarbij moet worden aangenomen dat de bloeding voortkomt uit een "normale" spinale epidurale vaatstructuur, werd een literatuurstudie verricht naar de vasculaire anatomie van de spinale epidurale ruimte. De resultaten van deze studie zijn beschreven in ***Hoofdstuk 5***. Tegen de achtergrond van dit onderzoek werden de verschillende theorieën over het ontstaan van het SSEH geëvalueerd. Dit leverde de indruk dat een algemeen gebrek aan kennis van de spinale epidurale vasculaire anatomie heeft geleid tot onjuiste conclusies over de oorzaak van het SSEH. In sommige gevallen werd door de auteur(s) een vaatmisvorming aangenomen, terwijl de beschrijvingen in de publicatie sterk wezen in de richting van de interne vertebrale veneuze plexus. Daarnaast bevinden zich op het ruggenmergsvlies zogenaamde "cluster-type" arteriën, die gemakkelijk kunnen worden aangezien voor een arterioveneuze malformatie (vaatmisvorming).

Uit de beschrijvingen van de interne vertebrale veneuze plexus door de anatomen Breschet (in 1828-1832) en Clemens (in 1961) blijkt dat er segmentale verschillen bestaan in het kaliber van de aderen alsook in het aantal aderen, waarbij met name een grote concentratie van vaten in het cervicothoracale gebied (C5-T3) wordt gesuggereerd. Dit zou een verklaring kunnen zijn voor de segmentale verdeling van het SSEH, zoals die in hoofdstuk 4 is beschreven.

Aangezien er uit de literatuur geen gegevens bekend zijn over de interindividuele en segmentale variabiliteit van de interne vertebrale veneuze plexus, werd een eigen anatomisch onderzoek verricht naar dit vaatnetwerk. In ***Hoofdstuk 6*** wordt dit onderzoek beschreven. Het vertebrale veneuze systeem (dit is het netwerk van aderen rondom de wervelkolom en binnen in het wervelkanaal) van tien menselijke lichamen werd ingespoten met een Araldite CY 221 mengsel (dit is een modern polymeer). Er is een speciale injectie methodiek ontwikkeld, om zeker te zijn van een volledige vulling van de spinale veneuze plexus. De leeftijd van de lichamen varieerde tussen de 64 en 93 jaar. Alle lichamen (N=10) werden na het uitharden van het polymeer ontleed, waarna de posterieure (het achterliggende gedeelte) en de anterieure (het voorliggende gedeelte) interne vertebrale veneuze plexus werden bestudeerd.

Het voorste gedeelte van de plexus had een zeer constant en gelijkvormig patroon in alle lichamen. Daarentegen vertoonde het achterste gedeelte van de interne vertebrale veneuze plexus aanzienlijke segmentale en interindividuele verschillen. Desondanks was het mogelijk ook in dit gedeelte van de veneuze plexus een globaal vaatpatroon te onderscheiden. Dit patroon vertoont, voor wat de vaatverdeling betreft, gelijkenis met de segmentale verdeling van het SSEH zoals die werd aangetroffen bij de groep patiënten van 41-80 jaar (zie hoofdstuk 4).

In het thoracale gedeelte (ter hoogte van de borstwervelkolom) van de posterieure interne vertebrale veneuze plexus vertoonden de overdwars verlopende aders een opmerkelijk verloop. Globaal leek dit op een "omgekeerde V". Er werden in dit traject geen kleppen in de aderen aangetroffen. Daarentegen was tijdens de injectieprocedure gebleken dat inspuiting via de liesaderen (de venae femorales) veel gemakkelijker en sneller verliep dan inspuiting via de bovenste grote holle ader (de vena cava superior). Deze bevinding vindt mogelijk zijn verklaring in de hierboven genoemde "omgekeerde V" vorm: het is denkbaar dat bloed dat van boven naar beneden stroomt in de posterieure interne vertebrale veneuze plexus een weerstandsverhoging ondervindt doordat het schotje, dat zich bevindt op de splitsingsplaats van de twee aderen, als een klepje gaat fungeren dat onder invloed van de benedenwaartse druk voor een (gedeeltelijke?)

afsluiting zorgt van één of beide vaten. Dit resulteert in verhoging van de vaatweerstand en in vertraging van de neerwaartse bloedstroom. Daarentegen ondervindt bloed dat van beneden naar boven wordt verplaatst géén hinder, omdat het "klepje" in dat geval in de richting van de stroom wijst.

Al deze bevindingen resulteren in de vaststelling dat de weergaven van de interne vertebrale veneuze plexus door Breschet en Clemens een simplificatie zijn van de werkelijke situatie. Het anatomisch onderzoek van de interne vertebrale veneuze plexus vond plaats bij oude lichamen. Aangezien er geen jonge lichamen beschikbaar waren, kan niet worden beoordeeld of er leeftijdsafhankelijke verschillen bestaan in de morfologie van dit vaatsysteem. Hoewel anatomische kleppen niet konden worden aangetoond in de interne vertebrale veneuze plexus, lijkt het injectie-onderzoek aanwijzingen te hebben gegeven voor het bestaan van een soort fysiologische (functionele) klep, die voortkomt uit de typische configuratie van de dwars verlopende adernetwerken in het thoracale traject van dit systeem.

In ***Hoofdstuk 7*** worden de resultaten van de klinische en anatomische studies geëvalueerd. De mechanismen die een rol spelen bij het ontstaan van neurologische verschijnselen worden besproken. Het lijkt waarschijnlijk dat zowel mechanische factoren (beschadigingen door rechtstreekse druk op de zenuwbanen) als circulatie stoornissen (beschadiging van zenuwweefsel ten gevolge van stoornissen in de aanvoer en afvoer van het bloed) verantwoordelijk zijn voor de ontwikkeling van de dwarslaesie-verschijnselen.

Uit de bevindingen van de hierboven beschreven studies kan worden geconcludeerd dat een ruptuur is van één van de vaten van de posterieure interne vertebrale veneuze plexus de meest waarschijnlijke oorzaak van het SSEH. Het verschil dat werd gevonden in voorkeursplaats van de bloedingen tussen jonge patiënten (0-40 jaar) en oudere patiënten (41-80 jaar) zou verband kunnen houden met (leeftijds-afhankelijke) veranderingen die in dit vaatsysteem optreden. Mogelijk spelen hierbij hydrostatische krachten (de invloed van de zwaartekracht op het vaatstelsel ten gevolge van de rechtop gaande houding van de mens) en wrijvingskrachten die samen hangen met de dagelijks optredende verkorting en verlenging van de wervelkolom een oorzakelijke rol.

Omdat er nog onvoldoende bekend is over de morfologie (er zijn met name geen gegevens beschikbaar over de anatomie van dit vaatstelsel bij jonge individuen) en de fysiologie van het vertebrale veneuze systeem, is verder onderzoek noodzakelijk. Dit zou kunnen leiden tot een beter inzicht in de functie van dit vaatstelsel en de ermee samenhangende pathologische omstandigheden.

Nawoord.

Vanaf deze plek wil ik allen die op directe of indirecte wijze een bijdrage hebben geleverd aan de totstandkoming van dit proefschrift oprecht bedanken. Een aantal van hen wil ik daarbij in het bijzonder noemen.

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Tenslotte en bovenal Marije. Lieve Marije, het schrijven van dit boekje heb ik nooit als een last ervaren. Ongetwijfeld komt dat mede doordat jij er vanaf het begin achter stond en mij de ruimte gaf om mij zo nu en dan achter de PC terug te trekken. Mede daarom draag ik dit werk op aan jou, en aan onze Bas en Merel.

Curriculum vitae

De schrijver van dit proefschrift werd op 31 juli 1960 te Groningen geboren. Nadat hij in mei 1978 aan het St. Maartenscollege te Groningen het diploma Atheneum B behaalde, begon hij in september van dat jaar aan de studie geneeskunde aan de Rijksuniversiteit, eveneens te Groningen. Tijdens zijn studie was hij gedurende 1 jaar student-assistent bij de werkgroep Multiple Sclerosis van de neurologische kliniek van het Academisch Ziekenhuis Groningen (Prof. dr. J.J.M. Minderhoud). Het artsexamen werd behaald in mei 1985, waarna hij in het kader van de militaire dienstplicht tot november 1986 als bataljonsarts was verbonden aan het 45e Pantser Infanterie Bataljon (Regiment Oranje Gelderland) te Steenwijk. In de daaropvolgende twee en een half jaar was hij werkzaam als arts-assistent-niet-in-opleiding, respectievelijk op de afdeling neurochirurgie van het Academisch Ziekenhuis bij de Vrije Universiteit te Amsterdam (Prof. dr. H.A.M. van Alphen) en op de afdeling neurologie van het Academisch Ziekenhuis te Groningen (Prof. dr. H.J.G.H. Oosterhuis). Van 1 oktober 1989 tot en met 30 september 1995 volgde hij de opleiding neurochirurgie aan het Academisch Ziekenhuis bij de Vrije Universiteit te Amsterdam (Prof. dr. H.A.M. van Alphen). In dat kader werden de stages neurologie en chirurgie vervuld, respectievelijk in het Academisch Ziekenhuis Groningen (Prof. dr. H.J.G.H. Oosterhuis) en het Medisch Centrum Alkmaar (Dr. P. de Ruiter). Tijdens en na zijn opleiding werd het onderzoek verricht dat te grondslag ligt aan dit proefschrift. Ten behoeve hiervan werd gastvrijheid genoten op de afdeling Anatomie en Embryologie van de Vrije Universiteit te Amsterdam (Prof. dr. H.J. Groenewegen). Sinds 1 oktober 1995 is de auteur neurochirurg in het Slotervaart Ziekenhuis te Amsterdam, in samenwerking met Dr. E.B. Bongartz, W.F. Luitjes en Dr. S.I. Tjahja. Hij is gehuwd met Marije Mol, logopedist en akoepedist. Samen hebben zij twee kinderen, Bas en Merel.

Publicatielijst.

1. Groen R.J.M. and H. Ponssen. The Spontaneous Spinal Epidural Hematoma. A study of the etiology. J Neurol Sci (1990) 98:121-138.
2. Groen R.J.M. and H. Ponssen. Vascular Anatomy of the Spinal Epidural Space: Considerations on the etiology of the Spontaneous Spinal Epidural Hematoma. Clin Anatomy (1990) 4:413-20.
3. Groen R.J.M., J.H. Begeer, J.T. Wilmink and R. le Coultre. Acute Cerebellar Ataxia in a Child with Transient Pontine Lesions Demonstrated by MRI. A case report. Neuropediatrics (1991) 22:225-7.
4. Groen R.J.M. and H.A.M. van Alphen. Venous Drainage in Hemorrhage from AVM's. Neurosurgical forum. J Neurosurg (1992) 77:822-3.
5. Groen R.J.M., O.G. Sie and T.W. van Weerden. Dominant inherited distal spinal muscular atrophy with atrophic and hypertrophic calves. J Neurol Sci (1993) 114:81-4.
6. Groen R.J.M. Spinal Arteriovenous Malformations. In: J.D. Palmer (ed.): Neurosurgery 93; A manual for European trainees in neurosurgery. Brighton, N. Thomson Ltd. (1993) I50-I55.
7. Groen R.J.M. en W.J.R. van Ouwerkerk. Cerebellair abces ten gevolge van een occipitale dermale sinus bij een zuigeling. Ned Tijds Geneesk (1994) 30:1575-7.
8. Groen R.J.M. and W.J.R van Ouwerkerk. Cerebellar dermoid tumor and occipital meningocele in a monozygotic twin: clues to the embryogenesis of craniospinal dysraphism. Child Nerv Syst (1995) 11:414-7.
9. Groen R.J.M. Spinal Arteriovenous Malformations. In: J.D. Palmer (ed.): Neurosurgery 96; A manual for European trainees in neurosurgery. London, Churchill Livingstone Ltd. (1995) 674-9.
10. Groen R.J.M. and H.A.M. van Alphen. Operative treatment of spontaneous spinal epidural hematomas: a study of the factors determining postoperative outcome. Neurosurgery (1996) 39:494-509.
11. Groen R.J.M., S.I. Tjahja, W.F. Luitjes, E.B. Bongartz and D.A. Bathelor. Klinisch denken en beslissen in de praktijk. Een vrijbuiter met een dreigende dwarslaesie. Ned Tijdschr. Geneeskd (1997) 141(13):652. Ingezonden brief.
12. **Groen R.J.M.**, M. van der Kuip, A. Jonker and P.V.J.M. Hoogland. The human radicular veins. Alternative anti-reflux-mechanisms in the absence of valves. Clin Neurol Neurosurg (1997) 99:S178. Abstract.
13. **Groen R.J.M.**, P.V.J.M. Hoogland, H.J. Groenewegen and H.A.M. van Alphen. The morphology of the human internal vertebral venous plexus. A cadaver study after intravenous Araldite CY 221 injection. Clin Neurol Neurosurg (1997) 99:S178. Abstract.
14. Groen R.J.M., H.J. Groenewegen, H.A.M. van Alphen and P.V.J.M. Hoogland. The morphology of the human internal vertebral venous plexus. A cadaver study after intravenous Araldite CY 221 injection. Anat Rec (1997) 249:285-94.