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Evaluation of Private Long-Term Care Facilities in Kaohsiung

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簡易輪椅改裝作步行訓練 — 病例經驗

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步行訓練是每個脊髓受傷病人所希望能達到的目標。但是並非所有脊髓受傷病人都能步行，部份運動功能障礙較嚴重者仍以輪椅活動爲主。因此，輪椅是殘障病患常用及常備的位移輔助用具，經過簡單的改裝亦可兼作步行訓練器。則既可增加輪椅的功用，亦可節省購買另一種步行輔助器的費用。

本研究將一輛標準型輪椅，經過簡單的改裝，在椅背推手處加裝前臂手杖並附有輪子，在後輪加裝煞車器，即可變成步行訓練器。並試用於一位第五節頸髓不完全傷害，導致四肢部份痲痺的32歲男性病人，發現比市面出售的帶輪步行輔助器walkerette在步行訓練時穩定度更佳。經常規復健訓練，及用改裝輪椅作步行訓練，病人在數星期後，可改爲使用walkerette步行。此改裝簡易而實用，值得推廣。

關鍵詞：輪椅改裝，步行訓練

前　言

隨著台灣工商業日漸發達，交通及工業意外日增，因而導致脊髓損傷的病患相當常見。他們大部份年紀較輕，在復健過程中，對能否步行往往很在意。但對高位受傷如頸椎的病人而言，部份病人雖有神經恢復，但康復期往往較長卻於出院後仍以輪椅代步者爲數不少。若能把輪椅稍作改裝即可兼作步行訓練器，則對病人的訓練及精神適應有幫助。故本研究在針對此問題進行輪椅改裝的可能性及實用性作進一步探討。

病例報告

病人爲32歲，男性過去健康情形良好。於民國78年10月7日在工作時，因高空落物擊中後腦部，曾有昏迷四至五小時，並四肢無力。病人先到台中光田醫院入院檢查，經電腦斷層檢查發現頸椎第七節棘突骨折及腦部基底骨折。病人於民國78年10月17日轉至本院，經保守療法後，於78年10月21日轉到復健科。當時他爲四肢痲痺，且大小便無法自解，薦部有5×6cm的褥瘡。其後他四肢運動功能漸有進步，但十分緩慢。故大小便控制訓練完成，及可乘坐輪椅活動後即出院回家，此時爲受傷後的3個月。其後病人曾進出長庚紀念醫院3次，主要是因爲壓瘡，尿道感染，並接受常規復健訓練，他的運動功能緩慢但持續進步，在輪椅移位及墊上運動都執行不錯。

他在受傷後10個月，接受步行訓練，當時他的Frankel scale爲class c，他的感覺在C5以下部份障礙，且有感覺異常。他的上肢肌力如下：

	三角肌	二頭肌	三頭肌	腕屈肌	腕伸肌	手握肌
右	N	G	F	P	P	P
左	N	G	F	F	F	F

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下肢肌力：

髖屈曲 髖外展 膝屈曲 膝伸展 踝背曲 踝庶曲

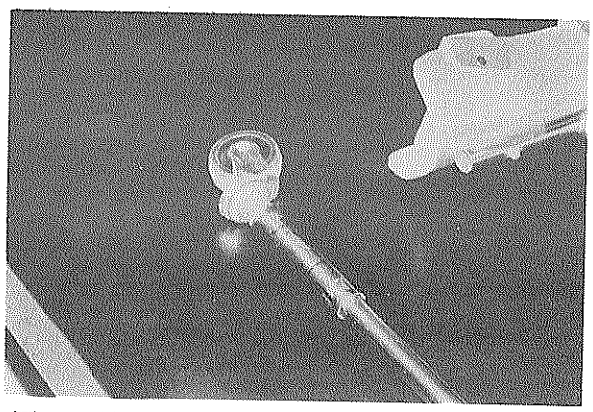
右 F F G G P F

左 G F G G F G

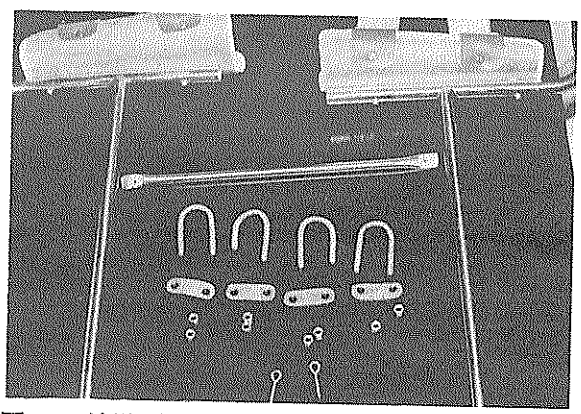
且四肢均有痙攣性，由於他上肢三頭肌肌力及肩部外展力量不夠，不足使用腋下拐杖及助行器作步行訓練，但由於病人有很強的學習動機使用帶輪步行輔助器 walkerette，則顯示穩定度不夠，故醫療人員採用標準輪椅改裝爲步行訓練器。

本研究將一輛標準型輪椅，在椅背推手處加裝前臂手杖並附有輪子(如圖一)。中間有一鐵條固定(如圖二)，前臂手杖高度爲肘關節屈曲 90 度下一英吋高度，並在後輪加裝自行車煞車器(如圖三，圖四)，即可變成步行訓練器(如圖五)。

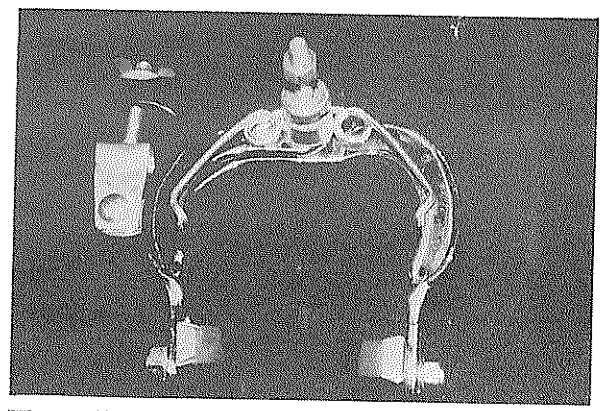
經此改裝輪椅作步行訓練，病人能向前，向旁邊移位(如圖六)，經數星期後，現可改爲 walkerette 行走。



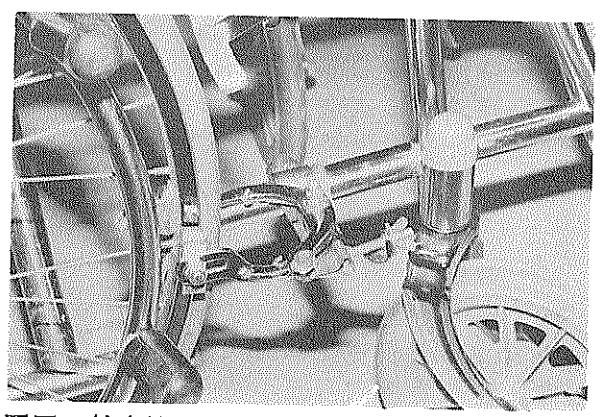
圖一：前臂手杖並附有輪子



圖二：前臂手杖及固定配件以及中間固定鐵條

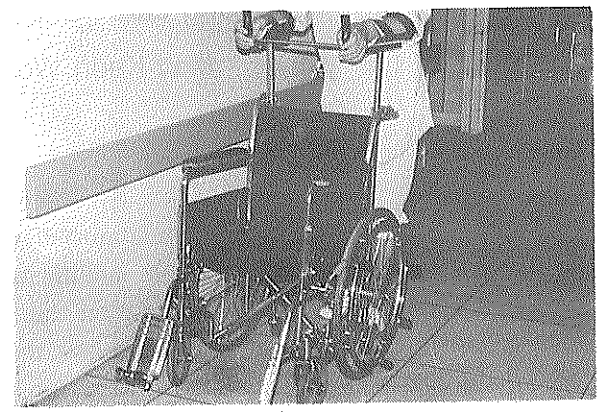


圖三：後輪自行車煞車器



圖四：煞車器固定於輪椅的情形

圖五：輪椅改裝後的情形



圖六：病人作爲步行訓練

討 論

脊髓受傷是一種嚴重的殘障，據1978-1981台北地區15家大醫院的統計，每年台北市每百萬人口約14.6人，換算成台灣地區人口，每年約有300名新病例。傷者以年齡在20-29歲發生率最高佔31.8% ，男女之比爲6.5：1[1]。可見這些脊髓受傷常發生於青壯年，對病人本身，家庭及社會均造成很多問題。而台灣近20年的趨向，頸髓受傷的比例百分率愈來愈高，在1987年已接近一半。對不完全脊髓受傷的病人，他們大部份在復健過程中最關切的是否能步行[2]。雖然有研究報告下肢痲痺的病人，在使用拐杖與長腿支架，其增加氧氣吸收量爲正常人的6倍，但行 走速度則祇有正常人的一半[3]。而本病人爲年青的病人，而無心肺方面的問題，且有强的動機；但因肌力不足及部份痙攣性而未能使用一般拐杖作行走訓練。若因此而放棄行走訓練，對病人是很大的打擊。

本改裝用前臂手杖在於病人其三頭肌肌力不足，故用前臂手杖固定於扶手地方，而加裝自行車用的煞車器是防止輪椅向前滑。

由於據Hong研究，有高達78%病人曾使用腿支架作步行訓練，但出院後以輪椅爲主[4]。可見大部份病人仍以輪椅爲主，若能將輪椅經簡單改裝，變步行訓練器，對病人而言，所費不高而且相當實用。可以訓練至較好程度，且病人行走意願很高，才花錢添置其他支架及輔助器，不失爲可行之道。且本改裝簡單而實用，故此值得推廣與進一步研究改良。

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Wheelchair Modification for Ambulation Training
--- Case Experiance

Yiu-Chung Lau Chau-Peng Leong Pong-Yuen Wong
May-Kuen Wong and Liang-Chieng Wu

Wheelchair is a common ambulatatory device for handicapped person. After simple modification, it could be used as a kind of ambulation training aid. It the study, a standard wheelchair was modified by adding a pair of axillary crutches on the back and brakers on the rear wheel. It was used by a 32 years old male patient with C5 incomplete spinal cord injury in Frankel class C, as ambulation training device 10 month after injury. After a period of home program for ambulation training, he could walk with walkeratter.

This modificatory wheelchair as a case experience is very simple in design, and very convienient for application, suitable for those patients who do not have enough muscle power for ambulation training with ordinary axillary crutches.

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Spinal Epidural Hemorrhage
Three Cases Report and Literature Review

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Spinal epidure hemorrhage is rare. It is often misdiagnosed and mistreated, and may result in severe paraplegia or death. About half of the cases reported in the literature had identifiable causes. Among these, trauma and anticoagulant therapy were most frequently encountered. The others were known as spontaneous spinal epidural hemorrhage. We report here 3 cases treated in NTUH. Their symptoms included sudden onset of severe localized back pain and flaccid paralysis of lower extremities which progressed within minutes to days. In case 1, early spinal decompression minimized the possible neurological sequelae. In case 2, hematoma occurred in the lower thoracic spine. Pain in the early stage was not differenciated carefully from acute abdomen. Diagnosis was made in case 3 when flaccid paralysis developed completely. Unlike that occurs in the skull, epidural hemorrhage in the spine is always a surgical emergency. It is important to differentiate from other causes of spinal cord injury, and to make early diagnosis and management.

Key words: spinal, epidural hemorrhage

INTRODUCTION

Spinal epidural hematoma is a rare disease. About one-half of the reported cases had identifiable causes such as trauma or anticoagulant therapy. The others were classified as spontaneous epidural hemorrhage[1,2]. The disease might progress within minutes to days. Chronic courses had also been reported[3]. Sudden onset of severe localized back pain might occur and lead to flaccid paralysis. Plan computerized tomography (CT), myelography, CT-myelography and magnetic resonance imaging with or without contrast[4] sharpen the diagnosis. The prognosis depends on the progression of the disease, the preoperative neurological deficits and the timing of operation. Prognosis usually is fatal if operation is not undertaken immediately[5]. Since they were often misdiagnosed and mistreated so as to affect the prognosis of the patient, we would like to present three cases varified by operation at National Taiwan University Hospital (NTUH).

CASE REPORT

Case 1: A 19 year-old male patient had a blow by a hammer over T4 spine level in Sep. '86. There was no obvious neurologic deficit initially. He suffered from sudden onset of severe pain over the previous hitted area in the morning on Mar.5,'87. He described the pain as an electric burn-like sensation. Numbness arised from the toes up to the thighs within 5 minutes and to the umbilical area within 10 minutes with concomittent paraplegia. The numbness progressed to T3 level and urine retention was noticed in the after-

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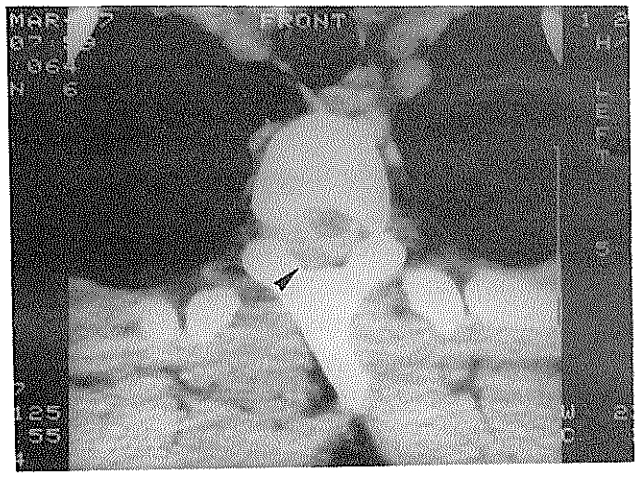
noon of the next day. He visited a local hospital and was transfered to NTUH at 6:00Pm on Mar.7. Neurological examination showed a complete paralysis of bilateral lower limbs. The tendon reflex was no response over left lower limb and markedly decreased over right lower limb. There was severe hypesthesia below T3 level. Emergent myelography showed a complete block at T3-T4 level. CT myelography revealed a biconvex mass at right dorsolateral canal of T2 level(Fig.1). Under the impression of spinal epidural hemorrhage, immediate T1-T2 laminectomy with evacuation of 4c.c. blood clot was done about 40 hours after the onset of the symptom. Reviewing his past history showed smoking more than one pack per day for 5 years and social drinking for about 3 years. There were no obvious laboratory abnormalities. The bleeding time was one minute and thirty seconds. After operation, hypesthesia subsided immediately. He was transfered to us for rehabilitation and follow up. Muscle power of the affected limbs improved to nearly normal 9 months after surgery. The residual symptoms were mildly increased deep tendon reflexes and mild urgency of urination.

Case 2: A 27 year-old male patient had rheumatic heart disease with mitral stenosis(MS), mitral regurgitation(MR), atrial regurgitation(AR) and tricuspid regurgitation(TR) diagnosed in Jan.'85. He had taken sustained coumadin 2.5mg per day since Mar.7,'85 after mitral valve replacement and bicuspidization for tricuspid regurgitation. Sudden onset of back pain radiating to anterior loewr abdomen developed on Apr. 27, '89. He visited a local hospital where laparotomy was done under the impression of acute abdomen. There was no positive finding during the operation. The pain improved thereafter. In the afternoon of May 1, '89, sudden onset of paraplegia developed. He visited the local hospital again. Plain CT did not show any obvious finding. He was sent to NTUH immediately . The neurological examination showed a complete flaccid paralysis with anesthesia below T11 dermatome. Myelography showed an extradural compression and complete block at T10-T11 level. T10-L1 laminectomy with evacuation of 10c.c. blood clot was done on May. 2. Laboratory finding showed prolonged bleeding time, prothrombin time, and partial thromboplastin time (5 minutes, 35.2/11.5sec, and 66.3/38.5sec respectively). Cardiac catheterization revealed the presence of MR, TR, AR and pulmonary hypertension, and atrial fibrillation was identified by EKG. Mitral valve and aortic valve replacements were done on Aug.7,'89. The post operative improvement of the neurological deficits was minimal. He could ambulate with the aid of bilateral long leg braces and bilateral axillary crutches or a walker for about 50 meters, so he was discharged on Oct.17,'89. Urination may be induced by anal stretch. Coumadin 2.5mg per day was given continuously.

Case 3: A 64 year-old housewife had hypertension for more than 5 years without regular treatment. Sudden onset of severe back pain with radiation to bilateral lower limbs followed by an acute paraplegia occurred at about 8:30Pm on Dec.7,'89. She was sent to a local hospital on Dec.8, where her blood pressure was 290/150 mmHg. The neurologic status were flaccid paralysis of lower limbs and anesthesia below L1 dermatome. Since there was no obvious finding on plan X-ray, myelography was done on Dec .9,'89 and a complete block at T12 level was found (Fig.2). CT myelogram showed a hyperdense biconvex lesion at the right posterolateral part of the spinal canal (Fig.3). Only conservative treatment was given until she was sent to NTUH on Dec.13,'89. The neurological examination showed a poor to fair muscle power in bilateral lower limbs. Tendon reflexes in lower limbs could not be detected. Babinski sign was positive bilaterally. Under the impression of epidural hematoma, T10- T12 laminectomy with evacuation of 2c.c. blood clot was done on Dec.14. The laboratory data did not show any coagulation deficit. Repeated test of creatinine clearance was around 21 ml/min. KUB showed an uneven kidney



LEGENDS FOR ILLUSTRATIONS

Fig.1 : CT myelogram revealed a biconvex mass (arrow) in right dorsolateral canal of T2 level in Case 1.

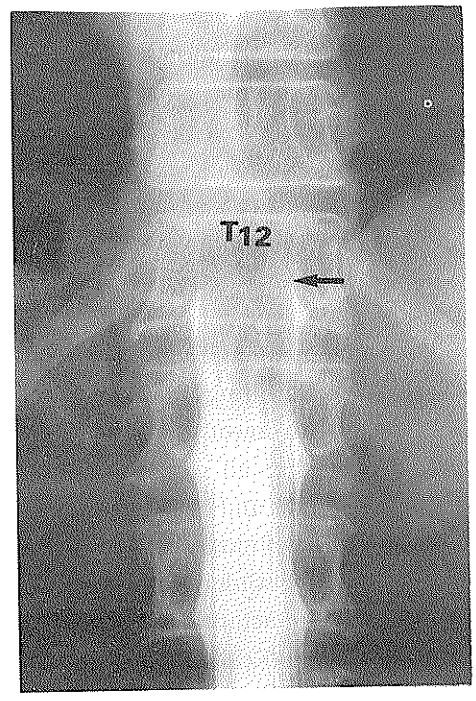


Fig.3 : CT myelogram showed a hyperdense biconvex lesion (arrow) at right posterolateral part of the spinal canal in case 3.

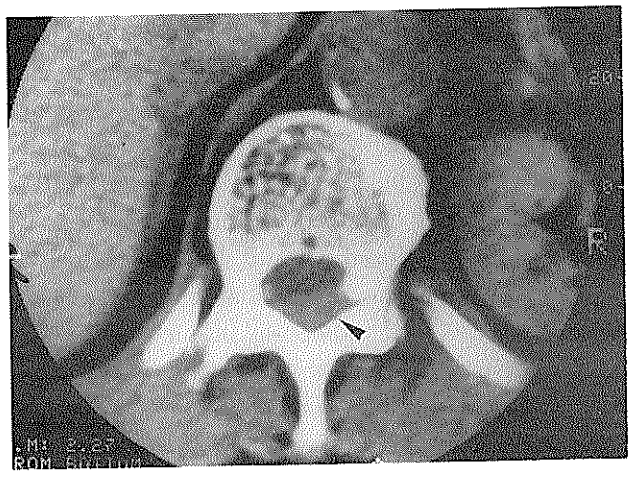


Fig.2 : Myelogram showed a complete block (arrow) at T12 level in case 3.

size. Hypertension caused by abnormal renal artery was suspected, but the patient refused further study. After operation, pin-prick sensation improved below T10 level including lower extremities. The muscle power was the same as preoperative condition. she was on wheel chair and discharged on Jan.24,'89.

DISCUSSION

Spinal epidural hematoma occurred about twice as commonly in male as in female. All age groups may be affected. The peak occurrences were after the 4th decade in male and 5th decade in female[6]. The attack is uaually acute, but chronic intermittent relapsing courses due to small, repeated hemorrhage and dissection[7] and chronic course presenting as spinal stenosis[3] were also reported. The most commonly affected areas were thoracic spine, extending downward to upper lumbar and upward to lower cervical spine[6].

About one-half of all cases had identifiable causes. Spinal trauma and anticoagulant medication[8] were among the most frequent causes, followed by hemostatic disorders, vascular malformation [9,10], lumbar puncture[11], and neoplasms. The other cases were so called spontaneous spinal epidural hemorrhage. The identifiable precipitating factors include trivial trauma[12], systemic hypertension, arthritis, old age, antiplatelet aggregation drugs[1], minor prolongation of bleeding time, pregnancy, etc.

The spinal epidural space, unlike that in the skull, is composed of loose areolar tissue. The blood supply of the meninges come with the nerve roots. Just inside the spinal canal, they leave the nerve and bifurcate to form a longitudinal channel in the epidural space. From this channel and midway between the adjacent nerve roots, free bridging vessels pass dorsally and medially to the side of the dural sac and then run transversely toward the midline where they anastomose with their counterpart from the opposite side. The veins from the anterior and posterior internal venous plexus form an arcuate pattern overlying the arteries[13]. These veins communicate freely with the intra- abdominal, retroperitoneal, and intrathoracic venous structures.

There are two main theories for the source of bleeding, ie, venous origin or arterial origin. Those who favored venous origin[2,7,14,15,16] observed factors as: 1. Spinal epidural veins are situated in a larger space that separates them from the adjacent bone and are less well protected than those of cranium, 2. They are vulnerable to trauma during lumbar puncture because of their thin walls and large size, 3. Since there are no valves within the epidural venous system, the epidural veins are not protected against changes of pressure in the neighboring venous structures such as intraabdominal, retroperitoneal or intrathoracic veins. Those who favored arterial origin[2,13,14] stated:1.The frequent lateral location, 2.The rapidity of onset and progression of symptoms, 3. The frequent association of hypertension, 4. Surgical confirmation of arterial bleeding. Bleeding from occult arteriovenous malformation or other microscopic vascular malformation[2,14] had also been reported. It seems that either origin is possible but they would manifest with different speed of progression and severity of neurological deficit.

The clinical presentation usually is sudden onset of severe localized back pain lasting some minutes, followed by severe pain of radicular quality, in more than half the cases. This symptom is easily misleading and should be differentiated carefully from acute myocardial infarction, pleurodynia[8] or acute abdomen as in our case 2. The back pain may subside a few hours after onset but sensory deficits will take place instead . This phenomenon should not be regarded as a true improvement. Thereafter, flaccid paralysis and impaired sphincter control will occur. The course may develop in minutes, hours,days or months[3,13, 14,17].

The differential diagnoses of clinical features include: acute intervertebral disc prolapse, acute

demyelinating disease, cord ischemia, infarction, intramedullary hemorrhage, acute epidural abscess, spinal neoplasia, dissecting aortic aneurysm, congenital cysts, spondylitis[13] and all chest and abdominal diseases[8].

The epidural hematoma is best evaluated noninvasively by a plane high resolution CT with 5mm thick (or less) section and sagittal reconstruction[15]. The CT appearance of hematoma depends on the duration of existance[18]. The density will be high in acute stage and relatively isodense in subacute stage. A biconvex-shaped lesion lying adjacent to the vertebral body or, more frequently, to the posterior arch separated from the less dense spinal cord and subarachnoid space[15,19] sharpen the diagnosis. In cases with less clearcut clinical features or plan CT findings, myelography with CT may be performed[16]. The findings include partial or complete extradural blocks and, less commonly, nonobstructing extradural defects which extend 2 or more segments. The spinal fluid examination may only show elevated protein content, typical for spinal cord compression[8]. Ultrasound has been used for the diagnosis of neonatal spinal problem before 6 month-old [20]. Recent report[15] of MR imaging suggests that using short and long repetition time (TR) sequences and multiple imaging planes will provide a rapid, noninvasive and accurate diagnosis. Gadolinium-DTPA contrast may be added to show the prominant enhancement of thickened meninges [5]. Operation or autopsy will verify the diagnosis.

In an animal study[5], the prognosis of spinal cord compression was related to the rapidity with which paralysis developed, the force of compression, and the duration of paralysis prior to decompression. In human studies, the prognosis also depends on 3 factors. The first is the length of time elapsing between the appearance of the first clinical symptom and the onset of paraplegia. The faster the neurologic symptoms develop, the worse the prognosis will be. The second is the length of time elapsing betweem onset of paraplegia and surgical decompression. The third is the severity of the preoperative symptoms[8,12,17,21]. Several reports pointed out that one-half of those patients operated on within 24 hours after the onset of the hematoma will recover completely from the paralysis[19]. Good result can be expected in young, if the preoperative deficit is not yet complete or when some transient improvement is observed[17]. Death maybe closely related to the failure of regaining motor function in patients who do not recover from cord compression[5].

The causes of "complicated" deaths include necrotic edematous spinal cord infarction and central myelomalacia.

Spinal epidural hematoma should be treated as a surgical emergency despite a few spontaneous resolution cases have been reported[1,2,9]. The operative results are sometimes disapppointing even after prompt diagnosis and treatment[8]. In the hospital with available facilities, immediate decompressive laminectomy with evacuation of the blood clot should be done. For hospital without emergent neurosurgical facilities, percutaneous aspiration using the standard discography approach[21] may be introduced as quickly as possible to relieve intraspinal pressure. It is an encouraging method to be tried for neurological and functional salvage, and may also be used as a method to differentiate posttraumatic multiple disc herniation.

In conclusion, spinal epidural hemorrhage is a rare emergency with good prognosis if diagnosis and treatment can be made promptly after the occurrence of the first neurologic symptom. In our 3 cases, the only one with fair result had a 40 hours delay before surgery. The other two cases, either misdiagnosed as an acute abdomen or treated too later without much attention to the disease, had severe and permanent sequelae. The absence of sensorimotor functions before operation does not necessarily indicate a poor prognosis[12]. So, even a patient with complete sensory-motor lesion

should be operated on. It is not only for surgens to be familliar to the curable castrophe and always keep in mind, but also for physicians to follow the rehabilitation guide.

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