

Dissociation of neurological deficits in spinal decompression illness.

S. TOGAWA^{1,2}, M. MARUYAMA¹, N. YAMAMI², H. NAKAYAMA², M. SHIBAYAMA³, M. KAWASHIMA⁴, K. SHIMADA⁵, Y. MANO²

¹*Department of Physiology, Faculty of Medicine, University of Miyazaki, Japan;* ²*Department of Hyperbaric Medicine, Tokyo Medical and Dental University Hospital, Tokyo, Japan;* ³*Department of Humanities, Komazawa Women's University, Tokyo, Japan;* ⁴*Kawashima Orthopedic Surgery Hospital, Nakatsu, Japan;* ⁵*Japan Aerospace Exploration Agency, Tsukuba, Japan*

Submitted 4/12/2005; final copy accepted 12/14/2005

Togawa S, Maruyama M, Yamami N, Nakayama H, Shibayama M, Kawashima M, Shimada K, Mano Y. Dissociation of neurological deficits in spinal decompression illness. *Undersea Hyperb Med* 2006; 33(4):265-270. Functional differentiation is found in the spinal cord. A unique set of neurological deficits follows a multi-focal injury. Clinically, sensory and motor disturbance present independently, often resulting in sensory and motor deficit dissociation. This study examined 103 spinal decompression illness (DCI) cases. The neurological deficit dissociation was classified as follows: 1) Cases with sensory impairment only, or motor dysfunction alone, were tagged as having “dissociation” (44 cases); when a case was with both sensory and motor dysfunction, the spinal level of the sensory impairment was determined and was matched with the spinal segments responsible for the motor dysfunction; 2) If the two spinal areas did not match (i.e. with no regional overlap), they were tagged as having “dissociation” for each motor dysfunction (32 cases). In total, dissociation was present in 76 out of 103 cases. We concluded that clinical neurological deficit dissociation is frequently observed in spinal DCI.

INTRODUCTION

Imaging evidence of spinal decompression illness (DCI) is rarely reported. Reuter (1) reported that spinal lesion had been detected by MRI in only one DCI case out of 7 definitive spinal DCI patients. We found 8 spinal DCI reports by Medline, that claim detection of spinal DCI lesions by MRI. Responsible lesions were identified by MRI, in 6 cases with paraparesis, in one case with hemiparesis, and in one fatal case. No MRI lesion was identified in mild DCI cases. We had a chance to conduct 22 MRI sessions on spinal DCI cases that were resistant to oxygen recompression therapy, and found only one MRI signature that was interpreted as a DCI focus. This may suggest that mild DCI lesions are too small to be imaged by MRI. Because motor and sensory neurons follow separate

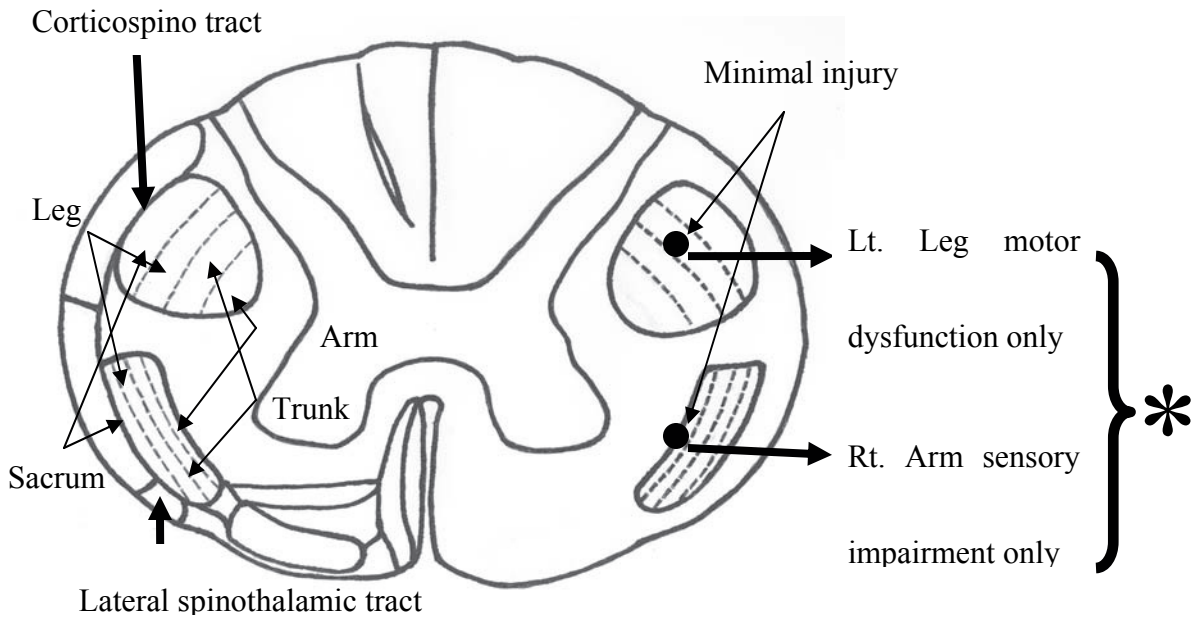
paths in the spine (2, 3, 4), multiple but small foci result in independent motor and sensory deficit, namely, neurological dissociation. For example, an injury to the left lateral path of the corticospinal tract causes motor dysfunction in the left lower limb, whereas an injury to the posteromedial path of the left spinothalamic tract causes impairment of temperature sense of the right upper limb. This is followed by sensory impairment and motor dysfunction in different areas (Fig. 1).

This study tested the hypothesis that there is dissociation between sensory and motor deficit in spinal DCI.

METHODS

We conducted a prospective study. We examined 583 patients who had clinical symptoms after diving at Tokyo Medical and

Fig. 1 Cross-sectional diagram of spinal cord showing details related to dissociation
 * Neurological dissociation between sensory and motor dysfunction



Dental University Hospital from January 2002 to February 2004. Of the 583 cases, 244 patients were initially seen by the physician specializing in neurology and diving medicine. We excluded 42 patients who had symptoms such as nausea, dizziness or abnormality in facial sense that indicate cerebral involvement; 15 patients who had Tinel sign indicating peripheral nerve involvement; 82 patients who were found to have no sensory or motor dysfunction; and 12 patients who had already been treated at other hospitals. Therefore, the number of subjects included was 103. Note that some patients had multiple reasons for exclusion. There were 62 male and 41 female patients, with mean age of 35.4 years, ranging 18 to 50.

The diagnosis of DCI was made by 1) presence of distinctive neurological deficits; and 2) alleviation of symptoms with hyperbaric oxygen therapy. Informed consent was obtained before treatment, at the initial outpatient visit. The standard form and procedure of the Tokyo

Medical and Dental University Hospital was applied.

Neurological observations were recorded at the first visit to the hospital before treatment in all cases. Dermal thermal sense was examined by applying a tube filled with cold water at about 5°C to the skin every 5 cm, starting from the proximal point to the distal. Right-left difference was stated by the patient. Then, the tube was slid on the skin from the proximal to the distal, and the patient was again asked to state the variation in the coldness. When we found a probable hypesthesia/ hyperesthesia area, we tried the area starting from normal periphery inbound to the suspected area. Then, to divert the patient's excess attention to one area, we reversed it to distal to proximal direction, or randomly touched the skin. Using a dressmaker's pin (multiple pins on a wheel), dermal pain sense was examined. This pinprick test process was performed in the same way as the thermal sense test. After localizing a

sensory impairment area, we performed the test proceeding from the hypesthetic to the area.

Final determination of sensory impairment due to DCI was made when it was alleviated by hyperbaric oxygen therapy. When the dermal distribution of hypesthesia for temperature and pain did not match, both temperature and pain deficit area was recorded as having hypesthesia. The spinal level of sensory impairment was assigned according to the dermatome of J. Walton. Two spinal levels were included when impairment affected two adjacent dermatome segments (5).

Muscle strength was tested manually on the muscles controlling seven movements of the upper limbs (Scapula Elevation, Shoulder Joint Abduction, Elbow Joint Flexion, Elbow Joint Extension, Wrist Joint Flexion, Wrist Joint Extension, and Index Finger Abduction) and five movements of the lower limbs (Hip Joint Flexion, Knee Joint Extension, Ankle Joint Dorsiflexion, Great Toe Extension, and Foot Eversion).

Motor dysfunction was identified to be caused by DCI when muscle strength was grade 4 (subnormal) or lower on the manual muscle test, and improvement was clearly observed (by one or more grade) after hyperbaric oxygen therapy.

A DCI case was classified to have neurological 'dissociation' when dermal hypesthesia was observed without motor dysfunction, or motor dysfunction was present without hypesthesia.

The spinal segments responsible for motor and sensory dermatomes were investigated when both motor and sensory deficit was present. The muscles involved in each joint movement and the spinal segments controlling them, as described by Hislop et al. (6), were reviewed. All spinal segments innervating the muscles involved in a motor function were identified as injured segments. For example, when a patient had a motor weakness in index finger abduction, it is possible that the dorsal interosseous muscle

(responsible spinal segments are C8 and Th1) or extensor digitorum muscles (C6, C7, and C8) are involved. Then we would assume that the involved spinal segments were C6, C7, C8 and Th1. During neurological examination on a particular part of the body, 'dissociation' was recorded when the motor deficit focus in the spine was totally separate (no overlap) from that of thermal/pain deficit focus. Motor deficit focus was defined by the spinal segments innervating the weakened muscles. Sensory deficit focus means the spinal levels that correspond with dermal hypesthesia. If there was any overlap in the two spinal foci, it was classified as NOT dissociated. We classified a patient as a case with 'dissociation' when there was at least one dermal site of neurological dissociation.

Statistics

The neurological dissociation patient group was compared to the non-dissociation group in following four aspects using the Mann-Whitney's U tests: 1) mean water depth of the last dive; 2) mean latency time from a dive to symptom onset; 3) progression rate of disorder prior to the initial treatment; and 4) complete relief rate by recompression. Since the latency time depended on airline flight time after the last diving, patients were classified in either the flight group or the ground transportation group.

RESULTS

Neurological dissociation was observed in 44 cases, which includes 33 cases of hypesthesia without motor dysfunction and 11 cases of motor dysfunction without hypesthesia. For the 59 cases with both hypesthesia and motor dysfunction, the spinal segments responsible for hypesthesia and motor dysfunction were matched. The number of cases without dissociation was 27, and with dissociation 32. Overall, we had 76 cases with neurological dissociation out of 103 (74%).

In the 59 cases with both sensory impairment and motor dysfunction, the areas of sensory impairment and motor dysfunction were examined in five districts, including the four limbs and trunk. Sensory impairment and motor dysfunction were observed in the same district in 37 cases, and motor dysfunction was also present in the area of no sensory impairment in 22 cases, showing the presence of motor dysfunction without sensory impairment in 47% of cases.

Without the airline flight group, the

mean latency time from dive to symptom onset was statistically different between the patient subgroup with dissociation and that without dissociation ($p < 0.01$). (Table 1)

DISCUSSION

There are several reports of pathogenic causes of spinal DCI. Neuman and Bove in 1990 (7) suggested bubble embolism. Hallenbeck in 1975 (8) associated the syndrome with ischemia due to congestion by intravenous

Table 1. Comparison of Max depth, Latency time, Progression rate of disease, Complete relief rate between dissociate and non-dissociate group

	Depth (m)	Latency with flight (h)	Latency without flight (h)	Progression (%)	Relief (%)
Dissociation	25.5 ± 7.5 (n=65) R; 11 to 40, M; 23	27.0 ± 20.0 (n=29) R; 2 to 72, M; 20	10.0 ± 21.6 (n=35) * R; 0.1 to 120, M; 2	50.4	52.3
Non-Dissociation	24.7 ± 5.2 (n=26) R; 15 to 34, M; 25	33.9 ± 32.9 (n=14) R; 5 to 120, M; 20	5.2 ± 5.5 (n=13) * R; 0.1 to 18, M; 4	58.3	49.3

Depth=The mean depth of the last dive; Latency=The mean of latency time; Progression=Progression rate of disorder prior to treatment; Relief=Complete relief rate by recompression, R=range; M=median

Symptom onset before last dive n=12

*Significant difference were noted between dissociate and non-dissociate group < 0.01 .

bubbles. Francis in 1989 (9) discussed the role of mechanical pressure from bubbles in the spinal parenchyma. He presented the canine experimental DCI spinal cord sections. These pathohistological photomicrographs showed many extravascular non-staining, space-occupying lesions (suggested as marks of bubbles) in the spinal cord. In this study, we proved our hypothesis neurologically that the spinal in DCI has multiple injuries. Francis's figures also show multiple small lesions, thus supporting our hypothesis. These small lesions could be induced not only directly by autochthonous bubble but also by arterial embolism of peripheral blood vessels distal to penetrating branches in the spine, since these end arteries supply extremely small areas (10).

Neurological dissociation was observed in many DCI patients in this study. Dissociation of sensory impairment is not uncommon in other neurological disorders found in clinical settings. Because of subjective factors in sensory tests on patients, the areas of hypesthesia are not identified objectively. Therefore, there is an inherent clinical limitation to the degree that dissociation of sensory impairment and motor dysfunction is characteristic of spinal DCI. Knowing this, we designed a prospective study in which the physical examination was performed following a procedure constructed to exclude patients' subjective influence as much as possible. We concluded from this prospective study that DCI is characterized by frequent occurrence of neurological dissociation. This study, however, does not imply that DCI be diagnosed by the presence of neurological dissociation.

Spinal anatomy suggests that if there are many extremely small lesions in the spine, neurological dissociation should be observed frequently, as shown in Fig. 1. Our clinical findings match this anatomical prediction. One might argue that neurological dissociation would be present if embolism occurred in each of the following vessels: anterior spinal artery, posterior spinal artery, and the central and

medullar vein group that compose spinal veins. Because embolism of these vessels injure the corticospinal tract and a spinothalamic tract separately, neurological dissociation follows. For example, we suspect that a case presented by Manabe (11) was caused by injuries in the posterior medullar vein group because sensory impairment was located in upper extremities bilaterally, and motor dysfunction presented as quadriplegia. Thus, neurological dissociation was present. However, since these embolisms occur in major vessels, they are easily visible in an MRI image. As mentioned earlier, spinal DCI lesions have not been detected often by imaging, therefore these embolisms may not play an important role in spinal DCI.

Since Tokyo Medical and Dental University Hospital is located relatively far from recreational diving sites, we rarely conduct emergency recompression therapy. Our mean latency time was longer than that reported by DAN (12). This latency makes it difficult to diagnose DCI. For us, the characteristics associated with frequent neurological dissociation are potential differential diagnosis aspects.

□ For the present paper, in order to focus on spinal DCI, we excluded DCI with brain or peripheral nerve lesion based on physical examinations and symptoms, thus the exclusion may or may not have been complete and some cerebral DCI patients may have remained among those classified as spinal type. For instance, a brain DCI patient included as a spinal DCI case, might show neurological dissociation, since the brain shows functional differentiation, like the spine. Principally there should be no obstacle to extend our study to include brain DCI.

CONCLUSION

Neurological dissociation of sensory impairment and motor dysfunction occurs frequently in patients with spinal DCI. The data supports the hypothesis that spinal DCI foci are multiple but often of small volume.

This knowledge is important in differential diagnosis and treatment of DCI.

REFERENCES

1. Reuter M. MR imaging of the central nervous system in diving-related spinal decompression sickness. *Acta Radiol.* 1997; Nov;38(6):940-944.
2. Patten JP. Neurological differential diagnosis, 2nd ed, Springer-Verlag, New York, 1996: 213-225.
3. An HS. Anatomy of the spine. In: An HS ed. Principles and techniques of spine surgery. Baltimore, MD: Lippincott Williams & Wilkins, 1998: 1-30.
4. Peter Duus. Neurologisch-topische Diagnostik (Neurological-topical diagnosis): Anatomie Physiologie Klinik (Anatomy Physiology Clinic), Georg Thieme Verlag Stuttgart, New York, 1987: 1-97.
5. John Walton ed. Brain's Diseases of the Nervous System. 10th ed. London: Oxford University Press, 1993:40-52.
6. Hislop HJ, Montgomery J. Muscle Testing: Techniques of manual examination. 6th ed. W. B. Saunders Company, 1995:401-410.
7. Neuman TS, Bove AA. Combined arterial gas embolism and decompression sickness following non-stop dives. *Undersea Biomed Res* 1990; 17:429-436
8. Hellenbeck JM, Bove AA, Elliott DH. Mechanisms underlying spinal cord damage in decompression sickness. *Neurology* 1975; 25(4):308-316.
9. Francis TJR. A current view of the pathogenesis of spinal cord decompression sickness in an historical perspective. In: Vann RD ed. The physiological basis of decompression. Bethesda, MD: Undersea Hyperbaric Medical Society, 1989:241-279.
10. Turnbull IM, Brieg A, Hassler O. Blood supply of cervical spinal cord in man. A microangiographic cadaver study. *J Neurosurg* 1966;24:951-965
11. Manabe Y, Sakai K, Kashihara K, Shohmori T. Presumed venous infarction in spinal decompression sickness. *AJNR Am J Neuroradiol* 1998 Sep;19(8):1578-1580.
12. Bennet PB. Report on Decompression illness, Diving Fatalities and Project Dive Exploration: 2003 ed (Based on 2001data). Divers Alert Network.2003:40-41