Reply

X.F. Yang^{a,b}, L. Wen^{a,b}, R.Y. Zhan^{a,b}

^aDepartment of Neurosurgery, First Affiliated Hospital, College of Medicine, and ^bInstitute of Brain Medicine, Zhejiang University, Hangzhou City, Zhejiang Province, PR China

We would like to thank Kilincer et al. for their interesting comments on our previously published paper [1]. Kilincer et al. analyzed the differences between patients with large hemispheric infarction and those with traumatic brain injury and explained why patients undergoing decompressive craniectomy (DC) for traumatic brain injury have a higher incidence of contralateral subdural effusion than those undergoing DC for hemispheric infarction.

We agree with the explanation of Kilincer et al. that the differences between these two populations lead to the distinct incidences of contralateral subdural effusion secondary to DC. As mentioned in the letter, patients who have head trauma are prone to develop subdural effusion. Moreover, we would like to emphasize three potential reasons for this extremely high incidence of contralateral subdural effusion: (a) the pressure gradient between the two hemispheres resulting from a rapid decrease in intracranial cerebral pressure as well as outward herniation after DC, (b) the probable existence of contralateral injury, which would cause a

torn arachnoid layer, and (c) the disturbance of CSF circulation that exists after head trauma could be aggravated after DC.

Currently we have a 35-year-old male patient who was involved in a traffic accident and had severe head trauma. DC was performed on the left, due to elevated intracranial cerebral pressure caused by subarachnoid hemorrhage in the right lateral cleft cistern. Six (fig. 1) and 11 days (fig. 2) later, we observed a small quantity of subdural effusion contralateral to the decompressive side. The over-cephalocele through cranial defect was also discovered, as shown in figure 3. One month later, the effusions had mass effect (fig. 3), which led to manifest midline shift. Consequently, drainage with a burr hole was performed. The patient is still on follow-up. We think that this case provides further evidence for the development of contralateral subdural effusion secondary to DC performed for severe traumatic brain injury.

Reference

- 1 Yang XF, Wen L, Li G, Zhan RY, Ma L, Liu WG: Contralateral subdural effusion secondary to decompressive craniectomy performed in patients with severe traumatic brain injury: incidence, clinical presentations, treatment and outcome. *Med Princ Pract* 2009;18:16–20.

L. Wen, MD

Department of Neurosurgery, First Affiliated Hospital
College of Medicine, Zhejiang University, No. 79 Qingchun Road
Hangzhou City 310003, Zhejiang Province (PR China)
Tel./Fax +86 571 8723 6803, E-Mail wenlneuron@126.com

Fig. 1. CT image performed on the sixth day after DC showing a small quantity of subdural effusion contralateral to the decompressive side (arrow).

Fig. 2. CT image performed on the eleventh day after DC.

Fig. 3. CT image performed 1 month after DC showing that the effusion had mass effect and led to midline shift.