

# The syndrome of the trephined

Decompressive craniectomy (DC) is an effective treatment in the management of post-traumatic intracranial hypertension.<sup>[1]</sup> It is also a recognized lifesaving intervention for the treatment of malignant middle cerebral artery infarct.<sup>[2]</sup> However, the DECRA (decompressive craniectomy in patients with severe traumatic brain injury) has shown that the DC is not a harmless procedure. If the indication threshold is sensibly reduced its associated risks can overcome its potential benefits.<sup>[3]</sup> In addition, the risks of the DC are not limited just to the surgery and the immediate post-surgical period. Deferred complications might appear either associated to the cranial opening or to the cranial repair (cranioplasty).<sup>[4]</sup>

Post-surgically, once the cerebral edema subsides and the risks of acute major complications have been left behind, craniectomized patients are frequently referred for rehabilitation. Many of these patients present severe disabilities, and sometimes the lack of improvement might erroneously be considered as sequelae of the primary brain injury. In 1939, Grant and Norcross<sup>[5]</sup> published a series of 83 patients, out of which 12 (14.5%) had undergone a surgery due to “syndrome of the trephine” (ST): Dizziness, undue fatigability, vague discomfort at the site of the defect, a feeling of apprehension and insecurity, mental depression and intolerance to vibration. In the 1970s, Yamaura and Makino coined the term “syndrome of the sinking skin flap” (SSSF) to describe the “objective” focal neurological deficits that can occur in the same population of patients. It has recently been proposed that “ST,” “SSSF,” and the “motor trephined syndrome” could be replaced by the more neutral term “neurological susceptibility to a skull defect.”<sup>[6]</sup> Ultimately, it is the neurological improvement once the cranial defect is repaired what in fact definitively confirms the diagnosis.<sup>[7]</sup>

The authors present a retrospective review of 29 craniectomized patients (due to various etiologies, i.e. trauma, infarct, infection, hemorrhage) out of which 7 (~25%) developed reversible neurologic symptoms or behavioral disturbance.<sup>[8]</sup> They establish a very interesting differentiation regarding the evolution of these patients: Five developed an *arrest of rehabilitation*, whereas the remaining two showed a *differed acute deficit*. The author’s series draws the attention to the significant incidence of symptoms reverted by cranioplasty. The reported radiological findings (ventricular effacement, midline shift, sunken scalp flap contour) as risk factors for ST are also interesting. As the authors established, their presence may be helpful for diagnosis of ST and also setting expectations with patients and families with regards to the cranioplasty. A point for future research is the lack of statistically significant association between ST and the cranial defect size. This seems at odds with the described radiographic findings that are intuitively expected in for large craniectomies rather than in smaller ones. Finally, ST should be considered in every craniectomized patient with arrest of rehabilitation or differed acute deficits because their symptoms are certainly reverted by cranioplasty.

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