

THE AUTHORS REPLY: We agree with Xydakis and colleagues that concussion (i.e., mild traumatic brain injury) can be accurately diagnosed only at the time of injury. We remain concerned that population screening for concussion and post-concussive symptoms months after injury will result in unintended iatrogenic effects from misdiagnosis, inconclusive neuropsychological or neuroimaging studies, costly referrals, or side effects of medication.^{1,2} The letter by Xydakis and colleagues, however, is also an example of the circular arguments and speculation about injurious primary-blast effects that permeate the recent literature about mild traumatic brain injury.³ The conclusion that “the diagnosis of mild traumatic brain injury can be made only over time” does not logically follow from the guidance regarding assessment within 24 hours after injury. Experimental blast models, such as air shock tubes, do not take into account vehicle and body armor, the high proportion of explosions that occur in open spaces, or the devastating effects of fragment dispersion and fire on persons close enough to be exposed to the primary overpressure wave.⁴

We agree with the comments by Das on the limitations of our study. However, our study focused on causal inferences at an important time when screening is becoming routine. If the suspected postconcussive symptoms 3 to 4 months after the return from deployment were caused by concussions, then we would expect this association both in soldiers with PTSD and in the larger group of soldiers without PTSD. This association was not observed in both groups; instead, the physical symptoms were clustered in the group with PTSD. The likely mechanism pertains to the very life-threatening context. PTSD is not known to be associated with sports-related concussions. Being knocked unconscious from a blast during combat is a life-threatening event that occurs in the context of other traumatic events. These experiences can precipitate stress responses and traumatic memory encoding that underlie the

development of acute stress disorder and PTSD, which, in turn, are strongly associated with generalized symptoms, including those in the “post-concussive” category, through autonomic nervous system and neuroendocrine dysregulation.⁵

We appreciate Stonesifer’s suggestion regarding pituitary dysfunction, but the literature to which he refers pertains almost exclusively to moderate or severe traumatic brain injury. We thank Ruff for detailing the multistep screening intervention in VA facilities. We only ask that Ruff consider the assumptions underlying each step and use scientific rigor to ensure that the expected risks and costs do not outweigh the hypothesized benefits. The value of screening would obviously be greater if effective treatments were available for concussions identified months after injury.

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Outcomes 18 Months after the First Human Partial Face Transplantation

TO THE EDITOR: The acceptance of a partial face allograft as a promising procedure for patients with facial disfigurement will depend on long-term functional results. An impressive recovery of sensory function was reported by Dubernard

et al. (Dec. 13 issue).¹ This recovery of sensory function is understandable, given that the maxillary and mandibular nerves were resutured in the recipient’s face. The recovery of motor function, however, remains a mystery, since only a single

branch of the facial nerve (described by Dubernard et al. as “the inferior branch of the left facial motor nerve”) was resutured. Such a nerve branch does not exist in the human facial nerve. Moreover, the authors do not explain why a circumscribed reanimation on the left side of the lower face would lead to recovery of motor function on both sides of the midface as well as the right side of the lower face. The video does not provide a convincing explanation. Perhaps what we see in the video is just passive movement in the transplanted tissue due to the sutures holding the graft muscles to the conserved muscles of the recipient. The authors should present electromyographic data to document active movement in the graft. Without an active nerve supply, the graft musculature will undergo progressive atrophy.

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1. Dubernard J-M, Lengelé B, Morelon E, et al. Outcomes 18 months after the first human partial face transplantation. *N Engl J Med* 2007;357:2451-60.

THE AUTHORS REPLY: We surmise that the recovery of muscle function inside the face graft occurred mostly through a phenomenon of intramuscular innervation along the multiple bilateral sites of muscular anastomoses and not through axonal regeneration arising from the single, small nerve anastomosis that was performed on the lower left part of the transplant. All depressor muscles on the lower face had been avulsed completely, along with the right and left mandibular (or inferior marginal) branches of the facial nerves. We repaired the nerves on the left

side of the face with the aim of reanimating the lower lip and the chin but could not do the same on the right side because the thin corresponding nerve was absent in the graft.¹ Early in the postoperative period, dynamic motions in the face graft were apparently due to contractions arising from the recipient’s remaining muscle bellies, transmitted to the homologous allogeneic muscles to which they were connected. We speculate that as intramuscular innervation progressed, motor-nerve ingrowth through anastomoses of the levator muscles resulted in secondary dynamic expressive motions, involving the midface muscle segments from the donor. Restoration of complete lip occlusion probably occurred as a result of both intramuscular innervation of the upper part of the orbicularis oris muscle and intraneural axonal regeneration inside its lower sling.

We also wish to clarify our statement in the Methods section regarding consent. We noted that the patient gave her consent for the face transplantation; we wish to add that she also gave her consent for publication of the manuscript and for development and release of the accompanying video.

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Selective Publication of Antidepressant Trials

TO THE EDITOR: The importance of the study reported by Turner et al. (Jan. 17 issue),¹ on selective publication of antidepressant trials, can hardly be overstated because it shows how researchers and clinicians are deprived of accurate data, resulting in a wrong understanding of antidepressant efficacy. The most serious implication of this study is that guidelines for the treatment of depression may be inaccurate, since they are often based on meta-analyses of published data alone.^{2,3}

This current system makes it impossible for scientific journals to provide a valid picture of

the efficacy of antidepressants. Therefore, we propose that meta-analyses be accepted only when they include an adequate analysis of the potential bias due to exclusion of unpublished studies (beyond Egger plots, since large negative trials may also remain unpublished).

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1. Turner BH, Matthews AM, Linardatos E, Tell RA, Rosenthal R.