To the Editor:
I wish to urge caution in interpreting the results of the article written by Bochicchio et al. suggesting that endotracheal intubation in the field does not improve outcome in patients with traumatic brain injury (TBI).1 As Aeromedical Director of the program that delivered two-thirds of the patients in this study, I feel compelled to point out that the TBI patients that our flight paramedics intubate in the field are physiologically different from those for which intubation is held until hospital arrival.

Our criteria for rapid sequence intubation (RSI), as referenced by the authors, demonstrate that RSI is selectively applied to TBI patients in our statewide system. Patients with a prehospital GCS of ≤8 generally do not receive RSI in the field if they are maintaining an oxygen saturation >90 percent on their own, are protecting their airways, and do not have evidence of on-going aspiration. By contrast, the majority of our patients that receive field RSI have blood and/or vomitus in their airways and are requiring bag valve mask assistance to maintain oxygen saturations whose mean is 85 percent. The authors1 reported results of their airways and are protecting their airways. I suggest that the authors study this important question further using a methodology that attempts to account for patient physiologic status. The current data set may not prove effective for this purpose, as the prehospital data were not collected in a uniform fashion for this specific intent. I further suggest that when patient physiologic status is considered, it will be shown that correcting hypoxia in the field does indeed benefit TBI patients. In the interim, restraint should prevail in considering this article as evidence to the contrary.

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REFERENCES

The Authors’ Reply:
We would first like to thank Dr. Floccare for bringing up many valid points for discussion about our article.1 We agree that any manuscript which reports that more aggressive therapy may result in worse outcome should be approached with caution.

We agree that one of the weaknesses of the study was lack of physiologic data when comparing the field-intubated group to the non-field-intubated group. Although the patients were matched by ISS and AIS, we agree that having more objective physiologic data would make the analysis more robust. We are in the process of attempting to gather that data.

Adding to our interest however, are recent reports from the trauma literature that are in agreement with our findings. Davis et al. recently reported that paramedic RSI protocols to facilitate intubation of head-injured patients were associated with an increase in mortality and decrease in good outcomes versus matched historical controls.2 These authors also concluded that factors that may have contributed to the increase in mortality included transient hypoxia, inadvertent hyperventilation, and longer scene times associated with the RSI procedure. Murray et al. similarly concluded that patients with severe head injury and prehospital intubation did not demonstrate an improvement in survival.3

Thus, there appears to be growing data in support of our conclusions. However, only a carefully planned randomized prospective study would be able to answer this question definitively. Again, we would like to thank Dr. Floccare for his thoughts and recommendations, and we look forward to studying this problem more carefully at the R. Adams Cowley Shock Trauma Center in con-
juncture with the Maryland Institute for Emergency Medicine.

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REFERENCES

To the Editor:
An Alternative Treatment Option for Management of Zone III Wounds of the Internal Jugular Vein

I read with great interest the case report of Drs. Sanabria and Jimenez.1 They describe the management of an injury to the internal jugular at the level of the jugular foramen. After controlling the bleeding with a Foley probe, permanent occlusion of the vessel was achieved with an endovascular plug of n-butyl cyanoacrylate.

Such wounds present a challenge for any surgeon. If enough of the vein is present below the skull base, ligation is appropriate. However, if the vein is injured flush with the base of the skull, ligation is impossible. I have successfully treated two injuries such as this with an alternative maneuver that worked quite well.

The first injury occurred when a cliff climbing C-ring penetrated a patient’s neck just below the base of the skull. His internal jugular vein was completely severed at the jugular foramen. The other patient was shot in the neck, with a through-and-through wound to the internal jugular vein at the level of the foramen. It was impossible to ligate or repair either vessel at this level.

I successfully controlled the bleeding in both these patients with a sternocleidomastoid muscle flap. I took the muscle off at its attachment to the clavicle, reflected it up, and plugged the jugular vein stump with the muscle. It was secured with sutures to the surrounding tissue. The patient injured with the climbing instrument developed intracranial hypertension and died of a herniated brain stem, however he showed no evidence of bleeding from the jugular vein. The other patient did well and went home a few days after surgery.

Occluding the internal jugular vein when it is injured too high to ligate or repair can easily be accomplished by turning a sternocleidomastoid muscle flap up into the base of the skull. No further treatment of the injury is necessary. I offer this suggestion as an alternative to endovascular occlusion or temporary control with a Foley catheter.

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REFERENCE

To the Editor:
Cerebral Fat Embolism: An Indication for ICP monitor?

A 25-year-old man was admitted following a motor vehicle crash with injuries isolated to bilateral femur and right tibia-fibula fractures. Twelve hours later, there was a reduction in conscious level from a GCS of 15 to 10. An urgent head CT scan was performed and was normal. Hemodynamically he was stable, and oxygen saturations was 94 percent.

Supplementary oxygen was administered. Before the change in GCS, the patient complained of a transient loss of vision in his right eye, which resolved after 1 hour. Some petechial hemorrhages also developed in the upper chest region. A blood count showed a hemoglobin level of 10 from 13 g/dL and platelet count from 160 to 70 × 10^9/L. Based on the history, a diagnosis of fat embolism was made, and surgery to the patient’s fractures was performed the following morning. Surgery was uneventful with no sudden drop in saturations, end tidal carbon dioxide, and blood pressure. Surgery was completed after 10 hours and the patient was transferred into the post-anesthetic care area for monitoring. He was sedated with propofol with morphine and ventilated overnight. Hourly neurologic observations were started. All his vital signs were satisfactory, his inspired oxygen concentration was 40 percent, and his pupils were equal and reacting to light. Twelve hours after completion of surgery, the patient suddenly developed fixed and dilated pupils. Cerebral protection strategies were commenced and an urgent CT scan was performed showing severe cerebral edema. He failed to recover and was declared dead 36 hours later.

Indications for insertion of an intracranial pressure (ICP) monitor include severe traumatic brain injury and mild to moderate injury fulfilling certain criteria.1 However, other clinical conditions may warrant insertion of ICP monitor such as in acute hepatic failure2 or in the case where monitoring of ICP is essential, such as in laparoscopic surgery where intracranial compliance is limited.3 Our patient had an unexpected outcome. The first head CT scan did not reveal radiologic changes with the drop in GCS of 5 points. An MRI scan at this point may reveal cerebral fat embolism though it may not have the changed the management.4 Following surgery, the patient was ventilated and sedated with propofol. The patient received morphine for analgesia in the post-anesthetic care area. In retrospect, an ICP monitor may be of benefit if inserted preoperatively for the following reasons.
Cerebral edema is a recognized complication of FES. A drop in GCS of 5 points is significant.

Serial neurological examination is precluded by anaesthesia, paralysis, and sedation given.

Intracranial hypertension may worsen until a point above which herniation occurs and pupillary signs may be a relatively late sign.

An ICP monitor will allow earlier detection and cerebral protection strategies to commence. Decompression craniectomy, although of benefit shown in children only is a debatable option. Death could have been averted. Based on this case, in cases of cerebral fat embolism with evidence of neurologic deterioration (in this case a change in GCS) and where assessment of neurologic status is affected by anesthesia or sedation, an ICP monitor may be useful if inserted preoperatively.

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