Predictors of outcome for gunshot wounds

TO THE EDITOR: I have read with great interest the article by Gressot et al. (Gressot LV, Chamoun RB, Patel AJ, et al: Predictors of outcome in civilians with gunshot wounds to the head upon presentation. J Neurosurg 121:645–652, September 2014). The authors concluded that several factors, including patient age, Glasgow Coma Scale score, nonreactive pupils, and the path of the bullet and its fragments on CT scans, have predictive value for patient survival, and they created a scoring system based on these parameters. In their series of 119 patients 19% had good functional survival. We published an article in 1979 dealing with the same issues. In our study of 82 patients with gunshot wounds sustained in civilian life, we evaluated the same presentation parameters as the authors and we assessed functional outcome. We also evaluated surgical findings, specifically the incidence of hematomas either at surgery or at autopsy in all patients who did not survive. Although we evaluated the importance of the same criteria, we concluded that the only truly meaningful parameter predicting patient survival was the state of consciousness on admission. While all the other parameters had some value, they were of secondary importance in predicting survival and its quality. There were 4 groups of patients based on the state of consciousness at the time of presentation: I) alert, awake; II) obtundation with or without neurological deficit; III) unresponsive to all but noxious stimulation in appropriate or semi-appropriate fashion; and IV) comatose inappropriate or no response at all.

We have found that this system provides as useful information as the point system proposed by the authors, although it is substantially simpler. Adding additional information did not increase the accuracy or reliability of our basic scale. For instance, while a bihemispheric bullet path generally indicates a poor prognosis, if the examination results fall into Group I or II, it is the state of consciousness that determines the prognosis rather than the path of the bullet. Similarly, if a patient has fixed, dilated pupils, the prognosis is poor, but patients in this case will undoubtedly be comatose with, at best, a decerebrate posture, which would place them in Group IV in which there are no survivors. Again, it is the state of consciousness that determines outcome. This simplified neurological examination makes it easier to rapidly assess patients with gunshot wounds, particularly if non-neurosurgeons in the emergency department triage the patients. It provides rapid and accurate early information for patients and their families.

I was intrigued by the fact that in our study, 39% of patients achieved a functional survival status compared to 19% in the authors’ study. The extent of injury caused by a bullet is determined to the greatest degree at the time of impact and is dependent on bullet mass and exit muzzle velocity squared. Passage of the bullet through brain tissue creates waves of massive increases in intracranial pressure in the wake of the bullet. Based on the above formula, the damage is greater with a greater bullet mass and greater muzzle exit velocity such as that seen in military grade weapons. Thus, the degree of neurological deficit is determined at time of impact in most instances. The development of mass lesions, such as hematomas, is rare and was seen in only 10% of our cases. Admitting that neither the authors nor we have studied the ballistic profile of the guns used, it is tempting to think that the difference in functional survival between our studies most likely reflects the currently greater availability of better quality weapons with larger bullet sizes and greater muzzle exit velocities. These cause much more damage than the “Saturday night specials” commonly used at the time of our study and could explain this discrepancy.

The authors are to be congratulated for continuing to study the problem of gunshot injuries, which are becoming more and more serious, particularly with easier access to higher-grade weapons in the civilian population.

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DISCLOSURE
The author reports no conflict of interest.

References
Response

We recognize Dr. Hubschmann and colleagues for their pioneering work in this area. Their study was performed between 1973 and 1975, and they did not have the benefit of routine CT scanning in evaluating patients with gunshot wounds to the head. While they found that the state of consciousness upon arrival was the only meaningful predictive measure of outcome, we found that the Glasgow Coma Scale score upon arrival was less important than pupillary reactivity and bullet trajectory as seen on CT scan upon arrival. As imaging technology continues to improve, we expect our understanding and evaluation of these patients to evolve as well.

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The blood-hammer effect and aneurysmal basilar artery bifurcation angles

TO THE EDITOR: The interesting study by Tütüncü et al.1 (Tütüncü F, Schimansky S, Baharoglu MI, et al: Widening of the basilar bifurcation angle: association with presence of intracranial aneurysm, age, and female sex. J Neurosurg 121:1401–1410. December 2014) seems to support the idea that unfavorable vessel design in arterial bifurcations may be a risk factor in intracranial aneurysm formation and progression. The hypothesis that aneurysmal basilar artery (BA) bifurcations present increased angles compared with nonaneurysmal basilar bifurcations was scrutinized. The BA bifurcation (α) angle, estimated in age-matched cohorts, was found to be significantly wider in patients with a BA aneurysm (146.7° ± 20.5°) than in patients with an aneurysm in another cerebral artery (non-BA) (111.7° ± 18°) and in a nonaneurysmal control group (103° ± 20.6°). Also in the non-BA aneurysm group, the α was significantly wider than in the control group.

Aneurysm size, height, and neck presented a significant positive correlation with the α angle, which was also independently predicted by the size of the aneurysm neck in multivariate analysis. In nonaneurysmal controls, α grew wider with increasing age, with a steeper dependence in female than in male patients. In computational fluid dynamics (CFD) simulations, done on parametric BA models (with radii of mother and symmetrical daughter vessels following the vascular optimality principle) as well as on patient-derived models of the BA bifurcation, low wall shear stress (WSS) at the area of flow impingement at the bifurcation apex was spatially contiguous with an acceleration area along the daughter branches (where WSS increased abruptly to maximal values) that was followed by a deceleration area (where WSS gradually decreased). Both the flow impingement area and the acceleration area increased with increasing α angles on CFD simulations. The vertebrobasilar junction angles were also evaluated, but no significant geometrical changes were observed in any patient group.

It is appealing to speculate that the observed strong correlation between the BA α angle and aneurysm presence, and the finding that in patients with aneurysms at sites other than the BA the α angle was significantly wider than in the control group (the authors suggested that this denotes a global arterial weakening at bifurcation sites throughout the cerebral circulation in patients with aneurysms), indicates that an increased α angle can precede aneurysm formation. The authors cautiously and correctly dismissed such a causal relation, but this hypothesis surely is worth keeping in mind in future observations (prospective observations would be necessary to ascertain temporal priority). Moreover, the difference in α angles in the BA aneurysm group and in the non-BA aneurysm group (35° on average) was much larger than the difference between the non-BA cases and the controls (8.7°), and can reasonably result from aneurysm formation and progression.

Apart from the intuitively understandable fact that a saccular aneurysm is a focus of vessel wall rupture and that a lumen herniation in a pouch of vessel wall remnants usually located just at the apex of the α angle would increase this angle even more, in terminal aneurysms every systolic blood pressure wave exposes the aneurysm sac to the “blood-hammer” effect.1,2 This phenomenon, known in hydraulic engineering as the “water hammer,” occurs when flow of fluid in a pipe is stopped by abrupt closure of a valve, reducing the kinetic energy of the upstream fluid to zero, briefly creating an overpressure at the valve that adds to the steady pressure, and causing a pressure wave to move upstream at the speed of sound that is followed by secondary (“bouncing”) waves until the fluid comes to rest. Ahlqvist3 provided a simple equation to estimate the overpressure (ΔP): ΔP = ρv[(K + d/E)b]1/2.

Assuming physiological or nearly analogous values for blood flow velocity (v), blood density (ρ), blood compressibility (K), vessel wall thickness (h), and elastic modulus (E) as provided by Ahlqvist in his hypothetical considerations about embolic occlusion of the middle cerebral artery, but adjusting the internal arterial diameter (d) to 3.5 mm for the BA and assuming the presence of a BA tip aneurysm whose neck is as wide as the BA, the overpressure provoked by the impact of the blood flow at the domus of the aneurysm, that is reflected upstream in the BA bifurcation, is approximately 55 mm Hg.

In other words, at the apex of the BA aneurysm sac a sudden arterial pressure increase of 55 mm Hg is added to the blood pressure at the parent artery at every heartbeat, and this pressure wave is transmitted upstream along the aneurysm wall to the aneurysm ostium and parent artery. The overpressure is added to and does not depend on the baseline arterial blood pressure. Compared to abrupt embolic occlusion of an artery, the primary wave probably spreads along a shorter distance (the posterior cerebral