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THE AUTHORS REPLY: We concur with Páramo that beyond modulating the ultimate clot size and lysis, the fibrinolytic cascade proteins (including TAFI¹) may also contribute to the pathophysiology of cardiovascular disease. Nevertheless, the exact role of fibrinolysis in atherosclerosis remains highly controversial to date. Owing to space limitations, we restricted the scope of the review and did not cover this topic.

Hypofibrinolysis has been proposed as a determinant of cardiovascular disease in previous studies.² Aside from their recognized effects on fibrin clot lysis, fibrinolytic proteins, including the enzyme plasmin, should be recognized for their involvement in regulating many other actions, such as the plasmin-mediated accelerated inflammation and degradation of matrix proteins. Plasmin activates distinct matrix metalloproteinases (MMPs), such as MMP-3, 9, 12, and 13,³ which have contributed to enhanced elastolysis and collagenolysis, tunica media destruction, and aneurysm formation in atherosclerotic mice

models.⁴ Furthermore, despite the atheroprotective effect of plasminogen and urokinase-type plasminogen activator, the loss of plasminogen activator inhibitor 1 in atherosclerotic mice has been paradoxically linked to increased plaque growth and extracellular matrix deposition.⁵ Hence, the net effects of a fibrinolytic imbalance between plasminogen activators and their inhibitors on atherosclerosis progression remain poorly understood.

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Since publication of their article, the authors report no further potential conflict of interest.

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In Vivo Biomechanical Measurements of a Football Player's C6 Spine Fracture

TO THE EDITOR: During an investigation of concussion in American football players, we captured in vivo biomechanical data on a cervical spine fracture as it occurred in a male athlete (age, 18 years; height, 189.0 cm; weight, 79.4 kg) who was performing a head-down tackling maneuver. The cornerback's helmet was equipped with the Head Impact Telemetry System (Simbex), a six-accelerometer array that measures the location and magnitude of an impact. The impact magnitude was quantified by measuring peak linear and rotational acceleration of the head with the use of the Gadd Severity Index (GSI) and

Head Injury Criteria (HIC).^{1,2} The GSI and HIC are mathematically weighted measures of head acceleration and the duration of impact, with higher scores representing increased likelihood of injury.

After being transported to the emergency department, the athlete reported having pain in the head, neck, and lower back (severity between 3 and 5 on a scale of 0 through 10, with 0 indicating no pain and 10 indicating most severe pain) and losing consciousness for less than 10 seconds at the time of injury. A computed tomographic (CT) scan of the brain was normal, but

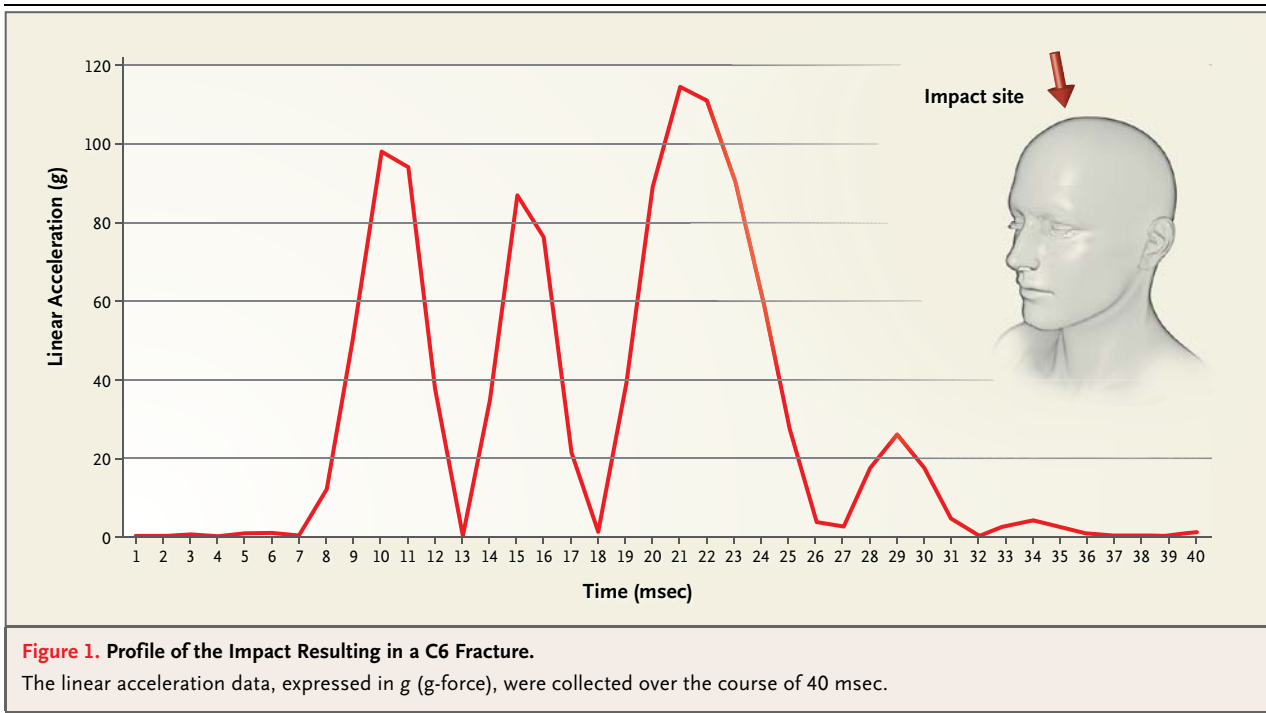


Figure 1. Profile of the Impact Resulting in a C6 Fracture.

The linear acceleration data, expressed in g (g-force), were collected over the course of 40 msec.

a CT scan of the cervical spine revealed a fracture of the left facet of C6 at the inferior articulating process that extended into the lamina junction. There were no other misalignments or injuries. A scan obtained with the use of magnetic resonance imaging showed left-sided joint effusion and muscle edema at C6 and C7, but no other abnormalities were detected. The on-call neurosurgeon confirmed a diagnosis of concussion and a stable left C6 facet fracture with no neurologic sequelae. The athlete was discharged within 48 hours with instructions to wear a hard collar. He was disqualified from any further participation in football, but a CT scan obtained at a 12-week follow-up assessment revealed complete healing, and the patient was cleared to participate in basketball.

Examination of video footage (available with the full text of this letter at NEJM.org) confirmed that the impact occurred at the top right side of the helmet (162 degrees of azimuth and 76 degrees of elevation), producing a peak linear acceleration of $114\times g$ (g is g-force, or 9.8 m per second squared) and a rotational acceleration of 3318 rad (radians) per second squared. In 16 other athletes for whom we have recorded similar types

of impact, the mean values were $98\times g$ and 6548 rad per second squared. However, the impact resulting in cervical fracture produced a GSI score of 812 and an HIC score of 487, both of which are substantially higher than the scores recorded for other athletes in similar circumstances (289.1 and 187.4, respectively). The higher scores for this athlete are probably a result of the longer duration of the impact (approximately 20 msec) (Fig. 1). The mechanism of injury is consistent with sports-related spine injuries produced by an axial loading of the neck that results from the combined force of the head impact and the inertial loading from the torso.³

Sporting and recreational activities are the second most common cause of cervical spine injury for persons younger than 30 years of age.⁴ These injuries have an average lifetime cost of more than \$3 million.⁵

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A video showing a football tackle resulting in a cervical spine fracture is available at NEJM.org

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Disclosure forms provided by the authors are available with the full text of this letter at NEJM.org.

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CORRECTION

Brief Report: A Hemoglobin Variant Associated with Neonatal Cyanosis and Anemia (May 12, 2011;364:1837-43). In Figure 1B (page 1838), the father's genotype should have been "WT/V67M," rather than "WT/WT." We regret the error. The article is correct at NEJM.org.

NOTICES

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