Commotio cordis due to high-velocity projectile ejected from an industrial lawnmower

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Introduction
Commotio cordis is a term used to describe ventricular fibrillation (VF) triggered by a blunt, nonpenetrating blow, often the result of a small ball or puck hitting the chest in the course of recreational or competitive sports.1 It is typically fatal, though rates of successful resuscitation have improved in recent years.2,3 Experimental studies have identified a 10–40-ms window during the upstroke of the T wave during which a blow of sufficient force can elicit VF, with 40 mph being the optimal projectile velocity for inducing VF in a swine model.1,4 Children engaged in sports with the potential for high-impact blows are most at risk.

Case report
A 22-year-old man was working with a landscaping crew when he was struck in the chest by a lacrosse ball that had been ejected by an industrial lawnmower. He immediately lost consciousness and was pulseless, and a bystander initiated cardiopulmonary resuscitation. Paramedics arrived within 10 minutes and found the rhythm to be VF. A single biphasic shock of 200 J was delivered, restoring sinus rhythm.

He was taken to the emergency department of a nearby tertiary care hospital where he was hemodynamically stable but vomiting and not following commands. A severe contusion over the precordium was noted (Figure 1). The initial electrocardiogram showed sinus rhythm with a broad atypical right bundle branch block and downsloping ST segment in lead V1, which raised concern during the initial evaluation for possible Brugada syndrome (Figure 2).

Because the patient was encephalopathic, therapeutic hypothermia was initiated for neurologic protection. Limited transthoracic echocardiogram showed global hypokinesis with left ventricular ejection fraction of 40%–50%. The computed tomographic scan of the chest showed parasternal parenchymal opacities but no fractures. He completed the hypothermia protocol (24 hours at 32°C–34°C with controlled rewarming) without incident. The transthoracic echocardiogram on hospital day 3 showed normalization of left ventricular ejection fraction to 65%–75% with no other abnormalities. Biochemical testing showed an elevated level of troponin I, with a peak value of 8.56 at 10 hours after the event. By hospital day 4, the patient had full neurologic recovery and was discharged from the hospital. The electrocardiogram had nearly normalized but had a persistent rSr′ pattern with mild residual ST-segment elevation in lead V1 (Figure 3). The lawnmower responsible for the projectile had a standard blade tip speed of 210 mph (338 km/h) as per the manufacturer’s specifications. Given the severity of the chest contusion, it was assumed that the projectile was likely traveling near this speed.

Discussion
The mechanism by which mechanical impact leads to VF has been studied extensively over the last 2 decades and is currently thought to result from a transient but dramatic
increase in left ventricular intracavitary pressure. It is sug-
gested that this increase in pressure leads, through electrome-
chancial coupling, to heterogeneous repolarization and high susceptibility to ventricular fibrillation.

Blows that cause commotio cordis may lead to other electrocardiogram abnormalities that could obscure the diagnosis. Brugada syndrome may be mimicked by concomitant cardiac contusion and bundle branch block (as seen in this case). Clinical history and examination are needed to make an accurate diagnosis, which has important implications for treatment.

Optimal projectile velocity for ventricular fibrillation induction through commotio cordis is 40 mph in a small swine model (8–25 kg). The relatively low incidence of commotio cordis in adults may be due, in part, to projectiles rarely traveling at a sufficient velocity to adequately compress the adult chest cage.

Conclusion
We have presented a case of commotio cordis in an adult occurring with an unusually high projectile velocity. The patient displayed evidence of myocardial injury, but the immediate collapse indicates this was likely a true case of commotio cordis. The relatively low incidence of commotio cordis in adults may be due, in part, to projectiles rarely traveling at a sufficient velocity to adequately compress the adult chest cage.
Figure 2  The presenting electrocardiogram showed sinus rhythm with a broad atypical right bundle branch block and downsloping ST segment in lead V1, which initially raised concern for Brugada syndrome.

Figure 3  On hospital day 4, the electrocardiogram had nearly normalized but had a persistent rSr' pattern with mild residual ST-segment elevation in lead V1.
Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at 10.1016/j.hrcr.2015.01.014.

References

1. Maron BJ, Estes NA III. Commotio cordis. N Engl J Med 2010;362:917–927.
2. Maron BJ, Haas TS, Ahlawat A, Barberich RF, Estes NA III, Link MS. Increasing survival rate from commotio cordis. Heart Rhythm 2013;10:219–223.
3. Link MS. Pathophysiology, prevention, and treatment of commotio cordis. Curr Cardiol Rep 2014;16:495.
4. Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zha W, Estes NA III. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). J Am Coll Cardiol 2003;41:99–104.
5. Link MS, Wang PJ, VanderBrink BA, Avelar E, Pandian NG, Maron BJ, Estes NA III. Selective activation of the K(+)(ATP) channel is a mechanism by which sudden death is produced by low-energy chest-wall impact (commotio cordis). Circulation 1999;100:413–418.
6. Kohl P, Nesbitt AD, Cooper PJ, Lei M. Sudden cardiac death by commotio cordis: role of mechano-electric feedback. Cardiovasc Res 2001;50:280–289.
7. Bode F, Franz MR, Wilke I, Bonnemeier H, Schunkert H, Wiegand UK. Ventricular fibrillation induced by stretch pulse: implications for sudden death due to commotio cordis. J Cardiovasc Electrophysiol 2006;17:1011–1017.
8. Madias C, Maron BJ, Supron S, Estes NA III, Link MS. Cell membrane stretch and chest blow-induced ventricular fibrillation: commotio cordis. J Cardiovasc Electrophysiol 2008;19:1304–1309.
9. Link MS. Commotio cordis: ventricular fibrillation triggered by chest impact-induced abnormalities in repolarization. Circ Arrhythm Electrophysiol 2012;5:425–432.
10. Link MS, Wang PJ, Pandian NG, Bharati S, Udelson JE, Lee MY, Vecchiotti MA, VanderBrink BA, Maron BJ, Estes NA III. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). N Engl J Med 1998;338:1805–1811.
11. Link MS, Maron BJ, Wang PJ, Pandian NG, VanderBrink BA, Estes NA III. Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. Pediatrics 2002;109:873–877.
12. Guinness Book of World Records. Bantam Books. New York. 2013.
13. Brun PM, Bessereau J, Chenaitia H, Barberis C, Peyrol M. Commotio cordis as a result of neutralization shot with the Flash Ball less-lethal weapon. Int J Cardiol 2012;158:e47–e48.
14. Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zha W, Estes NA III. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio cordis). J Am Coll Cardiol 2003;41:99–104.