

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



Jay A. Montgomery, MD,^{*} Dan M. Roden, MD, FHRS[†]

From the ^{*}Department of Medicine, Vanderbilt University, Nashville, Tennessee, and the [†]Departments of Medicine and Pharmacology, Vanderbilt University, Nashville, Tennessee.

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



Figure 1 A severe contusion was noted over the precordium upon presentation to the emergency department, 45 minutes after the initial injury. The lacrosse ball is also shown.

KEYWORDS Brugada syndrome; Cardiac contusion; Commotio cordis; High-velocity death; Ventricular fibrillation

ABBREVIATIONS VF = ventricular fibrillation
(Heart Rhythm Case Reports 2015;1:172–175)

Address reprint requests and correspondence: Dr. Jay A. Montgomery, Division of Cardiovascular Medicine, Vanderbilt University, 383 PRB, 2200 Pierce Avenue, Nashville, TN 37323-6300. E-mail address: jay.a.montgomery@vanderbilt.edu.

KEY TEACHING POINTS

- Commotio cordis is a term used to describe ventricular fibrillation caused by a nonpenetrating blow to the precordium during a 10–40-ms window before the peak of the T wave. It occurs most often in children engaged in recreational or competitive sports.
- The mechanism of commotio cordis appears to be related to a rapid rise in the intracavitary pressure of the left ventricle. This leads, through electromechanical 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.

increase in left ventricular intracavitary pressure. It is suggested that this increase in pressure leads, through electromechanical coupling, to heterogeneous repolarization and high susceptibility to VF, possibly through activation of the adenosine triphosphate-sensitive potassium channel.^{1,5–9} Commotio cordis is not induced by blows away from the precordium or outside the 10–40-ms window before the peak of the T wave and is more likely to occur with smaller, harder objects.^{10,11}

The US Commotio Cordis Registry for the years 1970–2012 reports a 72% mortality rate.² However, there has been improvement in outcomes, with the most recent 6-year period showing 58% survival. This improvement is thought to be primarily due to earlier resuscitation and

defibrillation.^{1,2} Although there was no automatic external defibrillator available onsite at the time of our patient's collapse, immediate bystander cardiopulmonary resuscitation was likely at least partially responsible for the favorable outcome.

It is rare to encounter projectiles traveling near 200 mph: bullets travel much faster and balls, pucks, and motor vehicles typically travel considerably slower. In sports, only the world record golf ball drive (211 mph) and jai alai serve (188 mph) approximate this velocity, with hockey, baseball, and lacrosse each having maximum recorded velocities slightly more than 100 mph.¹² Over the past few decades, some law enforcement agencies have adopted “less lethal” technology using foam rubber or “beanbag” projectiles traveling up to 267 mph to deliver nonlethal injuries to aggressive suspects. There has been at least 1 fatal report of commotio cordis owing to a “rubber bullet” impact over the precordium in an adult suspect.¹³

Link et al¹⁴ have previously shown in a juvenile swine model (weighing 8–25 kg) that 40 mph was the optimal velocity for inducing commotio cordis with a baseball. Higher velocities often resulted in myocardial tears or papillary muscle rupture. In the same study, intracavitary left ventricular pressure increases of 250–450 mm Hg had the highest likelihood of causing VF. It is also notable that bundle branch block and ST-segment elevation (as seen in our patient) were uniformly seen after impacts of 60 mph or greater in the swine model, likely a reflection of myocardial contusion in addition to commotio cordis-related VF.

It has been hypothesized that the rarity of commotio cordis in adults, in addition to being related to decreased exposure to sports, is likely the result of a less compliant chest cage, which blunts the impact of a precordial blow.¹ The rarity of commotio cordis in adults is also likely due, in part, to the significantly higher velocities that may be required to generate the same sudden rise in intracavitary pressure. To our knowledge, this hypothesis has not been tested in large-scale animal experiments.

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.

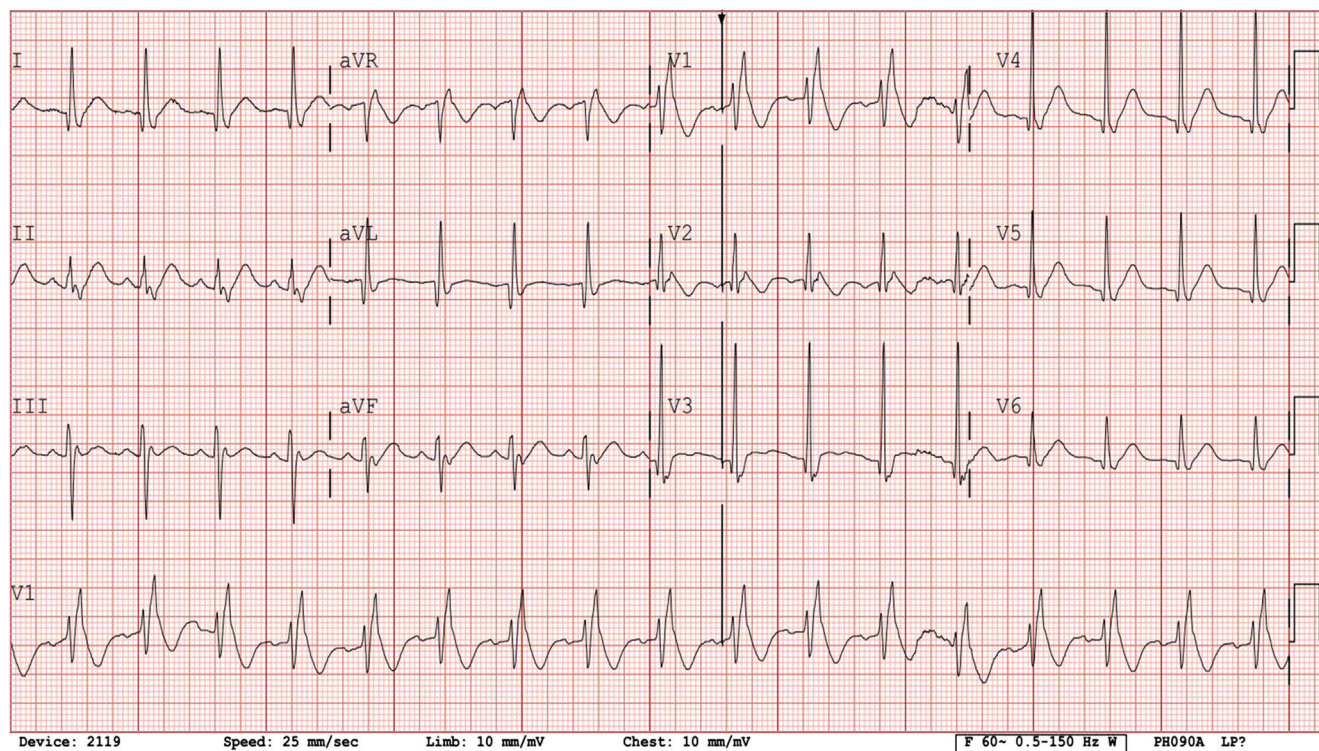


Figure 2 The presenting electrocardiogram showed sinus rhythm with a broad atypical right bundle branch block and downsloping ST segment in lead V₁, which initially raised concern for Brugada syndrome.

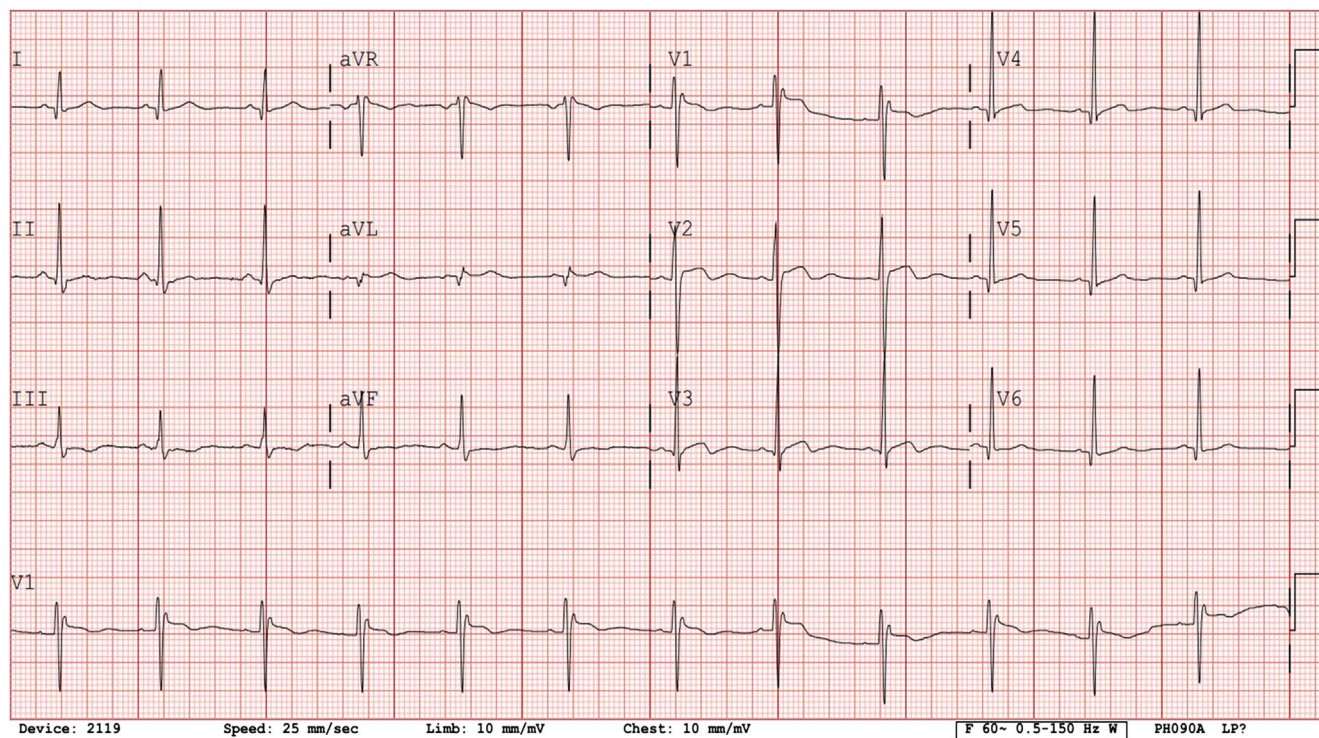


Figure 3 On hospital day 4, the electrocardiogram had nearly normalized but had a persistent rSr' pattern with mild residual ST-segment elevation in lead V₁.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at [10.1016/j.hrcre.2015.01.014](https://doi.org/10.1016/j.hrcre.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, Ahluwalia 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.