**PAWĘSKA, Wojciech, DOMINIK, Hanna, RADWAŃSKI, Jan, CZUDY, Zuzanna, POLATOWSKA, Marika, GADOMSKA, Anna, KUREK, Justyna, GORZYŃSKI, Rafał, GARSTKA, Michał & MOLCZYK-SIEŃCZAK, Justyna. Commotio cordis – a review of a rare cause of sudden death in young athletes. Journal of Education, Health and Sport. 2023;40(1):83-93. eISSN 2391-8306. DOI http://dx.doi.org/10.12775/JEHS.2023.40.01.008**
**https://apcz.umk.pl/JEHS/article/view/44074**
**https://zenodo.org/record/8052816**

**The journal has had 40 points in Ministry of Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of December 21, 2021. No. 32343.**
**Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical Culture Sciences (Field of Medical sciences and health sciences); Health Sciences (Field of Medical Sciences and Health Sciences).**
**Punkty Ministerialne z 2019 - aktualny rok 40 punktów. Załącznik do komunikatu Ministra Edukacji i Nauki z dnia 21 grudnia 2021 r. Lp. 32343. Posiada Unikatowy Identyfikator Czasopisma: 201159.**
**Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).**

**The authors declare that there is no conflict of interests regarding the publication of this paper.**
**Received: 19.05.2023. Revised: 15.06.2023. Accepted: 18.06.2023. Published: 21.06.2023.**

# Commotio cordis – a review of a rare cause of sudden death in young athletes

1. Wojciech Pawęska MD, ORCID: 0009-0007-0836-4983, District Hospital, Krakowska 3, 32-700, Bochnia, Poland e-mail: wojciech.paweska@gmail.com
2. Hanna Dominik MD, ORCID: 0000-0003-0371-2276, University Hospital, Zyty 26, 65-046 Zielona Gora, Poland e-mail: hania.dominik31@gmail.com
3. Jan Radwański MD, ORCID: 0009-0009-1441-8057, Provincial Specialist Hospital No. 4, Aleja Legionow 10, 41-902 Bytom, Poland e-mail: jan.radwanski97@gmail.com
4. Zuzanna Czudy MD, ORCID: 0000-0002-1619-4343, University Hospital, Collegium Medicum University of Zielona Gora, Zyty 28, 65-046 Zielona Gora, Poland e-mail: zuzanna.czudy@o2.pl
5. Marika Polatowska, 5th year medical student, ORCID: 0009-0007-8191-4566, Collegium Medicum University of Zielona Gora, Zyty 28, Zielona Gora 65-046, Poland e-mail: marika85588@gmail.com
6. Anna Gadomska MD, ORCID: 0009-0004-4029-3925, University Hospital, Zyty 26, 65-046 Zielona Gora, Poland e-mail: anna.gadomska@hotmail.com
7. Justyna Kurek MD, ORCID: 0009-0000-3828-9303, Clinical Hospital No. 2, Lwowska 60, 35-301 Rzeszow, Poland e-mail: jotkurek@gmail.com
8. Rafał Gorzyński MD, ORCID: 0009-0001-0815-1247, Clinical Hospital, Collegium Medicum University of Poznan, Dluga ½, 61-848 Poznan, Poland e-mail: rafal.gorzynski2@gmail.com
9. Michał Garstka MD, ORCID: 0009-0007-6152-9388 Medical Center HCP, 28 Czerwca 1956 r. 194, 61-485 Poznan, Poland e-mail: lek.garstka@gmail.com

10. Justyna Molczyk-Sieńczak DMD, ORCID: 0009-0007-8037-6305 NZOZ Alicja Ulanecka, Moscickiego 14, 33-100 Tarnow, Poland e-mail: justymolz@gmail.com

**Corresponding author**
Wojciech Pawęska MD,
District Hospital, Krakowska 3, 32-700, Bochnia, Poland
+48 535 298 631
wojciech.paweska@gmail.com

**Abstract**

**Introduction**: Commotio cordis, from Latin "agitation of the heart" is a potentially fatal sudden disruption and alteration of heart electric activity. It is usually caused by relatively mild blunt non-penetrating trauma to the specific point on anterior part of the chest wall during beginning of the heart muscle repolarization phase. It leads to ventricular fibrillation presenting as a collapse few seconds after the blow. It can be lethal without taking proper steps of management in a short ammount of time.

**Materials and Methods**: databases including "PubMed", "Cochrane" and "Google Scholar"

**Keywords**: commotio cordis; ventricular fibrillation; sudden death; sudden cardiac death; sudden cardiac arrest; AED

**Abbreviations:** CC - commotio cordis, VF - ventricular fibrillation, AED - automatic external defibrillator

## History

CC is not a new phenomenon, first mention of incident resembling CC dates to 1879 and it describes a man dying shortly after being struck into upper sternum with a stone.[30] Another case comes from a note in a British newspaper from 1898 mentioning death of a 13 year old boy struck by a cricket ball during practice game. Cause of death was back then described as a "shock to the heart".[1] From 1932 to 1934 Schlomka and Hinrichs conducted number of studies that resulted in describing, dividing and naming non-penetrating chest wall injuries depending on severity of heart damage.[28] They distinguished commotio cordis and contusio cordis - first of which came with no evidence of structural heart trauma and the latter with visible damage. Results of their studies on rabbit models proved that blow to a chest wall can

produce VF and other ECG abnormalities like ST-segment shifts and conduction blocks. However, the mechanism was unclear. Some of the theories suggested abnormal vagal nerve response or mechanically induced coronary vasoconstriction, both of which were subsequently proved unlikely. During the post-war period number of publications about CC decreased. The condition started to draw interest again during 1970s, bringing deeper understanding of its epidemiology. Multiple studies on porcine models, conducted in 1990s, confirmed VF as a direct cause of death in CC.[31] A study on rabbits from 2006 examined action potential of left ventricle muscle in response to sudden stretch. It threw a light on a mechanism of CC proving that sudden stretch of myocardium during a vulnerable period of repolarization can elicit VF.[14] In 1996, The United States Commotio Cordis Registry was founded to gather data about CC incidences. The oldest case included in this register comes from 1958 with further gap of almost no cases until late 1970s when more incidents of CC started to be described**.** This gap shows a probable underreporting of cases due to low recognition of the disease.[25] [26]

**Epidemiology**

Commotio cordis, even though being relatively rare, is the second leading cause of a sudden cardiac death among young athletes in the USA. It is outnumbered only by hypertrophic cardiomyopathy and followed by coronary-artery anomalies.[2] It is estimated that occurrence of death related to commotio cordis ranges between 10 - 20 incidences a year.[3] Typically affected are young people without any medical history of previous heart disease, either structural or electrical. Mean age of patients is 15 years. Most cases occurre between 10 and 20 years of age, although people younger and older can also be affected. It is thought that more compliant chest wall at young age predisposes to occurrence of commotio cordis. Thin physique is also a risk factor as less tissue surrounds the heart. Young men more often than women or older people engage in contact sports and risky behaviours, making them 95% of cases. 78% of patients are white. Sports with the highest risk of CC occurrence include baseball (leading in number of cases), softball, lacrosse, hockey and martial arts. Majority of activities mentioned above include playing with a small object with hard core. Bigger balls with soft cores tend to deform after impact and absorb much of the energy, thus cases of commotio cordis, although happen, are rarer in sports like soccer or basketball. Smaller objects disperse less energy and are more likely to induce CC.[27] Even though commotio cordis often happens during sports, it has also been reported in other situations, such as collisions with body parts e.g. fists, knees and elbows. For more than 190 cases reported in

the United States 47% happened while playing sports.[8] Mortality related to commotio cordis consecutively decreased over the course of years.[10] This phenomenon was most likely observed due to increasing awareness about the condition and its treatment, broader availability of AED and better knowledge of proper cardiopulmonary resuscitation amongst the population. Data reports that survival rate of acknowledged cases of CC before 1975 was 0% (8 out of 8 people died). The mortality level was constantly decreasing – survival rate reached up to 58% during the period of 2006-2012 (31 out of 53 people were successfully resuscitated).[10]

**Mechanism**

In a study explaining possible mechanism of sudden death due to low-energy impact to the chest experimental swine model showed that appropriately timed blow using a wooden item (resembling a regulation baseball) at a velocity of 40-60 mph can trigger ventricular fibrillation. Six lead coronal electrocardiograms were mounted on experimental animals to monitor electrical activities of the heart. 9 out of 10 impacts timed 30 to 15 msec before peak of T wave (which makes 1-3% of cardiac cycle) provoked ventricular fibrillation. Impacts outside this window, but still on the upslope of T wave, were more likely to produce nonsustained VF.[9]

Another study proved that only direct blows over the cardiac silhouette result in triggering VF (12 of 78: 15% vs. 0 of 100 for noncardiac sites: $p < 0.0001$). Blows to the center of the heart (between 2nd and 4th rib at left side of the sternum) were more effective in generating VF than to the other sites of precordium (7 of 23; 30% vs 5 of 55; 9%, $p = 0.02$).[7] Besides that, 4 out of 10 impacts synchronized with QRS complex caused transient complete atrioventricular heart block lasting from 2 to 7 beats with consecutive spontaneous regain of physiological AV node function.[4] Considering transient character of the block, it can be assumed that it could lead to collapse but wouldn’t be fatal, so it is not a proposed mechanism for commotio cordis. Out of 22 impacts during times of cardiac cycle other than upslope of a T wave 0 provoked ventricular fibrillation. Another ECG findings after blow to the chest, irregardless of heart's electrical phase, were: ST segment elevation, left bundle-branch block, polymorphic ventricular tachycardia - none of which resembled life threatening event. It was estimated that for a baseball the most effective velocity in causing VF was that of 40 mph (effectiveness of 70%). Higher velocities -50 mph and above, tended to produce structural damage (e.g. valve rupture, cardiac muscle rupture, tamponade) rather than electrical disturbances.[9]

Rapid rise in left ventricular pressure after blow may result in mechanical activation of electrical system of the heart.[14] Depending of shape of the object, its velocity, hardness and location of impact the pressure it generates can vary.[23] Threshold pressure for generating VF was estimated to be around 250-300 mmHg and the highest probability of triggering VF occurred around 600 mmHg.[7] Further increase of generated peak pressure decreased likelihood of generating VF and increased rate of structural damage.

On the cellular level the mechanism is still not completely understood. It is assumed that sudden increase in pressure during early repolarization phase causes stretch of cellular membrane and activation of stretch-sensitive cation-nonselective channels. It is followed by mechano-electric coupling that triggers afterdepolarization and causes premature ventricular contraction that starts to circulate around myocardium producing VF.[17] [20] Blockade of stretch-sensitive potassium channels with glibenclamide in porcine models reduced incidence of CC-caused VF. As these channels are more active during hypoxic state, strenuous physical exercise can potentially increase likelihood of developing VF due to chest blow.[14] [15] [16]

In addition, experimental swine models showed that there was an exceptionally increased risk of generating chest impact-induced VF in 2% of researched subjects suggesting that there can exist a genetic predisposition. Besides that the study showed a great variety between researched subjects in terms of susceptibility to generating VF due to blow ranging from 0% to 100%.[6]

**Presentation and diagnosis**

CC usually presents as collapse immediately or few seconds after blunt chest trauma. In this few seconds, injured person can be still standing or continue previous activity. For instance, one of the cases reports a 7-years old boy that after being hit by a baseball still managed to pick up the ball and throw it.[29]

Severity of the event may remain unrecognized for the first seconds to minutes, as the impact to the chest may seem insignificant and harmless to surrounding people. It can delay accurate diagnosis and thus worsen the outcomes. In some sports like box or other martial arts the incident can resemble losing consciousness due to brain concussion (known as knock-out), concealing true nature of the event and extending reaction time. Mortality rate reaches up to 97% when proper action is not taken under 3 minutes, so it is crucial to assess vital signs immediately after person loses consciousness and start CPR if pulse is absent.[8] Proper

diagnosis is often made afterwards based on circumstances of the event and, if available, ECG readings showing VF from the time of cardiac arrest. After excluding other probable causes of cardiac arrest, a diagnosis of CC can be made.

**Post mortem diagnosis**

It is often impossible to give a certain diagnosis of death as a result commotio cordis as there are other possible causes of sudden cardiac death that cannot be excluded e.g. inherited arrhythmias disorder or arrhythmogenic cardiomyopathy. Being given the circumstances of the incident one can suspect CC with high probability. The criteria that should hint into diagnosing CC in post mortem evaluation are as follows:

1. Cardiovascular collapse preceded by blunt, non-penetrating trauma to the precordium.
2. Lack of structural damage of the chest wall, thoracic cavity or heart resembling contusio cordis as a result of the blow.
3. Negative medical history of underlying heart disease that could also possibly lead to sudden cardiac death.[5]
4. VF on ECG if it was obtained at the time of the incident.

**Treatment**

After diagnosis of cardiac arrest, the management is a standard CPR until a cardiac defibrillation can be performed using either AED or manual external defibrillator. Healthcare professionals, if not present, should be alerted immediately. The time appears to be vital. The initiation of CPR after trauma should be immediate. Survival rate equals 40% when assistance is rendered before 3 minutes passing and 5% if CPR begins after that time.[10] Defibrillation allows to cease VF and restore a normal heart rhythm so it should be performed as soon as possible to shorten the period of cerebral hypoxia and thus to improve patient's prognosis.[18] [19] In animal model, defibrillation after a minute of inducing VF appears to be successful in restoring spontaneous circulation in 100% of cases, 92% after 2 minutes, 45% after 4 minutes and 25% if it's done after six minutes.[22] Additional measures, such as airway support with ventilation and epinephrine administration, can be taken in cases of prolonged resuscitation. After successful restoration of normal heart rhythm, post-resuscitation care algorithm should be applied.[17] Survivors of CC should undergo extensive workup on underlying heart disease including ECG, echocardiogram, cardiac MRI, stress testing, testing for Brugada and long-QT syndromes as possible causes of cardiac arrest.[21]

**Prevention**

Generally, commotio cordis is hardly preventable. However, there are steps that can be taken to decrease the risk of sudden ventricular fibrillation due to trauma.

1. Education and AED

All competitors and spectators of competitive athletics should be aware of this potentially fatal condition and trained to perform CPR. Availability, proper placement of AED and understanding of its usefulness in situations of sudden cardiac arrest seem to be essential in increasing survival rate. AED should be located in a visible place in sport venues during every match and training. People should be advised to protect their chests against trauma and try to avoid being hit in precordial area. Rules forbidding blocking balls with chests in some sports could be implemented.

2. Avoiding high-risk sports among survivors

As there can be a genetical predisposition to developing VF as a result of chest blow, survivors are generally advised to avoid contact sports or other activities that carry risk of being hit on the chest wall.[6][21]

3. Better sport gear

In a study that evaluated the mechanism of commotio cordis using swine models, researchers tried to use three types of softer baseballs compared to regulation ball to conclude if using softer balls would reduce the risk of ventricular fibrillation. It appeared that incidence rate of ventricular arrhythmia decreased significantly with softer balls. The softest balls corresponded with the lowest risk.[4] [24] That study proves that there is room for improvement in terms of preventing commotio cordis amongst baseball players and possibly players of other ball-related activities with risk of being struck on the chest.

Studies on commercially available chest wall protectors did not show their effectiveness in preventing commotio cordis, but extensive research is needed to decide whether protectors that cover every part of the heart, even during body movement, would be effective. [11] [12] In a recent study from 2017, the researchers managed to construct a chest wall protection, from specific materials, that was significantly reducing risk of CC (from 54% to 5%) on animal models.[13] Keeping lives of young athletes in mind, it would be desirable to incorporate the results of this study into design of the sport gear.

**Summary**

Commotio cordis is rare but unpredictable and hardly preventable cause of death in otherwise healthy young people. Raising medical awareness amongst population can improve the

prompt recognition of the CC and help to act appropriately in order to prevent possible death. Knowing that the mortality rate is 97% if the condition is not treated under 3 minutes should indicate the importance of spreading knowledge about CC and taking proper preventing steps. Using risk-lowering sport gear is a method of prevention that often requires making some compromises in terms of convenience but should be taken under consideration especially within the group at risk as the life could be at stake.

**References**


1. Maron BJ, Doerer JJ, Haas TS, Estes 3rd NA, et al. Historical observation on commotio cordis. *Heart Rhythm*. 2006;3:605–606. doi: 10.1016/j.hrthm.2005.12.011. PMID: 16648070.
2. Maron BJ. Sudden death in young athletes. *N Engl J Med*. 2003;349:1064–1075. doi: 10.1056/NEJMra022783. PMID: 12968091.
3. Maron BJ, Estes 3rd NA. Commotio cordis. *N Engl J Med*. 2010;362:917–927. doi: 10.1056/NEJMra0910111. PMID: 20220186.
4. Link MS, Wang PJ, Pandian NG, et al. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). *N Engl J Med*. 1998;338:1805-1811. doi: 10.1056/NEJM199806183382504. PMID: 9632447.
5. Lupariello F, Curti SM, Di Vella G. Diagnostic Criteria for Commotio Cordis Caused by Violent Attack: Review of the Literature. Am J Forensic Med Pathol. 2018 Dec;39(4):330-336. doi: 10.1097/PAF.0000000000000433. PMID: 30234548.
6. Alsheikh-Ali AA, Madias C, Supran S, Link MS. Marked variability in susceptibility to ventricular fibrillation in an experimental commotio cordis model. *Circulation*. 2010;122:2499–2504. doi: 10.1161/CIRCULATIONAHA.110.955336. Epub 2010 Nov 29. PMID: 21126977.
7. Link MS, Maron BJ, VanderBrink BA, et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. *J Am Coll Cardiol*. 2001;37:649–654. doi: 10.1016/s0735-1097(00)01142-6. PMID: 11216992.
8. Palacio LE, Link MS. Commotio cordis. Sports Health. 2009 Mar;1(2):174-9. doi: 10.1177/1941738108330972. PMID: 23015869; PMCID: PMC3445066.
9. Link MS, Maron BJ, Wang PJ, VanderBrink BA, Zhu W, Estes 3rd NA. Upper and lower limits of vulnerability to sudden arrhythmic death with chest-wall impact (commotio

cordis). *J Am Coll Cardiol*. 2003;41:99–104. doi: 10.1016/s0735-1097(02)02669-4. PMID: 12570951.

10. Maron BJ, Haas TS, Ahluwalia A, Garberich RF, Estes NA 3rd, Link MS. Increasing survival rate from commotio cordis. Heart Rhythm. 2013 Feb;10(2):219-23. doi: 10.1016/j.hrthm.2012.10.034. Epub 2012 Oct 26. PMID: 23107651.
11. Doerer JJ, Haas TS, Estes 3rd NA, Link MS, Maron BJ. Evaluation of chest barriers for protection against sudden death due to commotio cordis. *Am J Cardiol*. 2007;99:857–859. doi: 10.1016/j.amjcard.2006.10.053. Epub 2007 Jan 26. PMID: 17350382.
12. Weinstock J, Maron BJ, Song C, Mane PP, Estes 3rd NA, Link MS. Failure of commercially available chest wall protectors to prevent sudden cardiac death induced by chest wall blows in an experimental model of commotio cordis. *Pediatrics*. 2006;117:e656–e662. doi: 10.1542/peds.2005-1270. Epub 2006 Mar 1. PMID: 16510614.
13. Kumar K, Mandleywala SN, Gannon MP, et al. Development of a chest wall protector effective in preventing sudden cardiac death by chest wall impact (commotio cordis). *Clin J Sport Med*. 2017;27:26–30. doi: 10.1097/JSM.0000000000000297. PMID: 27014942; PMCID: PMC5181132.
14. 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. doi: 10.1111/j.1540-8167.2006.00547.x. PMID: 16948746.
15. Quinn TA, Jin H, Lee P, Kohl P. Mechanically Induced Ectopy via Stretch-Activated Cation-Nonselective Channels Is Caused by Local Tissue Deformation and Results in Ventricular Fibrillation if Triggered on the Repolarization Wave Edge (Commotio Cordis). Circ Arrhythm Electrophysiol. 2017 Aug;10(8):e004777. doi: 10.1161/CIRCEP.116.004777. PMID: 28794084; PMCID: PMC5555388.
16. 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. doi: 10.1161/01.cir.100.4.413. PMID: 10421603.
17. Link, M.S. Pathophysiology, prevention, and treatment of commotio cordis. *Curr. Cardiol. Rep.* 2014, *16*, 495. doi: 10.1007/s11886-014-0495-2. PMID: 24760424.
18. Salib EA, Cyran SE, Cilley RE, Maron BJ, Thomas NJ. Efficacy of bystander cardiopulmonary resuscitation and out-of-hospital automated external defibrillation as

life-saving therapy in commotio cordis. J Pediatr. 2005;147:863–6. doi: 10.1016/j.jpeds.2005.07.041. PMID: 16356450.

19. Marenco JP, Wang PJ, Link MS, Homoud MK, Estes 3rd NA. Improving survival from sudden cardiac arrest: the role of the automated external defibrillator. JAMA. 2001;285:1193–200. doi: 10.1001/jama.285.9.1193. PMID: 11231750.
20. Kohl P, Nesbitt AD, Cooper PJ, Lei M. Sudden cardiac death by Commotio cordis: role of mechanico-electrical feedback. Cardiovasc Res. 2001;50:280–9. doi: 10.1016/s0008-6363(01)00194-8. PMID: 11334832.
21. Link MS, Estes NA 3rd, Maron BJ; American Heart Association Electrocardiography and Arrhythmias Committee of Council on Clinical Cardiology, Council on Cardiovascular Disease in Young, Council on Cardiovascular and Stroke Nursing, Council on Functional Genomics and Translational Biology, and American College of Cardiology. Eligibility and Disqualification Recommendations for Competitive Athletes With Cardiovascular Abnormalities: Task Force 13: Commotio Cordis: A Scientific Statement From the American Heart Association and American College of Cardiology. Circulation. 2015 Dec 1;132(22):e339-42. doi: 10.1161/CIR.0000000000000249. Epub 2015 Nov 2. PMID: 26621653.
22. Link MS, Maron BJ, Stickney RE, et al. Automated external defibrillator arrhythmia detection in a model of cardiac arrest due to commotio cordis. *J Cardiovasc Electrophysiol*. 2003;14:83–87. doi: 10.1046/j.1540-8167.2003.02017.x. PMID: 12625616.
23. Madias C, Maron BJ, Dau N, et al. Size as an important determinant of chest blow-induced commotio cordis. *Med Sci Sports Exerc*. 2018;50:1767–1771. doi: 10.1249/MSS.0000000000001630. PMID: 29620687.
24. Link MS, Maron BJ, Wang PJ, et al. Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. *Pediatrics*. 2002;109:873–877. doi: 10.1542/peds.109.5.873. PMID: 11986449.
25. Lateef F. Commotio cordis: an underappreciated cause of sudden death in athletes. Sports Med. 2000 Oct;30(4):301-8. doi: 10.2165/00007256-200030040-00005. PMID: 11048776.
26. Nesbitt AD, Cooper PJ, Kohl P. Rediscovering commotio cordis. Lancet. 2001 Apr 14;357(9263):1195-7. doi: 10.1016/S0140-6736(00)04338-5. PMID: 11323067.
27. Kalin J, Madias C, Alsheikh-Ali AA, et al. Reduced diameter spheres increases the risk of chest blow-induced ventricular fibrillation (commotio cordis). *Heart Rhythm*.

2011;8:1578–1581. doi: 10.1016/j.hrthm.2011.05.009. Epub 2011 May 17. PMID: 21699851.

28. Schlomka G, Hinrichs A. Experimentelle Untersuchungen uber den Einfluss stumpfer Brustkorbverletzungen auf das Elektrokardiogramm. Z Gesamte Exp Med 1932;31:43-61. https://doi.org/10.1007/BF02598668
29. Dickman GL, Hassan A, Luckstead EF. Ventricular fibrillation following baseball injury. Physician Sports Med 1978;(July):85-6. doi: 10.1080/00913847.1978.11948393. PMID: 29287513.
30. Meola F. La commozione toracica. *Gior Internaz Sci Med* 1879; 1:923–937.
31. Viano DC, Andrzejak DV, Polley TZ, King AI. Mechanism of fatal chest injury by baseball impact: development of an experimental model. Clin J Sport Med 1992;2:166-171