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Rediscovering commotio cordis

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> "Sudden death from relatively minor chest wall blows (commotio cordis) has been described in the medical literature since the late 1970s."[1]

This statement indicates a misconception about the history of commotio cordis, since the term and the concept had been established more than a century earlier.[2] In this paper, we outline current views on commotio cordis, illustrate how the concept of the disorder has developed since the 19th century, identify some forgotten lessons, and suggest a description of commotio cordis that takes into account both past and present insights.

Contemporary commotio cordis

Commotio cordis is currently understood to mean "instantaneous cardiac arrest [that] is produced by non-penetrating chest blows in the absence of heart disease or identifiable morphologic injury to the chest wall or heart".[3] Reports of the condition since the late 1970s are almost all from North America. Most describe accidental death of otherwise healthy children or adolescents after chest impact during recreational or competitive sport[4–8] or, less frequently, during physical combat[9] road-traffic accidents.[10]

Such fatalities receive extensive media coverage, provoke legal debate,[11,12] and stimulate research into public-health aspects (eg, the capacity of protective gear to avoid mechanically-induced cardiac arrest in sporting competitions, and the possibility of developing safer sporting equipment such as softer baseballs).

The current concept of commotio cordis emerged from a description of 70 cases[3] by Barry Maron and colleagues at the Minneapolis Heart Institute Foundation, MN, USA. Their report portrays commotio cordis as a rare but dangerous condition in which there is usually a poor response to resuscitatory measures. Most of those affected were young (mean age 12 years [SD 6]), male (all but one), and, at the time of accident, engaged in sports (>90%). The event leading to sudden death was a precordial impact, most commonly by projectiles such as baseballs,[4] softballs,[5] or pucks,[6] probably occurring during an electrically vulnerable phase of the cardiac cycle.[8] Contemporary experimental investigations into commotio cordis with anaesthetised pigs confirmed the existence of such a vulnerable period during early ventricular repolarisation[13,14] and showed the involvement of ATP-dependent potassium channels[15] in the electrophysiology.[16]

Lancet 2001; **357:** 1195–97

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Commotio cordis is seen as a little-understood, usually lethal, condition that mainly affects children and adolescents, and which had not previously been investigated in any detail.

Commotio cordis, pre-1940s

The term commotio cordis was in use in textbooks as early as 1857.[17] A review from 1896 shows that the term was applied to various forms (both lethal and non-lethal) of cardiovascular disorder caused by mechanical impact to the chest (both in the presence and absence of minor cardiac bruising).[18] Case reports published in the 1870s include an account by an Italian doctor, Felice Meola, of a patient who died instantaneously after receiving a blow from a thrown stone to the upper sternum,[19] and a description by a French surgeon, Auguste Nélaton, of the sudden cardiac death of a porter who hit the ground chest-on after a yoke he was pulling snapped.[20] A necropsy on the latter case did not show any structural damage, apart from minor bruises to the anterior chest wall.

By the turn of the 20th century, Germany was at the forefront of research in medicine and physiology. Strong trade unions and legislation on workplace safety and welfare of labour had created a demand for the assessment of claims under professional-accident compensation schemes, including the identification of causal links between precordial impact and cardiac malfunction in the absence of morphological damage.[21,22] Ferdinand Riedinger identified commotio cordis as an independent entity by systematically distinguishing between non-penetrating precordial impact in the presence (contusion) or absence (commotion) of cardiac bruising.[21,22] He considered that pure commotional pathologies were rare. Indeed, he had only witnessed two cases of "commotio thoracica"[21] (an early synonym for commotio cordis)[18,19] and suggested that the mechanisms involved were based on autonomic nervous responses.[21,22] With this concept, he followed the "profound vagal reflex" hypothesis of Meola, who experimentally investigated the condition in the 1870s.[19] Meola described the effects of chest impact on cardiac activity in rabbits, including sudden death, which he attributed to an autonomic reflex that arrested the heart.

Interrupted by World War I, the sustained legal demand for the assessment of commotio-cordis-related disorders triggered further research, mainly in Germany and in Switzerland.[23–30] The most comprehensive studies were done in the early 1930s by Georg Schlomka at Bonn University.[23] On the basis of analysis of electro-cardiograms, respiration, and arterial and central venous pressures in more than 800 experiments on anaesthetised rabbits, cats, and dogs, he identified three factors that determined the induction of arrhythmias by moderate precordial impact: type of impact, with swift impulse-like stimulation of a relatively small contact area being particularly arrhythmogenic; location of impact, where a correlation between impact site and type of arrhythmia (including ectopic beats, conduction block, tachycardia,

and fibrillation) was established; and force of impact, where moderate to large subcontusion forces were most arrhythmogenic. In studies with parasympathetic denervation of the heart, Schlomka disproved the vagal reflex theory. He suggested that dysrhythmias arise as a direct consequence of mechanical transmission of impact from the precordium to underlying cardiac tissues, and established the "vascular crisis" concept of mechanically-induced coronary vasospasms to explain commotio cordis.

European case reports and experimental studies did not portray commotio cordis as a necessarily lethal event.[18,21–25] The condition affected mainly adult manual labourers, although cases of sports-related commotio cordis were also reported.[30] Risk factors were identified and the utility of protective gear, such as boxing gloves, in avoiding commotio cordis was discussed.[23]

1940s–1970s

After World War II, studies of commotio cordis resumed slowly: it took about three decades for the total number of post-war publications to reach that of the 1930s alone. A fair proportion of such publications were in German accident or insurance-medicine journals, emphasising the driving force behind the studies.[31–35] Reduced research capacity in post-war Europe should not be seen as indicative of disregard of commotio cordis among practitioners. The diagnosis was used in cases where symptoms seemed disproportionate to the causal mechanical impact. These symptoms included chest pain (angina pectoris traumatica), dyspnoea, and altered consciousness, with irregular pulse and fluctuating blood pressure. Effects of precordial impact could be immediate or delayed, mild or serious, resolve spontaneously or persist, and give rise to (potentially fatal) post-commotional complications such as ventricular dilatation. Sudden cardiac arrest after a blow to the chest was thought to be a rare manifestation of the condition and referred to as "sudden cardiac death due to commotio cordis".[32] Further contributions included the description of commotio cordis in road-traffic accidents,[36,37] the particular susceptibility of children,[34] and first case reports on commotio cordis during soccer[35] and baseball.[4] This last paper is indicative of a shift in emphasis of commotio cordis studies between 1945 and the late 1970s, as their focus changed from adults to children and adolescents, from manual labour to sports activities, and from Europe to North America.

History and renaissance

Both early and contemporary research into commotio cordis appear to have been motivated by case reports of sudden death. The combination of public interest and legal implications seems to have been a crucial driving force. Experimental designs, too, are similar. Conclusions were and are based on mechanically induced changes in electrocardiograms and blood pressure in anaesthetised animals subjected to precordial impact. The risk factors identified by Schlomka in the 1930s (type, force, and location of impact) are still relevant, whereas the identification of a fourth factor (timing of impact) had to wait for technological advances.

An important difference between historical and contemporary concepts of commotio cordis lies in the use of the term itself. Currently, commotio cordis is taken to refer to mechanically induced sudden cardiac death, whereas the original term had a broader diagnostic scope, with death only one possible outcome. The contemporary concept does not necessarily represent an improvement over the historical view, since it fails to acknowledge that the same underlying mechanism—mechanoelectric feedback[16]—might be involved in a variety of clinically relevant manifestations.[37]

Why did the substantial historical body of work on commotio cordis disappear from the contemporary scientific knowledge base? Although reduced post-war research and the shift in contextualisation will have contributed, the most compelling reason is that the early work was published largely in German. This, together with the fact that electronic reference systems tend to retrieve few 19th and early 20th century publications, made access to the original European studies difficult. It is also possible that there was, after the war, resentment against any German heritage. Nonetheless, it is interesting to see that the term commotio cordis is still used to describe essentially the same condition as over a century ago.

In the contemporary age of rapid exchange of information and worldwide integration, language or cultural barriers should not be allowed to obscure access to the heritage of medical knowledge amassed in the past in the non-English speaking world. With respect to commotio cordis, it is remarkable to find many seemingly contemporary concepts documented in the earliest publications to which we have access. In an attempt to integrate historical and contemporary information, we propose the following definition for the condition.

> Commotio cordis: mechanical stimulation of the heart by non-penetrating, impulse-like impact to the precordium that, through intrinsic cardiac mechanisms, gives rise to disturbances of cardiac rhythm of varying type, duration, and severity, including sudden cardiac death, in the absence of structural damage that would explain any observed effects.

This study was supported by the British Heart Foundation and the UK Medical Research Council.

References

1 Link MS. Commotio cordis: sudden death due to chest wall impact in sports. *Heart* 1999; **81:** 109–10.
2 Kohl P. Commotio cordis: early observations. *Heart* 1999; **82:** 397.
3 Maron BJ, Link MS, Wang PJ, Estes NAM III. Clinical profile of commotio cordis: an under appreciated cause of sudden death in the young during sports and other activities. *J Cardiovasc Electrophysiol* 1999; **10:** 114–20.
4 Dickman GL, Hassan A, Luckstead EF. Ventricular fibrillation following baseball injury. *Phys Sportsmed* 1978; **6:** 85–86.
5 Green ED, Simson LR, Kellerman HH, Horowitz RN, Sturner WQ. Cardiac concussion following softball blow to the chest. *Ann Emerg Med* 1980; **9:** 155–57.
6 Kaplan JA, Karofsky PS, Volturo GA. Commotio cordis in two amateur ice hockey players despite the use of commercial chest protectors: case report. *J Trauma* 1993; **34:** 151–53.
7 Edlich RFJ, Mayer NE, Fariss BL, et al. Commotio cordis in a lacrosse goalie. *J Emerg Med* 1987; **5:** 181–84.
8 Maron BJ, Poliac LC, Kaplan JA, Mueller FO. Blunt impact to the chest leading to a sudden death from cardiac arrest during sports activities. *N Engl J Med* 1995; **333:** 337–42.
9 Frazer MM, Mirchandani H. Commotio cordis, revisited. *Am J Forensic Med Pathol* 1984; **5:** 249–51.
10 Michalodimitrakis EN, Tsatsakis AM. Vehicular accidents and cardiac concussion: a traumatic connection. *Am J Forensic Med Pathol* 1997; **18:** 282–84.
11 Selvin P. Man is sentenced in son's death. *Washington Post*, May 30, 1998, B08.
12 Froede RC, Lindsey D, Steinbronn K. Sudden unexpected death from cardiac concussion (commotio cordis) with unusual legal complications. *J Forensic Sci* 1979; **24:** 752–56.
13 Link MS, Wang PJ, Pandin NG, et al. An experimental model of sudden cardiac death due to low-energy chest-wall impact (commotio cordis). *N Engl J Med* 1998; **338:** 1805–11.
14 Cooper GJ, Pearce BP, Stainer MC, Maynard RL. The biomechanical response of the thorax to nonpenetrating impact with particular

reference to cardiac injuries. *J Trauma* 1982; **22:** 994–1008.
15 Link MS, Wang PJ, VanderBrink BA, et al. 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:** 413–18.
16 Kohl P, Nesbitt AD, Cooper PJ, Lei M. Sudden cardiac death by *Commotio cordis*: role of mechano-electric feedback. *Cardiovasc Res* 2001; **50:** 280–89.
17 Casper JL. Practisches Handbuch der gerichtlichen Medicin, 1st edn. Berlin: Verlag von August Hirschwald, 1857.
18 Bernstein R. Ueber die durch Contusion und Erschütterung entstehenden Krankenheiten des Herzens. *Z Klin Med* 1896; **29:** 519–55.
19 Meola F. La commozione toracica. *G Int Sci Med* 1879; **1:** 923–37.
20 Nélaton A. Elements de pathologie chirurgicale, 2nd edn. Paris: Librairie Germer Bateliere, 1876.
21 Riedinger F, Kümmell H. Die Verletzungen und Erkrankungen des Thorax und seines Inhaltes. In: von Bergman E, von Bruns P, eds. Handbuch der Praktischen Chirurgie, 2nd edn. Stuttgart: Enke, 1903: 373–456.
22 Riedinger F. Über Brusterschütterung. Festschrift zur dritten Saecularfeier der Alma Julia Maximiliana Leipzig. Leipzig: Verlag von F C W Vogel, 1882: 221–34.
23 Schlomka G. Commotio Cordis und ihre Folgen. Die Einwirkung stumpfer Brustwandtraumen auf das Herz. *Ergebn Inn Med Kinderheilkd* 1934; **47:** 1–91.
24 Glatzel H. Spätfolgen nach Commotio cordis. *Dtsch Med Wochenschr* 1936; **10:** 377–79.
25 Nordmann M. Zur Praxis und Theorie der Commotio cordis. *Z Kreislaufforschung* 1942; **11:** 361–80.
26 Jacobi. Zur Commotio cordis. *Arch Orthopäd Unfall Chir* 1937; **42:** 418–21.
27 Brunner W. Zur Commotio cordis. *Zb Chirurgie* 1939; **39:** 2145–48.
28 Hadorn W. Ueber Commotio cordis. *Schweiz Z Unfallheilkd* 1940; **1:** 156–64, 227.
29 Spühler O. Zur Frage der Commotio cordis. *Schweiz Med Wochenschr* 1937; **26:** 571–76.
30 Deutsch F. Sekundenherztod im Boxkampf durch Commotio cordis. *Wien Arch Intern Med* 1930; **20:** 279–86.
31 Greiner H. Ein kasuistischer Beitrag zur Commotio cordis bei Hypertrophie des Herzens. *Monatsschr Unfalheilkd* 1953; **56:** 90–92.
32 Albers A, Isfort A. Zur Prognose der Commotio cordis. *Lebensversicherungsmedizin* 1966; **18:** 9–11.
33 Walther G. Der plötzliche Tod beim Fussballsport—Commotio cordis bei besonderen Verhältnissen am Herzen. *Lebensversicherungsmedizin* 1968; **20:** 136–40.
34 Isfort A. Commotio cordis beim Kind. *Monatsschr Unfallheilkd Versicher Versorg Verkehrsmed* 1969; **72:** 409–16.
35 Wirth E. Stumpfe Brustkorbverletzungen tödlich verletzer Pkw-Insassen nach Frontalzusammenstößen unter besonderer Berücksichtigung der klinischen und gerichtsärztlichen Diagnostik der Commotio cordis. *Beitr Gerichtl Med* 1973; **30:** 458–64.
36 Gögler E. Unfallopfer im Straßenverkehr. Basel: Geigy, 1962: 51.
37 Kohl P, Hunter P, Noble D. Stretch-induced changes in heart rate and rhythm: clinical observations, experiments and mathematical models. *Prog Biophys Mol Biol* 1999; **71:** 91–138.

Uses of error: Addison's disease in pregnancy

A 33-year-old primigravida presented with severe hyperemesis in the eleventh week of pregnancy. Family history revealed that all surviving females on the maternal side had a diagnosis of autoimmune thyroid dysfunction and/or systemic lupus erythematosis. On examination she was thin, pigmented, and dehydrated. Biochemistry revealed hyponatraemia, with a marked metabolic acidosis, but normal plasma potassium.

The fetal heart beat was detected on admission but was found to be absent on day 5. Following rehydration with intravenous normal saline, an evacuation of retained products was performed. Vomiting improved following this, but did not fully resolve. Hyponatraemia persisted despite five litres per day of intravenous normal saline. Plasma cortisol was <50 mmol/L and there was no increase after 250 ug of intravenous tetracosactide (Synacthen). Adrenal autoantibodies were raised at 13 558 IU/mL (normal <50) and a diagnosis of autoimmune Addison's disease was made.

Autoimmune Addison's disease is the commonest cause of adrenal insufficiency in the UK. The clinical features and biochemistry in this case are certainly consistent with adrenal insufficiency, even with normal plasma potassium, as hyperkalaemia is not essential for the diagnosis. Diagnosis was delayed due to the rarity of Addison's disease first presenting during pregnancy, and the severe vomiting suggestive of hyperemesis.

However, the diagnosis of Addison's disease is often delayed because of the non-specific way in which it may manifest. In any case, such severe acidosis is an indication of marked metabolic disturbance and should evoke the possibility of adrenal insufficiency. Delayed diagnosis of Addison's disease has a significant morbidity and mortality and should remain high on the list of differential diagnoses in any patient with a significantly deranged biochemical profile.

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