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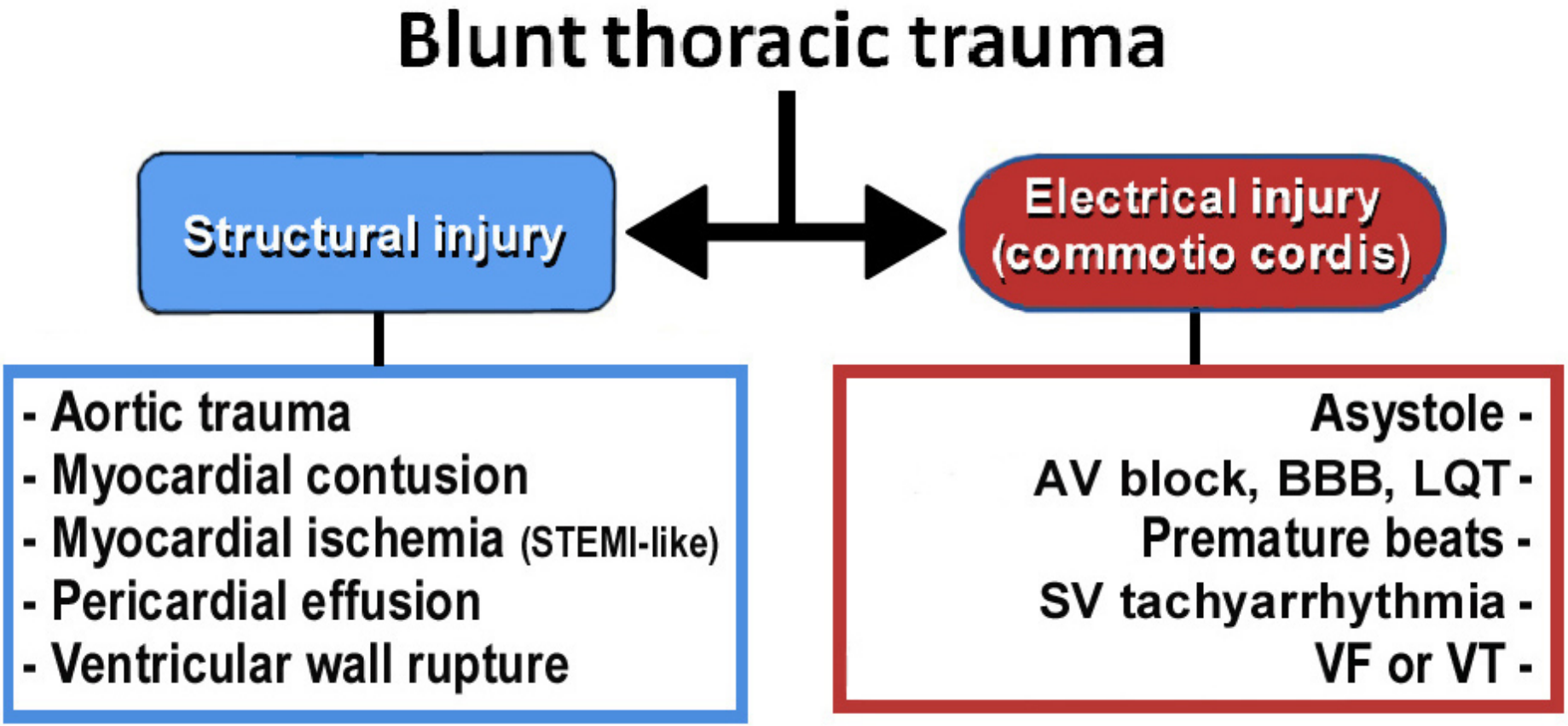

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REVIEW

EXERCISE AND SPORT CARDIOLOGY

Blunt thoracic trauma and cardiac injury in the athlete: contemporary management

Cesare DE GREGORIO [1] *, Ludovico MAGAUDDA [2]

[1]Department of Clinical and Experimental Medicine, University Hospital of Messina, Messina, Italy; [2]Department of Biomorphology and Functional Imaging, Postgraduate University School of Sports Medicine, University Hospital of Messina, Messina, Italy

*Corresponding author: Cesare De Gregorio, Department of Clinical and Experimental Medicine, University Hospital of Messina, Via Consolare Valeria, 98125 Messina, Italy. E-mail: cdegregorio@unime.it

ABSTRACT

Commotio cordis and cardiac injuries are rare events usually following a chest blunt traumas during sports activities. Various etiologies have been identified to cause electrical (commotio cordis) and/or structural (contusion and further injuries) damage, but high-velocity tools such as baseballs or hockey pucks (also called projectiles) have been chiefly identified. Clinical consequences are challenging, varying from uncomplicated supraventricular arrhythmias to cardiac wall rupture. Ventricular fibrillation is the most remarkable outcome leading to cardiac arrest in some individuals. In this article, up-to-date epidemiological and pathophysiological issues are discussed, along with the most suitable assistance protocols of the injured athlete in the sports arena. Current knowledge about traumatic sports injuries and ensuing cardiovascular sequelae made significant steps forwards than in the past. The majority of athletes (especially the youngest ones) wearing chest protectors are usually preserved from serious outcomes and sudden cardiac death, but further technical effort is encouraged to attain more satisfactory barriers against projectile's impact. Educational campaigns among students, closer team surveillance, implementation of the sports arenas with adequate rescue devices and medical assistance remain mandatory items in contact sports activity.

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Key words: Athletes - Wounds and injuries - Myocardial contusions - Commotio cordis - Prognosis - Sports.

Commotio cordis (CC) is a clinical picture characterized by cardiac functional injury following severe, usually blunt (nonpenetrating), trauma onto the chest wall (sternal region) during sports activities. This condition can generate arrhythmic disorders, at times fatal, usually in the absence of previously known heart or vessel disease. Though general information available from the USA CC Registry, the true incidence in the sports arena has not been established yet. Commotio cordis should be differentiated from *cardiac contusion*, effusion or rupture, which are structural diseases going on within 24 hours from the trauma (Figure 1).[1-5]

Using rigorous methodology, prospective studies have reported a sudden cardiac arrest rate of 1:69,000

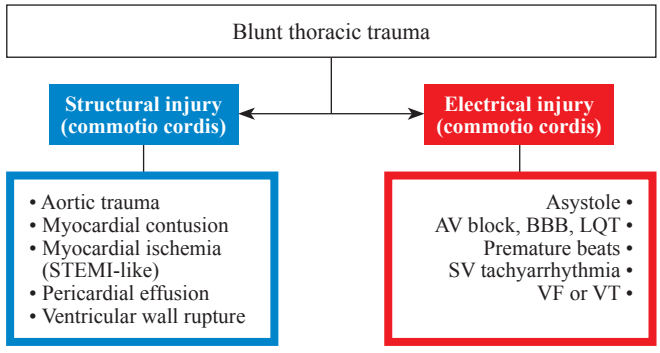

Figure 1.—Blunt cardiac injuries during sports activities.
AV: atrio-ventricular; BBB: bundle branch block; LQT: QT interval prolongation; STEMI: ST elevation myocardial infarction; SV: supraventricular; VF: ventricular fibrillation; VT: ventricular tachycardia.

in 14 to 24-year-old and 1:22,000 in 25 to 35-year-old athletes, but only in a minority of cases a chest trauma was the causal event.[3-6]

Current science is devoted to minimize the risk of fatal arrhythmias, like ventricular tachycardia (VT) or fibrillation (VF), and myocardial injuries as well, which can occur as the result of high-velocity impact of stiff objects against the chest wall.[2, 4, 7-9]

However, special conditions are needed for traumas to cause cardiac events. In particular, the crash is potentially harmful when impacting the anterior chest wall at high velocity rate and within a short electrical interval on ECG, called "vulnerable period", which has been identified 10 to 40 ms prior to the nadir of the T-wave (Figure 2).[10]

Apart from an acute arhythmogenic outcome, myocardial contusion and infarct-like ST segment elevation have been described as consequences of chest traumas, potentially leading to asystole. Of note, athletes ≤40 years of age are more likely to get CC, even if fatal outcomes have been reported only in a minority of them (Figure 3).[4, 5] A thoracic shock is responsible approximately for 25% of post-traumatic deaths and 25% of injuries, which are not so exceptional in the real world. The most likely pathophysiological hypothesis is the weakness of the chest wall, but the occurrence of CC also depends on the sports discipline. Baseballs, softball, hockey on ice, American football, lacrosse are the most shocking sports activities.[1-10]

CC usually occurs at sports arenas, where survival of victims largely depends on the quick rescue team assistance and the availability of automated external defibrillators (AED) on site.[11] Fatal arrhythmias can also be triggered by strong chest traumas during myocardial repolarization, but even a small blow may lead to VF when crashing within the vulnerable period. A prompt recognition of the VF is a crucial lifesaving step. Rapid defibrillation with immediate BLS/ACLS maneuvers are mandatory in these (frequently young) victims.[3-5, 11, 12]

Arrhythmic outcomes

In order to explain both triggers and mechanisms of cardiac arrhythmias following a chest trauma, knowledge over the last decade has been gained from laboratory models. High-speed nonpenetrating impact of dense objects (usually called projectiles) against the (anterior) chest wall, *e.g.* baseballs, hockey pucks, javelins, handlebars, steering wheels, but also the body thrown against an edge, may cause paroxysmal arrhythmias and CC. Air-inflated balloons (soccer, basketball, handball) have been seldom described as potential causes of fatal arrhythmias when compared to baseball and ice-hockey pucks (highly-density tools) or American football (shaped and hard-covered balloons) (Figure 4).[13] Although rare, horse kicks, ski and martial-arts related injuries, head shots onto the chest wall (famous that one from a French to an Italian soccer player in 2006) may determine a blunt cardiac trauma.[2-9]

In bat-and-ball games like baseball, the ball thrown by the pitcher at a velocity rate of 40 to 70 Kmph is likely to determine VF if the impact against the anterior chest wall occurs within the vulnerable R-R interval on

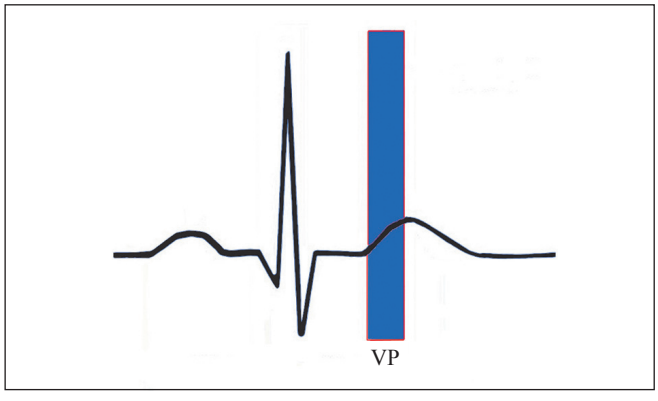

Figure 2.—The vulnerable triggering period (VP) for ventricular arrhythmias on chest trauma (adapted from Talving *et al.*).[10]

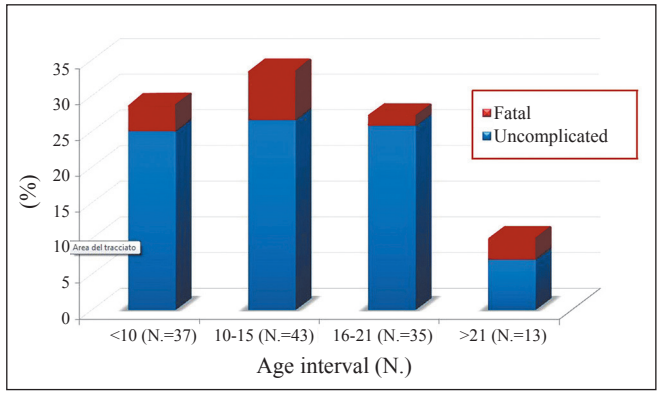

Figure 3.—Cardiac outcomes after blunt trauma (adapted from Maron *et al.*).[4, 5]

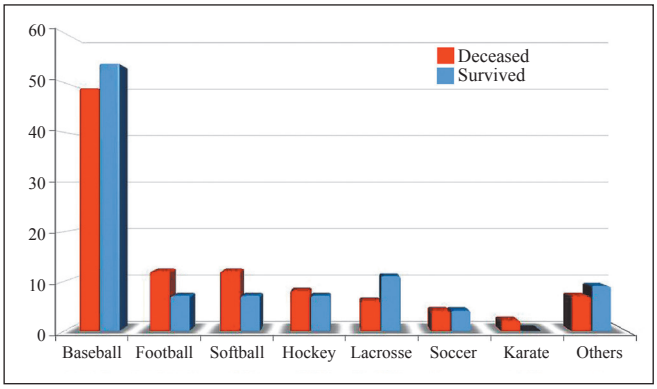

Figure 4.—Statistics of non-survivors and survivors form the US CC Registry (adapted from Maron *et al.*).[13]

ECG. When launched at a higher speed, such projectiles may cause myocardial contusion, ventricular wall rupture, pericardial fluid or isthmic aortic rupture.[2, 5-12]

In the majority of cases, post-traumatic arrhythmias are caused by a rapid rising of intraventricular pressure up to 450 mmHg, with ensuing acute myocardial fiber stretching and abnormal potassium and calcium ion currents.[5-8]

At times, low-energy projectiles provoke abnormal potassium ATP channel inflow possibly leading to arrhythmogenic outcomes.[14] On the contrary, the hypothesis that L-type calcium ($I_{Ca,L}$) channels might be activated by fiber stretching with abnormal calcium ion influx into the cell has been recently argued by Madias *et al.*[15] These authors demonstrated that infusion of verapamil did not modify the frequency of VF induction in their CC experimental models, disproving a strong arrhythmogenic role for such channels. Verapamil, however, reduced the amount of ST-segment elevation and the frequency of bundle branch block following chest impact.

Of note, because of the fact that I_{ca++} overload is the underlying mechanism of catecholaminergic polymorphic VT in young individuals, a pathogenetic role cannot be ruled out in the hurt athlete.[15, 16]

Traumatic injuries

In 1958 a landmark study by Parmley *et al.*[7] based on autopsy findings revealed a range of myocardial tissue damage following thoracic traumas, ranging from a "simple" wall edema or hematoma (contusion) to more severe myocardial or pericardial necrosis and hemorrhage. In general, such lesions are consistent with external wall wound or drilling, in some cases leading to pericardial tamponade or rupture of the ventricular septum.[17-19]

Due to its thinner wall, the right ventricle is the most susceptible chamber to traumatic injuries. However, the outcome's rate is less clinically relevant than in the left ventricular wall. The mid-apical segment of interventricular septum is the most frequent site of myocardial wall rupture, often occurring hours or days after the trauma.[19, 20]

In past times, when chest protectors were less consistent than now, approximately 50% of individuals experiencing chest traumas were getting more than a single chamber harmed, often together with the mitral valve apparatus.[6, 10, 17, 18]

Nowadays, cardiac wall injury is much rarer than in the past, but slim teenager athletes (usually <12 years old), cyclists or riders, keep a high risk anyway. Especially when presenting to the emergency room with a multi-trauma, rib fracture and/or hemothorax, a cardiac trauma should be ruled out.[18-20]

More recently, myocardial ischemic-like pattern (edema and delay enhancement) has been found at cardiac magnetic resonance imaging approximately in 5% of individuals presenting with a blunt thoracic thrauma.[21, 22]

The full list and classification of thoracic traumas are presented in the position papers and guidelines from the American Association for Surgery and Trauma Organ Injury.[23]

On-field management of blunt cardiac traumas

Clinical outcomes in the athlete with blunt thoracic trauma are linked to immediate medical or paramedical assistance. Two potential scenarios along with their basic management are summarized in Figure 5. In general, chest blows without loss of consciousness indicate a benign clinical picture, usually free of life-threatening arrhythmias, although premature beats and supraventricular arrhythmias are even possible.

However, a structural damage like myocardial contusion, hematoma or pericardial effusion may occur over the next few hours. These latter subjects, usually presenting with normal heart rate and blood pressure soon after the trauma, should undergo standard 12-lead ECG, echocardiogram and laboratory sample (especially troponin I or T, creatin-kinase and myoglobin) within 48

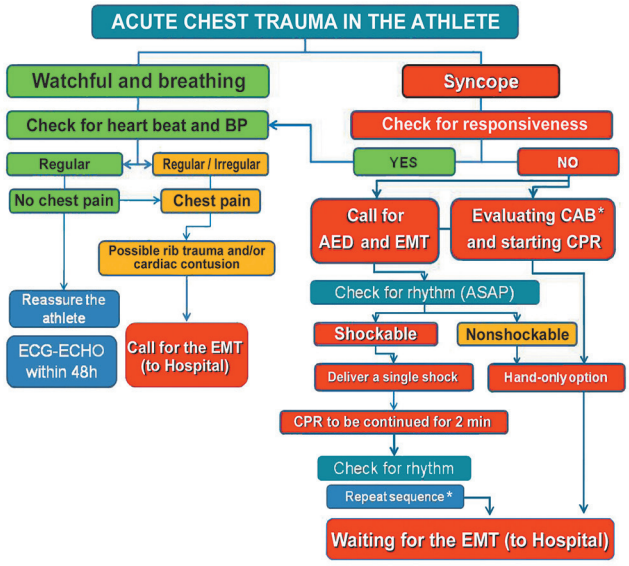

Figure 5.—Proposal of a simplified flow-chart for on-field assistance to hurt athletes.

hours from the event, in order to rule out possible cardiac injuries.

On the contrary, on-field occurrence of syncope soon after a thoracic trauma needs to deliver BLS approach shortly. Accessibility to an AED is a bystander priority, especially when the Emergency Medical Service is not readily available.[11, 12] Unfortunately, as from the 2010 Arena study,[11] only 72% of the participant Soccer Clubs reported the presence of an AED in their arenas and only 64% of them had definite safety programs. In this regard, a recent Italian Health-care regulation (Decreto Legge n.189, 16 October 2016) is going on entailing professional sports arenas with precise safety procedures, included on-field AED and skilled medical assistance.

In fact, the use of AED and chest wall protection have led to a significant decrease in the number of CC victims in the USA Registry.[13, 24]

Chest barriers

Based on the U.S. CC Registry, approximately one-fourth of competitive athletes is likely to have arrhythmic disorders in the sports arena. African American athletes are up to 8 times more likely to get CC compared to Caucasians.[2-5] Of note, 45% of the victims in the U.S. Registry were engaged in recreational sporting and/or daily activities.[22, 23]

At present time, however, these events are less frequent than in the past, even if clinical outcomes may be crushing. Opportunely, prevention from traumatic injuries has been such a mandatory item in high-risk sports disciplines. Chest protectors have been implemented with new materials leading to approximately two-third risk reduction by wearing such devices.[24-26]

In a recent experimental study,[27] several combinations of protectors have been tested. The combination of Accelleron (high density elastomer) foam, Airilon (low density elastomer) foam, TriDur (flexible elastomer coated aramid), and ImpacShield (polypropylene polymer) was found to reduce the incidence of VF as from 54% to 5%, whereas other materials were less or not effective. Although this study did not exclude other materials or combinations to be satisfactorily safe, it replicated inadequacies of some commercially available chest protectors while tested in experimental models reproducing the human scenario. Recent chest protectors, released from 2010 and tested under the same laboratory conditions, demonstrated a VF rate as by 40-56% with ball strikes.[27, 28]

Treatment, prognosis and return to play

The increased knowledge about CC pathophysiology and the use of chest protectors allowed more victims to be preserved from acute traumas.[13]

However, reentrant VT can occur on a different basis, often occurring on the chronic stage long after a blunt chest injury, likely due to underlying structural or functional heart disease (arrhythmogenic tissue, myocardial fibrosis, QT segment prolongation, etc.), often misdiagnosed. These latter clinical pictures usually require specific electrophysiological screening, antiarrhythmic drug use and/or catheter ablation. The need for an implantable cardioverter defibrillator (ICD) should be established by the heart team based on inherent algorithms.[16, 18, 29]

Victims experiencing CC must be acknowledged in order to rule out any structural heart disease. Diagnostic workup includes, but is not restricted to, ECG and echocardiography. In some cases, ambulatory ECG monitoring, exercise stress or cardiopulmonary testing should be performed, also recommending cardiac magnetic

resonance imaging in selected cases. Pharmacological screening and accurate follow-up must be performed in carriers of a Brugada pattern or prolonged QT syndrome, even if no typical electrocardiographic features are present.[30] In those instances, as the long-QT syndrome is a persistent concern, genetic testing may also be considered. Likewise, T-wave anomalies should be carefully interpreted in young athletes eligible to competitive sports, based on the most recent international recommendations. In those individuals, qualification to competitive sports is not possible without ruling out any cardiac disease.[25, 28, 29, 31]

Once structural or functional diseases have been ruled out, ICD is not recommended to CC survivors. Return-to-play decision is strictly dependent on the persistence of arrhythmogenic triggers. Considering the numbers of determinants to merge for replicating a chest trauma, healthy athletes are unlikely to get one more CC. However, especially in punier and younger subjects, it would be prudent to avoid impact sports activities. Strengthening of the chest wall with age reduces the risk of its recurrence.[5, 6]

Maron *et al.*[13] reported a complete physical recovery and return to sports practice in 70% of athletes with recent CC. The remaining 30% presented with clinical sequelae, often of psychological or neurological nature. In some cases, a residual left ventricular dysfunction was observed late after cardiac contusion. This latter peculiar condition restricts from competitive sports in the short term, but it does not prevent from cardiac rehabilitation and reassessment.

A different approach is required for other cardiac injuries. Clinical concerns arise in seriously damaged athletes, such as with aortic rupture, LV wall perforation, chest wall perforation or cardiac tamponade. They need to be addressed to a Cardio-thorax Surgical Center.[17-20]

Wounds can be limited to myocardial contusion, with or without pericardial effusion, and usually require hospitalization if cardiac enzymes (troponin-I, creatin-kinase, myoglobin) increase in the next few hours, as well as ventricular dysfunction is recognized at echocardiography. Arrhythmias can occur all through that period and full functional recovery usually needs 15 to 30 days to be accomplished. Cardiac rehabilitation should be promoted in the absence of arrhythmic risk, but return-to-play implies impact-less sporting activities.[20-23]

In any cases, pronouncement about the return-to-play in difficult athletes should be delivered by a "sports-med team" (cardiologist, sports physician, pulmonologist, neurologist, psychologist and generalist) in agreement with their trainer(s) or coach, taking into account the athlete's safety above business interest.[32]

Conclusions

Current knowledge about blunt chest trauma injuries during sports activities allows the athlete to be preserved from life-threatening outcomes, far better than in the past. Modern chest protectors almost are effective, but further technical improvement in order to minimize their residual covering failure is still required.

Prompt and skilled assistance in the sports arena, strengthened by the use of an AED, remain mandatory items for athletes and amateurs, as well as educational campaigns among students, closer team surveillance and safety implementation.

In some case, the hurt athlete may represent a forensic matter, due to psychological troubles like fear or panic in resuming its usual sports activity.

In the lack of underlying structural or functional disease, athletes surviving a CC or cardiac contusion are unlikely to experience further events. However, return to play should be established on a case by case basis by a composite medical board.

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