



# Blunt cardiac injury associated with cardiac valve insufficiency: Trauma links to chronic disease?

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## KEYWORDS

Trauma;  
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## Summary

**Context:** Cardiac injury has been well recognised as a complication of blunt chest trauma. Its clinical spectrum ranges from blunt cardiac injury (BCI) to complete rupture of cardiac tissues, with cardiac valvular injury often being overlooked.

**Objective:** To determine whether hospitalised BCI is associated with increased risk of cardiac valve insufficiency in a large multi-state hospitalised population.

**Methods:** Cases with BCI and cardiac valve insufficiency were identified based on discharge diagnoses in 1997 statewide hospital discharge data from 19 states. Four valvular outcomes were studied: (1) mitral valve insufficiency, incompetence, regurgitation (MVIIR); (2) aortic valve insufficiency, incompetence, regurgitation, stenosis (AVIIRS); (3) tricuspid valve insufficiency, incompetence, regurgitation, stenosis (TVIIRS); and (4) pulmonary valve insufficiency, incompetence, regurgitation, stenosis (PVIIRS).

**Results:** Among 1,051,081 injury discharges, 2709 (0.26%) people had BCI; 13,087 (1.25%) had MVIIR; 9811 (0.93%) had AVIIRS; 1338 (0.13%) had TVIIRS; 178 (0.02%) had PVIIRS. Independent of potential confounding factors, discharge for BCI was associated with a 12-fold increased risk for TVIIRS and a 3.4-fold increased risk for AVIIRS.

**Conclusion:** Cardiac valve insufficiency has been well recognised as an important risk factor for congestive heart failure. With the findings that BCI is associated with an

**Abbreviations:** AVIIRS, aortic valve incompetence, insufficiency, regurgitation, stenosis; BCI, blunt cardiac injury; MAISS, maximum abbreviated injury severity score; MVIIR, mitral valve incompetence, insufficiency, regurgitation; MVT, motor vehicle transportation; N/A, not applicable; PVIIRS, pulmonary valve incompetence, insufficiency, regurgitation, stenosis; S.D., standard deviation; TVIIRS, tricuspid valve incompetence, insufficiency, regurgitation, stenosis

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increased risk of specific valvular disorders, it is possible that trauma may play an important and heretofore largely unrecognised role in a portion of the burden of cardiovascular morbidity and mortality.

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## Introduction

Blunt thoracic trauma may result from motor vehicle crashes, sporting activity, assaults, and falls. In the United States, thoracic trauma accounts for at least 25% of all trauma deaths<sup>29</sup>. Blunt cardiac injury (formerly known as “myocardial contusion”), following blunt thoracic trauma has been described as the most common yet unsuspected visceral injury causing death in injured victims.<sup>6,46</sup>

The clinical significance of BCI remains unknown.<sup>33</sup> In those patients with cardiac trauma who reach the hospital alive, however, the greatest contributor to fatality was found to be BCI<sup>54</sup> and there is research that this is due to incomplete diagnosis and assessment.<sup>43</sup> For instance, life-threatening ventricular arrhythmias as a complication of BCI were described in up to 16% of patients with blunt thoracic trauma.<sup>55</sup>

BCI can be characterised by patchy areas of muscle necrosis and haemorrhagic infiltrate, rupture of small vessels, haemorrhage into the interstitium and around the muscle fibres.<sup>12,36,45,46</sup> Such patchy and irregular myocardial cell necrosis can be well recognised at surgery or autopsy but not with conventional imaging studies.<sup>46</sup> The magnitude of BCI itself may vary from absence of any clinical symptoms to complete rupture of cardiac tissues with sudden death.<sup>3,46,55–57</sup> Patients with BCI might sustain certain intracardiac lesions that are in the middle of this spectrum, but still compatible with survival.

Currently, the primary clinical significance of BCI lies in its effects on the heart itself and the great vessels, with cardiac valvular injury, which has been recognised as a common clinical problem, often being overlooked.<sup>43</sup> Specifically, aortic and mitral valve damage has been recognised as a complication of non-penetrating chest injury.<sup>41,42</sup> Most patients with traumatic rupture of the aortic valve suffer it immediately after the initial trauma; however, “delayed” rupture may occur after BCI.<sup>43</sup> Traumatic tricuspid insufficiency is a rarer lesion, although, its frequency is thought to be underestimated.<sup>19</sup>

The objective of this research is to explore whether BCI is associated with increased risk of cardiac valve insufficiency. We hypothesise that BCI is significantly associated with insufficiency of certain cardiac valves. To examine this issue, we conducted a

cross-sectional analysis of the association between BCI and cardiac valve insufficiency based on a large database of all hospital discharges from 19 states during a one-year period.

## Methods

Hospital discharge data were collected from 19 states (Arizona, California, Florida, Massachusetts, Maryland, Maine, Michigan, Nebraska, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, South Carolina, Utah, Virginia, Vermont, Washington, and Wisconsin). Information was obtained for all trauma admissions in 1997 based on ICD-9-CM classification (codes 800–959). The combined 19-state dataset had 1.049 million injury cases, of which 0.967 million were acute. These were drawn from a total of about 17.8 million hospitalisations. This dataset has fields for 15 diagnoses and 6 E-codes, however, the number of diagnosis fields provided by the states ranged from 7 (Rhode Island) to 25 (California).

Extensive filtering and editing were applied to the data.<sup>58</sup> Specific algorithms were used to exclude cases of non-injury.<sup>58</sup>

Diagnosis codes representing injuries (codes 800–959) were categorised into subgroups based on injury mechanism and intent. The mechanism of injury and its associated intent were classified on the basis of the external cause of injury codes, defined within ICD-9-CM.

MVIIR was identified based on ICD-9-CM (424.0), that excludes mitral valve disease (394.9), mitral valve failure (394.9) and mitral valve stenosis (394.0). It also excludes the following conditions: specified as rheumatic (394.1), unspecified as to cause but with mention of diseases of aortic valve (396.0–396.9), and mitral stenosis or obstruction (394.2). AVIIRS was identified based on ICD-9-CM (424.1), that excludes hypertrophic subaortic stenosis (425.1), rheumatic (395.0–395.9) and that of unspecified cause but with mention of diseases of mitral valve (396.0–396.9). TVIIRS was identified based on ICD-9-CM (424.2), that excludes rheumatic tricuspid valve incompetence, insufficiency, regurgitation and stenosis or of unspecified cause (397.0). PVIIRS was identified based on ICD-9-CM (424.3). This diagnosis excludes rheumatic

pulmonary valve incompetence, insufficiency, regurgitation and stenosis (397.1). Bacterial or syphilitic valve insufficiency were excluded since “other diseases of endocardium” (ICD-9 code 424) excludes bacterial endocarditis (421.0–421.9), rheumatic endocarditis (391.1, 394.0–397.9) and syphilitic endocarditis (093.20–093.24). BCI was identified based on ICD-9-CM (861.01).

Age has been shown to significantly influence the prevalence of valve regurgitation.<sup>51</sup> Singh et al.<sup>51</sup> found that the prevalence of mitral regurgitation, tricuspid regurgitation, and aortic regurgitation increased with age and are consistent with earlier Doppler studies.<sup>2,8,32</sup> Although left-sided valves (aortic and mitral) are exposed to higher pressures and are likely to undergo degenerative changes earlier than right-sided valves, prevalence rates of mitral and tricuspid regurgitation have been shown to be comparable at all ages.<sup>32</sup>

Some degenerative conditions (myxomatous degeneration and mitral annulus calcification) have been shown to cause MVIIR and AVIIRS<sup>53</sup>, however these conditions do not have ICD-9-CM codes. Since age was shown to dramatically influence degenerative conditions,<sup>53</sup> age was an adjustment factor in all regression analyses.

Ischaemic heart disease has been shown to cause MVIIR and AVIIRS.<sup>28</sup> Therefore, ischaemic heart disease was identified based on ICD-9-CM (410–414) and was further entered as a covariate in regression analysis for MVIIR and AVIIRS.

Population estimates for states by age, race, sex were identified from “Population Estimates” Program, Population Division, U.S. Census Bureau, Washington, DC 20233, available at: <http://www.census.gov/population/estimates/state/sasrh/sasrh97.txt> Statistical analysis was conducted using SPSS 11 for Windows (SPSS, Inc., Chicago, IL). Univariate analysis was performed with Chi-square

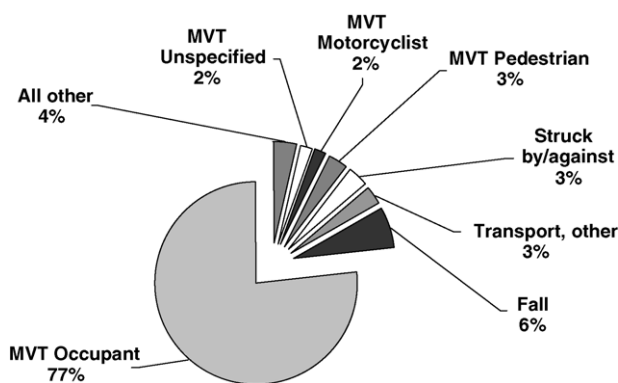
testing and Fisher exact testing for certain categorical variables (BCI, MVIIR, AVIIRS, TVIIRS, PVIIRS).

All analyses were adjusted for age (10 year age strata), gender (males versus females), race (whites versus non-whites), presence of BCI (yes versus no), maximum abbreviated injury severity score (MAISS) (6 levels, the lowest being “0” and the highest being “6”) and sources of payment (Medicare, Medicaid, worker’s compensation, other government, commercial, HMO, self-pay, charity, other) (Table 2). Multivariate logistic regression was done using the default method (enter mode) of entering covariates.

## Results

Among 1,051,081 injury discharges, 2709 (0.26%) had BCI; 13,087 (1.25%) had MVIIR; 9811 (0.93%) had AVIIRS; 1338 (0.13%) had TVIIRS and 178 (0.02%) had PVIIRS. Injuries to motor-vehicle occupant were the leading mechanism of injury for BCI (77%), followed by falls (6%) (see Fig. 1). Transportation-related injuries combined accounted for 87% of the attributed mechanisms for BCI. There were slightly fewer females with BCI (43.9%); however, there were more females with either type of valvular disorder (73.9% for MVIIR, 70.3% for AVIIRS, 69.4% for TVIIRS and 64.8% for PVIIRS) (see Table 1). The overall rate (per 100,000 persons) for BCI, MVIIR, AVIIRS, TVIIRS, and PVIIRS was respectively 1.94, 9.37, 7.02, 0.96, 0.13 (see Figs. 2 and 3).

In the univariate analysis, BCI was significantly associated with MVIIR ( $P < 0.001$ ), AVIIRS ( $P < 0.001$ ), and TVIIRS ( $P < 0.001$ ); and was not significantly associated with PVIIRS ( $P > 0.05$ ). In the regression analysis, BCI was found to be a significant predictor for AVIIRS and for TVIIRS after



**Figure 1** Mechanism of blunt cardiac injury (mechanisms that cause BCI in less than 1% cases were combined into “all other” category). MVT, motor vehicle transportation.

**Table 1** Patient characteristics

Patient characteristics	BCI	MVIIR	AVIIRS	TVIIRS	PVIIRS
Age mean (S.D.)	54.6 (22.2)	71.7 (18.6)	82.1 (10.9)	78.1 (13.9)	74.7 (19.7)
Females (%)	1190 (43.9)	9672 (73.9)	6893 (70.3)	929 (69.4)	114 (64.8)
Nonwhite (%)	441 (16.3)	888 (6.8)	554 (5.6)	144 (10.8)	17 (9.7)
MAISS median	3	2	3	2	2

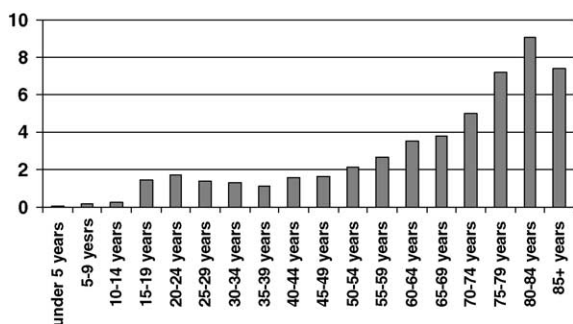
AVIIRS, aortic valve incompetence, insufficiency, regurgitation, stenosis; BCI, blunt cardiac injury; MAISS, maximum abbreviated injury severity score; MVIIR, mitral valve incompetence, insufficiency, regurgitation; PVIIRS, pulmonary valve incompetence, insufficiency, regurgitation, stenosis; S.D., standard deviation; TVIIRS, tricuspid valve incompetence, insufficiency, regurgitation, stenosis.

adjustment for potential confounders (see Table 2). BCI was not found to be a significant predictor for MVIIR and for PVIIRS after adjustment for potential confounders.

## Discussion

BCI is a spectrum of injuries ranging from electrical conduction abnormalities to acute cardiac rupture.<sup>55</sup> Despite several studies, no consensus on standard diagnostic approach to evaluate BCI has been reached. Low incidence of BCI was found in a study where the cardiospecific enzymes were used yielding 19%.<sup>23</sup> Low incidence of BCI varying between 3 and 26% of patients with blunt chest trauma were also found in transthoracic echocardiography studies.<sup>1,13,16,24,27,30,49</sup> Incidences were higher when a transesophageal approach was used so that BCI was detected in 27–56% of the patients.<sup>10,17,30,59</sup> High incidences however, were also found in ECG studies of patients with blunt chest trauma ranging from 29% to 56%.<sup>7,23,24,27,49</sup> Finally, using highly specific troponin I or T, incidences of BCI varied from 15% to 24%.<sup>1,13,49</sup>

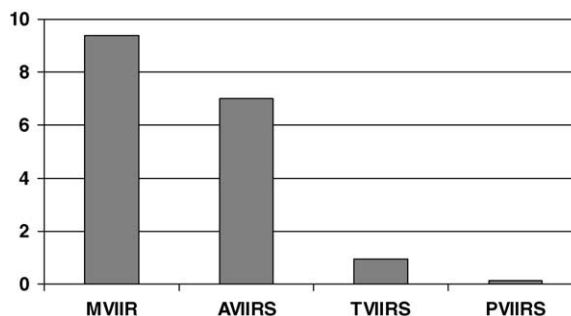
Independent of age, race, sex, injury severity, and source of payment, we observed that BCI was associated with a 12-fold increased risk for TVIIRS. Acute severe elevation of right intraventricular



**Figure 2** Hospitalised age-specific rates (per 100,000 persons) for blunt cardiac injury among different age groups.

pressure has been shown to result in injury of the tricuspid valvular apparatus.<sup>44</sup> Furthermore, the right ventricle is immediately behind the sternum, which make it more vulnerable to blunt trauma.<sup>5</sup> Rupture of the valve is more likely to occur if the injury occurred during diastole when the right ventricular pressure is low,<sup>34</sup> particularly when the right ventricle is compressed between the sternum and the vertebral column.<sup>9,33</sup> The most frequently reported injury is chordal rupture, followed by rupture of the anterior papillary muscle and leaflet tear, primarily of the anterior leaflet.<sup>44</sup> The right ventricle has also been shown to be vulnerable to indirect injury by a sudden increase in intracardiac pressure from compression of the upper abdomen.<sup>5</sup>

We also found that independent of age, race, sex, injury severity, source of payment, and presence of ischaemic heart disease, a person with BCI is 3.4 times more likely to have AVIIRS. The mechanism of “delayed” rupture has been shown to cause traumatic aortic regurgitation, explained by the presence of BCI.<sup>43</sup> Post-traumatic aortic valve regurgitation has been found to affect all ages and is often found with sternal or multiple rib fractures.<sup>37</sup> The valve damage is more likely to occur during systole or early diastole because of



**Figure 3** Hospitalised rate (per 100,000 persons) for MVIIR, AVIIRS, TVIIRS and PVIIRS. AVIIRS, aortic valve incompetence, insufficiency, regurgitation, stenosis; MVIIR, mitral valve incompetence, insufficiency, regurgitation; PVIIRS, pulmonary valve incompetence, insufficiency, regurgitation, stenosis; TVIIRS, tricuspid valve incompetence, insufficiency, regurgitation, stenosis.

**Table 2** Results of multivariate logistic regression analysis on AVIIRS and TVIIRS

Independent variable	AVIIRS OR ( <i>P</i> -value)	TVIIRS OR ( <i>P</i> -value)
BCI	3.41 (0.001)	11.54 (0.001)
Age (10 levels)	(<0.001)	(<0.001)
Race <sup>a</sup>	1.54 (0.017)	0.68 (0.024)
Sex <sup>b</sup>	1.08 (0.379)	1.07 (0.563)
MAISS (6 levels)	(0.418)	(<0.001)
Sources of payment (9 levels)	(0.662)	(0.701)
Ischemic heart disease	1.38 (<0.001)	N/A

AVIIRS, aortic valve incompetence, insufficiency, regurgitation, stenosis; BCI, blunt cardiac injury; MAISS, maximum abbreviated injury severity score; N/A, not applicable; TVIIRS, tricuspid valve incompetence, insufficiency, regurgitation, stenosis.

<sup>a</sup> Referent group: white.

<sup>b</sup> Referent group: males.

compressive forces that may arise following a deceleration injury. At this point in the cardiac pressure and volume cycle, the aortic valve is closed and the transvalvular gradient is maximal.<sup>47</sup>

Although we found that direct trauma to heart – namely BCI – was associated with MVIIR in the univariate analysis, we did not find that such trauma increases the risk of MVIIR in the multivariate regression analyses. The lack of association between BCI and MVIIR could be explained by the confounding by degenerative conditions such as myxomatous degeneration, which as we mentioned above, do not have ICD-9-CM. In some other studies, patients sustained traumatic mitral valve insufficiency have been shown to present with either entire papillary muscle avulsion from its ventricular attachment or with chordal and leaflet damage.<sup>3,4</sup> Those with papillary muscle avulsion presented with symptoms of acute mitral regurgitation with torrential regurge back into the left atrium.<sup>3,11,14,18,20,22,39,52,61</sup> Those patients who experienced chordal and leaflet damage presented with less severe clinical symptoms or were completely asymptomatic.<sup>4,25,29,35,38,40,60</sup>

There are limitations to consider when interpreting the results from this study. Despite adjusting for confounding factors, a strong temporal relationship between BCI and cardiac valvular disorders cannot be established through the current study design. Readers should bear in mind that this database contains discharge-level records, not patient-level records. This means that individual patients who are hospitalised several times in 1997 may be present in the dataset several times. There might be a higher probability of sustaining BCI in the presence of pre-existing valvular disease. Such probability may have been higher because cardiac tissues may have been weakened due to the effects of chronic dilation and hypertrophy such that compression of the cardiac chambers and increase of intracardiac pressure from injury forces were particular significant. On the other hand, pre-existing cardiac valvular disease might lead to a higher likelihood of the

diagnosis of BCI being made, however, this database may provide only limited insight into this issue since acceptance of a specific diagnosis varies not only among hospitals but also across physicians.<sup>26,48,50</sup> Medical chart review, although more expensive, may provide more detailed clinical information.

Another limitation is the fact that the three diagnostic codes reflecting AVIIRS, TVIIRS and PVIIRS may also include stenosis, which has not been shown to have a traumatic origin. This study was based on secondary data analysis, therefore under-reporting could occur.

Other problems related to administrative data include coding accuracy and limited ability to risk adjustment.<sup>26,48,50</sup> As mentioned earlier, diagnostic codes for MVIIR do not exclude myxomatous degeneration, which does not have an ICD-9-CM code. Myxomatous degeneration has been shown to be one of the leading causes for mitral valve insufficiency.<sup>53</sup> Long-standing mechanical stress was hypothesised to play a role in the wear and tear of the valve, resulting in valvular regurgitation.<sup>51</sup> Future research should answer the question whether the existing myxomatous degeneration may increase the risk of getting traumatic mitral valve insufficiency due to the long-term wear and tear of the valve.

We did not find that BCI increases the risk of PVIIRS. The lack of association between BCI and PVIIRS could be explained by a limited statistical power due to the sample sizes. There is no published research that has looked at pulmonary valvular insufficiency as the result of trauma. PVIIRS is quite rare, and future research should focus on possible association between BCI and PVIIRS.

It is important to establish, quantify and better understand the relationship between trauma and cardiac valvular disorders. Traumatic tricuspid valve insufficiency may necessitate early surgical treatment.<sup>31</sup> In general, valvular heart disease has been described as an important risk factor for congestive heart failure. Research shows that

moderate-to-severe right heart failure eventually develops in patients with tricuspid valve injury, even though in chordal rupture the estimated interval is 10–25 years.<sup>15</sup> Levy et al.<sup>35</sup> showed in the Framingham Heart Study that cardiac valvular disease confers more than twice the adjusted risk for development of congestive heart failure and a population-attributable risk of 7% to 8%. This issue becomes particularly important when it is noted that about 2 million people in the US suffer from congestive heart failure with 400,000 cases diagnosed each year.<sup>18</sup> It is the leading cause of hospitalisation and a major cause of chronic disability among patients 65 or older.<sup>21,22</sup> Moreover, heart failure is the only major cardiovascular disease that is increasing in incidence and prevalence.<sup>22</sup>

Cardiac valve insufficiency has been well recognised as an important risk factor for congestive heart failure. With the findings that BCI is associated with an increased risk of specific valvular disorders, it is possible that trauma may play an important and heretofore largely unrecognised role in a portion of the burden of cardiovascular morbidity and mortality. Longitudinal studies with in-depth clinical information are needed to better quantify such burden. They may further our understanding of the relationship between BCI and cardiac valve insufficiency.

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