Sudden cardiac death rates in an Australian population: A data linkage study


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Sudden Cardiac Death Rates in an Australian Population: a Data Linkage Study

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Competing interests

The authors declare that they have no competing interests.
Abstract

Objective. To develop criteria for identifying sudden cardiac death (SCD) and estimate population rates of SCD using administrative mortality and hospital morbidity records in Western Australia.

Methods. We developed criteria for identifying SCD and estimated average crude, age-standardised and age-specific rates of SCD using population person-linked administrative data.

Results. A total of 9,567 probable SCDs were identified from 1997 to 2010, with one third aged ≥35 years having no prior admission for cardiovascular disease. SCD was more frequent in men (62.1%). The estimated average annual crude SCD rate for the period was 34.6 per 100,000 person-years with an average annual age-standardised rate of 37.8 per 100,000 person-years. Age-specific standardised rates were 1.1 per 100,000 person-years and 70.7 per 100,000 person-years in people aged 1-34 and ≥35 years, respectively.

Ischaemic heart disease (IHD) was recorded as the underlying cause of death in approximately 80% of cases aged ≥35 years, followed by valvular heart disease and heart failure. IHD was the most common cause of death in 1-34 year-olds, followed by unspecified cardiomyopathy and dysrhythmias.

Conclusions. Administrative morbidity and mortality data can be used to estimate rates of SCD and therefore provide a suitable methodology for monitoring SCD over time. The findings highlight the magnitude of SCD and its potential for public health prevention.
What is known on this topic?

- There is considerable variability in rates of sudden cardiac death worldwide.
- Different data sources and varied methods of case ascertainment likely contribute to such variation.

What does this paper add?

- The rate of sudden cardiac death in Australia is low in comparison to international estimates.
- Two in every three cases of sudden cardiac death aged $\geq 35$ years had a hospitalisation history of cardiovascular disease highlighting the opportunity for prevention.

What are the implications for practitioners?

- High-quality person-linked administrative hospital morbidity and registered mortality data can be used to estimate rates of sudden cardiac death in the population.
- Understanding the magnitude and distribution of sudden cardiac death is imperative for developing effective public health policy and prevention measures.
Introduction

Cardiovascular disease (CVD) is a leading cause of mortality worldwide, accounting for more than 17 million deaths annually.\(^1\) Up to 50% of all such deaths are sudden,\(^2\) highlighting sudden cardiac death (SCD) as a public health concern. Considerable variation in rates of SCD exists ranging from 60 to >150 per 100,000 person-years in the United States (US)\(^3,4\) and varying from 40 to 100 per 100,000 person-years in Europe and Asia.\(^5-8\) Such differences are likely due to varied data sources and different methods of case ascertainment. New strategies for SCD case ascertainment such as use of routinely collected administrative morbidity and mortality data, offer an opportunity to derive estimates of SCD rates over time and thereby advance our knowledge and provide a platform for its prevention.

Few population studies of SCD are Australian. An indirect measure of SCD in Western Australia (WA) estimated the out-of-hospital cardiac arrest rate at 60.2 per 100,000 person-years.\(^9\) An earlier small Australian study estimated the out-of-hospital SCD rate at 40 per 100,000 person-years in 25-94 year-olds.\(^10\) Additionally, two Australian and New Zealand studies report low rates of SCD in 1-35 year-olds (1.1 per 100,000 person-years)\(^11\) and 0-40 year-olds (2.0 per 100,000 person-years) respectively.\(^12\) The contrasting age-specific difference in rates of SCD is likely explained by the disparities in underlying mechanisms that occur with age, methodologies for case ascertainment, and definitions of SCD.\(^13\)

Improving our knowledge of SCD is contingent upon having a consistent, reliable and measurable definition. The widely accepted definition of SCD is unexpected natural death that occurs within one hour from the onset of symptoms if death is witnessed, and within 24 hours of being seen alive and well when unwitnessed.\(^14\) However, this definition is difficult to apply due to determining the precise timing from symptom onset to cardiac collapse and
the fact that many SCD cases go unwitnessed, despite many of them occurring at home.\textsuperscript{7} As argued by others,\textsuperscript{14} the definition of SCD should be less specific and sufficiently flexible for an epidemiological study that sheds light on the general population rather than individual cases. Further, there is no national registry for SCD in Australia, and thus the population impact of this life-ending event is poorly understood.

Despite the availability of a specific code for SCD in the International Classification of Diseases (ICD), SCD is rarely coded as the cause of death (COD). This is likely due to coding guidelines stating the SCD code may only be assigned if resuscitation is administered regardless of the patient outcome. The objectives of this study were to develop criterion for the identification of probable SCD cases using population-wide person-based administrative morbidity and mortality data and to quantify the magnitude of SCD in a representative Australian state.

**Methods**

**Population**

In 2010, the population of WA was 2.3 million, 75% of whom resided in the capital city, Perth.\textsuperscript{16} Net overseas and interstate migration rates are low and estimated at 1.3% and 0.1%, respectively.\textsuperscript{16} WA has comparable key socio-demographic and health economic indicators with other Australian States, including age, sex, indigenous status, rural/remote population, out of state migration, available hospital beds, health expenditure, and Medicare benefits paid.\textsuperscript{17}

**Data sources**
In Australia, the patient’s hospital chart is the primary source of information for the coding of inpatient morbidity. ICD codes for episodes of care and comorbidities are recorded using information from the patient discharge summary and the clinical notes. Information about a death is recorded on a death certificate, and coded by the Australian Bureau of Statistics (ABS).\textsuperscript{18} Death certification on a natural unexpected demise is completed in one of two ways: (i) the police, funeral director and/or the medical practitioner provides supporting evidence and a determination as to the COD; or (ii) if none of these groupings can certify the COD then the case is referred to a coroner for a determination where police investigation, autopsy, and toxicology analysis are involved.\textsuperscript{18} Where the coroner is unable to reach a decision on a case, a recommendation is made to assign a code for an undetermined death, as regulated by the ABS.\textsuperscript{19}

\textit{Data linkage system}

Administrative data for all hospital morbidity records and deaths in WA are contained in the Hospital Morbidity Data Collection (HMDC) and Mortality Register, respectively, both of which are regularly audited for quality.\textsuperscript{20} They are linked at an individual level through the WA Data Linkage System by probabilistic matching, with accuracy of > 99%.\textsuperscript{20} The morbidity dataset covers all public and private hospital admissions in WA. The mortality dataset covers multiple COD that include categories for underlying, all associated COD (including direct, antecedent, and contributory causes), and occurrence of a post-mortem. Contributory causes were excluded from this study as they are pre-existing conditions and do not directly influence the death.\textsuperscript{18} Multiple COD data in WA became available for research from 1997 onwards. The linked dataset for this study contains all hospital admissions and death records for CVD from 1987 to 2010, permitting a 10-year look-back period for CVD hospitalisation history.
Criteria for SCD cases

Four criteria were developed to identify probable SCD cases (Table 1). Each criterion utilised a combination of two or three components including place, death within 24 hours, diagnosis, underlying cause, and/or additional information (associated COD and occurrence of a post-mortem). We estimated death within 24 hours using admission and death dates. No priority was applied to any of the components in each criterion. Some cases were identified by more than one criterion but only counted once. The prevailing ICD version and relevant modifications (ICD-9 from 1 July 1979, and ICD-10 from 1 July 1999) were used to identify cases. The term ‘SCD-related disease’ was created to incorporate a number of conditions from the literature known to be associated with SCD. It includes myocardial infarction (MI), other ischaemic heart disease (IHD), cardiomyopathy, conduction disorder, ventricular fibrillation (VF), ventricular tachycardia (VT), cardiac arrest, other dysrhythmias, heart failure, myocarditis, endocarditis, pericarditis, valvular heart disease, pulmonary heart disease, rheumatic heart disease, and congenital heart disease3,15 (Supplementary Table 1). Inclusion of all IHD codes is based on previous validation studies in Australia26, and international recommendations for identifying CHD deaths from administrative data27. We excluded cases <1 year old, where COD is often related to sudden infant death syndrome and/or to gene defects not exclusively cardiac in origin.11 In cases of SCD in ≥35 year-olds, prior CVD (ICD-9 390-459, ICD-10 I00-I99) was determined from a 10-year review of hospitalisation records of all diagnosis fields. This study was approved by the Human Research Ethics Committees of The University of Western Australia (#2014/55) and the Department of Health (Western Australia) (#RA/4/1/1491).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Criteria for probable sudden cardiac death identification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Place, time, diagnosis</td>
<td>Underlying cause</td>
</tr>
<tr>
<td>(from death record)</td>
<td>(from death record)</td>
</tr>
</tbody>
</table>


Criterion 1

died out-of-hospital  
OR  
died in-hospital \( \leq 24 \) hours of any-cause admission  
AND  
underlying COD was a SCD-related disease*  
AND  
associated COD† was VF, VT or cardiac arrest

Criterion 2

died in-hospital  
\( \leq 24 \) hour of admission for  
VF, VT or cardiac arrest recorded in any diagnostic field  
AND  
underlying COD was a SCD-related disease*

Criterion 3

died out-of-hospital  
OR  
died in-hospital \( \leq 24 \) hours of any-cause admission  
AND  
underlying COD was SCD-related disease*  
AND  
indications of whether post-mortem was conducted

Criterion 4

died out-of-hospital  
OR  
died in-hospital following an  
admission for MI as principal diagnosis  
within last 28 days  
AND  
underlying COD was a SCD-related disease*

*SCD-related disease included ischaemic heart disease, cardiomyopathy, dysrhythmias, heart failure, myocarditis, endocarditis, pericarditis, valvular heart disease, pulmonary heart disease, rheumatic heart disease, and congenital heart disease.

†Associated COD included direct and antecedent cause(s) as determined from the death record.

COD, cause of death; SCD, sudden cardiac death; VF, ventricular fibrillation; VT, ventricular tachycardia; MI, myocardial infarction.

Statistical analyses

All statistical analyses were performed using SAS (v9.3). A value of \( P<0.05 \) was considered statistically significant for the comparison analyses. Significant differences between groups were determined using the 2-sided t-test and Pearson’s Chi-squared test for continuous and categorical variables respectively. An average crude rate for the period was calculated using the total number of SCD cases for each calendar year divided by the corresponding annual total WA population aged over 1 year. Rates of SCD by gender and age were standardised by
the direct method using 5-year age groups with the 2006 WA population as the standard. To permit comparisons of our rate with published standardised rates, we also recalculated our age-standardised rates (ASRs) using the standard population, age groups, and calendar years for each of the corresponding reported studies. For those studies only providing the crude rates, we recalculated our crude rate for the same calendar years and ages. Two sensitivity analyses were conducted. First, we identified SCD cases as occurring outside the hospital (including emergency room) and using only the underlying COD where it was SCD-related disease. Secondly, ASRs were recalculated after excluding cases aged ≥35 years with prior hospitalisations for heart failure, valvular heart disease or implantable cardiac defibrillator, to reduce potential misclassification of non-SCDs.

### Results

**Number and distribution of SCD cases**

Between January 1, 1997 and December 31, 2010, a total of 9,567 probable SCDs were estimated to have occurred in WA, of which 5,943 (62.1%) were men. Almost 10% of cases were identified by more than one criterion (Figure 1). Nearly a third of all SCDs had documented life-threatening arrhythmia (Criterion 1 and/or Criterion 2). Approximately 40% of cases died out-of-hospital or in-hospital within 24 hours of any-cause admission and had a post-mortem (Criterion 3). Approximately 20% of cases occurred in-hospital. Most SCDs (98%) occurred in those aged ≥35 years, with about one-third of these (34.0%) having no hospitalisation history of CVD in the ten years prior to death. Only 155 (1.6%) cases of probable SCD were identified in 1-34 year olds. Mean age was significantly different between males and females [68.8 (15.1) years versus 77.9 (14.6) years; p<0.0001]. The proportion of men versus women succumbing to SCD narrowed with increasing age and reversed in the oldest age group (Supplementary figure 1).
SCD rates

The estimated average annual crude SCD rate for the entire population from 1997 to 2010 was 34.6 per 100,000 person-years, with an average annual ASR of 37.8 per 100,000 person-years. The estimated average annual ASRs for males and females were 51.5 per 100,000 person-years and 24.1 per 100,000 person-years, respectively. In people aged 1-34 years, the average annual ASR was 1.1 per 100,000 person-years, compared with 70.7 per 100,000 person-years in those aged ≥35 years. The average annual age-specific rates of SCD were higher in men than in women (Figure 2).

Cause of death

The distribution of the recorded underlying COD for SCD is shown in Table 2. Overall, IHD was recorded as the underlying cause in 87.2% of cases. The most common underlying COD in 1-34 year-olds was IHD (43.2% of cases). Rheumatic heart disease, congenital heart disease, pulmonary heart disease, myocarditis, endocarditis, pericarditis, and hypertrophic cardiomyopathy were more common in the younger group.
<table>
<thead>
<tr>
<th>Underlying cause of death*, %</th>
<th>1-34 year-olds</th>
<th>≥35 year-olds</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=155)</td>
<td>(n=9,412)</td>
<td>(n=9,567)</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>13.5</td>
<td>43.5(^c)</td>
<td>43.0</td>
</tr>
<tr>
<td>Other ischaemic heart disease†</td>
<td>29.7</td>
<td>44.4(^b)</td>
<td>44.2</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>3.9</td>
<td>2.4</td>
<td>2.5</td>
</tr>
<tr>
<td>Heart failure</td>
<td>0.6</td>
<td>1.9</td>
<td>1.9</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>1.3</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>1.9</td>
<td>0.04(^c)</td>
<td>0.1</td>
</tr>
<tr>
<td>Unspecified cardiomyopathy§</td>
<td>11.0</td>
<td>1.7(^c)</td>
<td>1.8</td>
</tr>
<tr>
<td>VF/VT and cardiac arrest</td>
<td>1.3</td>
<td>1.2</td>
<td>1.4</td>
</tr>
<tr>
<td>Dysrhythmias, other‡</td>
<td>9.7</td>
<td>1.2(^c)</td>
<td>1.2</td>
</tr>
<tr>
<td>Conduction disorders</td>
<td>0</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Pulmonary heart disease</td>
<td>5.8</td>
<td>1.5(^b)</td>
<td>1.6</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>5.2</td>
<td>0.4(^c)</td>
<td>0.5</td>
</tr>
<tr>
<td>Congenital heart disease</td>
<td>4.5</td>
<td>0.2(^c)</td>
<td>0.2</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>7.1</td>
<td>0.1(^c)</td>
<td>0.2</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>2.6</td>
<td>0.4(^a)</td>
<td>0.4</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>1.9</td>
<td>0.3(^b)</td>
<td>0.3</td>
</tr>
</tbody>
</table>

VF, ventricular fibrillation; VT, ventricular tachycardia

\(^a\) p-value <0.05, \(^b\) p-value <0.001, \(^c\) p-value <0.0001 for comparison between age groups, from chi-squared test.

\(^\ast\) From the International Classification of Diseases versions 9 and 10.

\(^\dagger\) Includes angina pectoris and chronic ischaemic heart disease.

\(^\S\) Includes other restrictive and non-obstructive hypertrophic cardiomyopathy.

\(^\ddagger\) Includes paroxysmal tachycardia, atrial fibrillation and flutter, and unspecified cardiac dysrhythmias.

Sensitivity analyses

The estimated overall crude rate of SCD was higher (100.6 per 100,000 person-years vs 34.6 per 100,000 person-years) when only underlying COD was used for identification of cases occurring outside hospital. Additionally, the estimated rate of SCD reduced to 62.9 per
100,000 person-years in cases aged ≥35 years, when the cases with prior hospitalisations for heart failure (n=769), valvular heart disease (n=230), implantable cardiac defibrillator (n=39) or any combination of the three aforementioned (n=36) were excluded.

Discussion

We have estimated the ASR of probable SCD in WA in a population of around 2 million, using a 14-year study period of person-linked administrative morbidity and mortality data. Our results suggest the rate of SCD is low, with IHD recorded as the main underlying cause of death in the majority of cases. The rates of SCD were mostly higher in men than in women. Importantly, two-thirds of SCD cases aged ≥35 years had a hospitalisation history of CVD in the ten years prior to their death, highlighting the potential for public health prevention.

The rate of SCD in our WA study was relatively low compared to previous studies that used a population-level approach (Table 3). Age and sex differences across studies complicate comparison of crude rates of SCD. Despite recalculating our rates using methods comparable to the reported studies, disparities persisted. Our estimated rate in WA is around one third of the US’s, half of the Netherlands, yet is approximately 1.4 times higher than that of Northern Ireland and China.
### Table 3: Estimated rates of sudden cardiac death in various populations and the comparable WA rate

<table>
<thead>
<tr>
<th>Setting</th>
<th>Study data collection</th>
<th>Population</th>
<th>Age included (years)</th>
<th>Study source</th>
<th>Average Annual rate (per 100,000 person-years)</th>
<th>Reported rate</th>
<th>Comparable WA rate§</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current study</td>
<td>1997-2010</td>
<td>2,023,662</td>
<td>≥1</td>
<td>Linked Hospital Morbidity Data Collection and Mortality Register</td>
<td>37.8</td>
<td>37.8</td>
<td></td>
</tr>
<tr>
<td>USA³</td>
<td>1989-1998</td>
<td>140,360,000</td>
<td>≥35</td>
<td>National mortality data</td>
<td>162.1</td>
<td>97.6</td>
<td></td>
</tr>
<tr>
<td>Belfast, Northern Ireland⁷</td>
<td>2003-2004</td>
<td>1,704,924</td>
<td>All</td>
<td>Emergency medical services and necropsy reports</td>
<td>88.0</td>
<td>27.4</td>
<td></td>
</tr>
<tr>
<td>The Netherlands⁸</td>
<td>1995-2001</td>
<td>11,991,000</td>
<td>≥18</td>
<td>Medical records</td>
<td>103.8</td>
<td>65.8</td>
<td></td>
</tr>
<tr>
<td>Kelamayi, Yuxian, Beijing, Guangzhou, China⁶</td>
<td>2005-2006</td>
<td>760,480,293</td>
<td>≥25</td>
<td>Death certificate, next-of-kin interviews, medical records</td>
<td>41.8</td>
<td>26.7</td>
<td></td>
</tr>
<tr>
<td>Current study</td>
<td>1997-2010</td>
<td>2,023,662</td>
<td>≥1</td>
<td>Linked Hospital Morbidity Data Collection and Mortality Register</td>
<td>34.6</td>
<td>34.6</td>
<td></td>
</tr>
<tr>
<td>Multnomah County, Oregon⁴</td>
<td>2002-2005</td>
<td>660,486</td>
<td>All</td>
<td>Medical records, clinical data, and available autopsy examination</td>
<td>58.0</td>
<td>24.4</td>
<td></td>
</tr>
<tr>
<td>Galway, Mayo, and Roscommon, West Ireland⁵</td>
<td>2005</td>
<td>380,057</td>
<td>All</td>
<td>Emergency room resuscitation records, autopsies</td>
<td>51.2</td>
<td>26.7</td>
<td></td>
</tr>
<tr>
<td>England and Wales¹³</td>
<td>2002-2005</td>
<td>3,997,664</td>
<td>1-34</td>
<td>Office of National Statistics mortality data</td>
<td>1.8</td>
<td>0.4</td>
<td></td>
</tr>
<tr>
<td>Sydney, Australia¹¹</td>
<td>1994-2002</td>
<td>3,140,645</td>
<td>1-35</td>
<td>Demographic, clinical and autopsy data</td>
<td>1.1¹</td>
<td>1.2²</td>
<td></td>
</tr>
</tbody>
</table>

£ Standard population and/or study population described in each corresponding study for rate calculation as the denominator. § Comparable WA age-standardised rates were calculated using the standard population, age groups, and calendar years from each corresponding study; # only calculated for our study period of 1997-2010; † WA crude rates were calculated using the WA population under the same study duration and age range from each corresponding study; ‡ calculated and estimated according to the literature.
Different methodology for case ascertainment is likely to account for some of the reported variation in the rates of SCD worldwide. A death-certificate based approach relying on disease codes for case ascertainment is a common method. It offers objective mortality information and provides uniform data where standard disease coding guidelines are employed. However, a validation study of the ‘death certificate’ method overestimated the SCD rate by 200 to 300 percent. In contrast, when multiple data sources (death certificates, hospital records, next-of-kin interviews, and/or autopsy reports) were used for case ascertainment, the rates of SCD reported (50 and 100 per 100,000 person-years) are considered more accurate. When using only underlying COD to identify SCD in our study, rates were overestimated by 166 percent. This lends further indirect support that Australia may have a lower rate of SCD than other developed countries, as has been shown for out-of-hospital cardiac arrest. The rates of SCD in our study are lower than in those studies using the most comparable data sources (national mortality statistics). However, differences in definitions of SCD and other methodological and case ascertainment factors such as cause of death and hospital admission coding practices may also be responsible for this apparent difference in rates. Differential rates of post-mortem although they are in general decline internationally, including Australia, may be another explanation for discrepancies in rates of SCD between studies.

Given current knowledge of the complex pathophysiological mechanisms of SCD, we are likely to enhance SCD case ascertainment by considering multiple criteria for underlying COD. Life-threatening conditions including fatal arrhythmias are an important pathophysiological mechanism precipitating SCD, and integral to capturing cases. However, alone it would provide a lower estimate of SCD due to many SCD cases being unwitnessed and the challenges of assigning death due to arrhythmia or asystole. Non-
arrhythmic causes are found especially in patients with recent MI,\textsuperscript{22} hence our inclusion of
patients dying within 28 days of MI hospitalisation. Additionally, a post-mortem indicates
that the death was unexpected and unexplained,\textsuperscript{19} thereby enabling our criteria to inform the
probable cases. However, this multifaceted approach to underlying COD potentially has
implications for public health measures. Under-reporting and under-recognition of conditions
such as pulmonary embolus and dysrhythmias as causes of death may reduce our ability to
effectively target prevention measures for SCD.

Different risk profiles for different study populations may be another explanation for the
observed variations in rates across studies. Conventional risk factors account for 75\% of the
CVD epidemic worldwide,\textsuperscript{23} although different associations of risk factors for SCD exist in
different populations.\textsuperscript{24} Environmental and societal factors e.g., lifestyle, dietary habits, and
genetic influences\textsuperscript{24} play a role in SCD risk. A notable finding in our study and other
studies\textsuperscript{5,22} is that SCD occurs earlier in men. This suggests males may have specific risk
factors that predispose to SCD which warrants further investigation. Further research
exploring the factors contributing to the differences in the rates of SCD would provide
insights into the potential for prevention.

In our study and elsewhere,\textsuperscript{3,5} IHD was the most common underlying COD, with MI recorded
for about half and other IHD for approximately 44\% of all COD. Interestingly, many of these
cases had a post-mortem (66\%, as shown in Supplementary Table 2), suggesting difficulty
with determining COD. Consistent with previous work,\textsuperscript{11} hypertrophic cardiomyopathy was
a less frequent cause of SCD in our younger group compared to other types of
cardiomyopathy, however was significantly more frequent than in the cases aged $\geq$ 35 years.
Rheumatic heart disease and pulmonary heart disease were likewise more common in the
younger group (1-34 year-olds). The ICD classification of ‘VT, VF, cardiac arrest, and other
dysrhythmias’ while not indicating a specific underlying disease state is speculated to include
SCD, and the significantly greater frequency of this coding in the younger group (1-34 year
olds), supports this.\textsuperscript{11}

We used high-quality person-linked administrative morbidity and mortality data and applied
four criteria based on best available evidence to derive population-wide estimates of SCD.
However, several limitations of this study warrant consideration. Access to individual
medical records, death certificates, and autopsy reports was not possible. Thus, potential
misclassification of diagnostic and COD coding cannot be excluded. However, based on a
previous study that reported the reliability of COD data at 74\% sensitivity and 97\%
specificity for IHD\textsuperscript{25} and our multiple criteria for case ascertainment, the misclassification of
SCD is considered low. It is also possible that the increasing awareness of IHD over the study
period may have led to earlier investigation and therapy, thereby altering the number of cases
that were captured and defined as SCD.

CONCLUSIONS

Administrative morbidity and mortality data can be used to estimate age-standardised and
age-specific rates of probable SCD and therefore provide a new method for monitoring SCD.
Regular validation studies on diagnostic and COD coding and availability of detailed
circumstances surrounding deaths would improve the use of person-linked data for the study
of SCD. Additionally, the findings underscore the magnitude of SCD, and thus the potential
for public health prevention.
References


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**Figure 1** Number of probable sudden cardiac death cases identified by the four criteria. 

*grey shadow represents the proportion of cases that were identified by more than one criterion.*

Criterion 1: died out-of-hospital OR died in-hospital \( \leq 24 \text{ hours of any-cause admission} \)
AND died of SCD related disease AND withVF, VT or cardiac arrest occurred.

Criterion 2: died in-hospital \( \leq 24\text{-hour of admission for VF, VT or cardiac arrest recorded in any diagnostic field} \)
AND died of SCD related disease.

Criterion 3: died out-of-hospital OR died in-hospital \( \leq 24 \text{ hours of any-cause admission} \)
AND died of SCD related disease AND with indications of whether post-mortem was conducted.

Criterion 4: died out-of-hospital OR died in-hospital following an admission for AMI as principal diagnosis within last 28 days AND died of SCD related disease.
Figure 2 Age- and gender- specific average annual rate of probable sudden cardiac death in Western Australia, from 1997 to 2010.