A n epidemic of coronary heart disease (CHD) began during the 20th century in most industrialized countries, where CHD is a leading cause of mortality among adults. Developing countries show the beginnings of the same epidemic. Reliable information on population incidence, prevalence, and case-fatality rates of CHD is essential to understanding, treating, and controlling the epidemic but is generally unavailable. Consistent and universal definitions of cases of CHD allow the determination of rates and comparisons within and between populations. These case definitions are essential to epidemiological studies and other research, such as clinical trials, quality assurance, and economic analysis of healthcare costs. The need for standardization is clear, and this statement recommends updated definitions.

Definitions of cases for epidemiology studies and clinical trials in acute CHD rest on World Health Organization (WHO) (1959) and American Heart Association (1964) reports, followed by the WHO European AMI Registry criteria. Myocardial infarction (MI) is based on cardiac symptoms, ECG changes, and/or elevation in biomarkers. This basic system has been widely used but variably interpreted, resulting in a lack of comparability among and within studies. Further specification and working definitions of CHD come from the Framingham Study. The WHO criteria were revised in a joint report with the International Society and Federation of Cardiology in 1979. More recently, the WHO MONICA Study and other surveillance and intervention studies, such as the Lipid Research Clinics in the United States, have modified further the definition of CHD cases. These changes are usually based on a greater specification to the original WHO definition to allow for application in different settings.

Advancing diagnostic technology, therapeutic interventions, and changing disease presentation in recent years forces a reevaluation of case definitions for acute CHD. New biomarkers, cardiac troponins, and creatine kinase (CK)-MB mass provide information that is more sensitive and/or specific in detecting even minor myocardial cell damage. New imaging methods, such as MRI and radioisotope imaging, although not widely available today, will add to the diagnostic tools. These developments were recently reviewed in a Joint European Society of Cardiology/American College of Cardiology Workshop on the Redefinition of Myocardial Infarction. That report, published in 2000, provided direction for clinicians faced with changing diagnostic testing and new information. Another recent report extends advice to clinical trials. However, they fall short of providing direction for epidemiologists faced with evaluating and interpreting trends in event rates on the basis of retrospective surveillance.

Patterns of CHD presentation are also changing. Whether because of changes in disease severity, improved diagnostic testing, increased professional awareness of the diagnosis, heightened public awareness of CHD symptoms, or insurance reimbursement to hospitals, cases of CHD in hospitalized
Definitions of Ischemic CHD

The definition of a CHD case depends on symptoms, signs, biomarkers, and ECG and/or autopsy findings. These data may vary in quantity, quality, and timing. On the basis of the extent and diagnostic quality of data, definite, probable, and possible cases of fatal and nonfatal MI, procedure-related events, and angina pectoris are defined. The following outlines the definitions and recommendations, which are summarized in Table 1. The recommendations emphasize biomarkers in a setting in which signs, symptoms, and/or ECG findings suggest acute ischemia.

Definition of Terms

Cardiac Biomarkers

Cardiac biomarkers are blood measures of myocardial necrosis, specifically CK, CK-MB, CK-MBm, or troponin (cTn). The order of diagnostic value is cTn > CK-MBm > CK-MB > CK.

A. Adequate set of biomarkers: At least 2 measurements of the same marker taken at least 6 hours apart

B. Diagnostic biomarkers: At least 1 positive biomarker in an adequate set (see A above) of biomarkers showing a rising or falling pattern in the setting of clinical cardiac ischemia and the absence of noncardiac causes of biomarker elevation

C. Equivocal biomarkers: Only 1 available measurement that is positive, or a rising or falling pattern not in the setting of clinical cardiac ischemia or in the presence of nonischemic causes of biomarker elevation

D. Missing biomarkers: Biomarkers not measured

E. Normal biomarkers: Measured biomarkers do not meet the criteria for a positive biomarker (see F below)

F. Positive biomarkers: At least 1 value exceeding the 99th percentile of the distribution in healthy populations or the lowest level at which a 10% coefficient of variation can be demonstrated for that laboratory

Cardiac Symptoms and Signs

Cardiac symptoms and signs are findings from patient interview and examination.

A. Cardiac symptoms: Presence of acute chest, epigastric, neck, jaw, or arm pain or discomfort or pressure without apparent noncardiac source. More general, atypical symptoms, such as fatigue, nausea, vomiting, diaphoresis, faintness, and back pain, should not be used as a diag-
nostic criterion, although they are clinically useful in arriving at the correct diagnosis.

B. Cardiac signs: Acute congestive heart failure or cardiogenic shock in the absence of non-CHD causes.

ECG Findings

One or more ECG(s) may be collected in a possible cardiac event. These should be adjudicated or classified when possible.

The evolution of ECG findings may be demonstrated (1) between the ECG(s) associated with the event or (2) between a previously recorded ECG and the event ECG(s). In cases in which only a single event ECG is available, an evolving diagnostic ECG pattern can be recorded only if a previous study ECG is available (eg, if there is no previous study ECG and only 1 event-related ECG, there can be no classification of “evolving diagnostic” ECG).

Precise measurement guidelines for measurement of wave onset and offset to determine wave duration and voltage must be followed. Most events likely to be MI occur in settings not controlled by epidemiology researchers, so that most ECG(s) will be hard copy, with varying levels of quality. The most extensively used measurement system for visual ECG findings is the Minnesota Code.12 These measurement guidelines should be coupled with validated biologically acceptable degrees of change in ECG wave forms to code an evolution of change. These rules for proportional change are described elsewhere.13 Another coding system that standardizes the measurement of ECG wave patterns is the Novacode (an extension of the Minnesota Code), which was designed for clinical trial ascertainment of MI.14 More details on ECG coding are available on the Minnesota ECG Coding Center web site.15

The categories are as follows:

A. Evolving diagnostic ECG: See Table 2
B. Positive ECG: See Table 3

### TABLE 2. Evolving Diagnostic ECG (Any of the Following: Q1 through Q4)

| Evolving Q1: | No Q-code in previous study ECG or first ECG in event set of ECG(s) followed by a record with a diagnostic Q-code (Minnesota Code 1-1-1 through 1-2-5 plus 1-2-7) OR any code 1-3-X or 1-2-6 in baseline ECG followed by a record with any code 1-1-X. |
| Evolving Q2: | An equivocal Q-code (Minnesota Code 1-2-8 or any 1-3 code) and no major ST-segment depression in previous study ECG or first ECG in event set of ECG(s) followed by a record with a diagnostic Q-code PLUS a major ST-segment depression (Minnesota Code 4-1-X or 4-2) and 100% increase in ST depression. |
| Evolving Q3: | An equivocal Q-code (Minnesota Code 1-2-8 or any 1-3 code) and no major ST-segment depression in previous study ECG or first ECG in event set of ECG(s) followed by a record with a diagnostic Q-code PLUS a major T-wave inversion (Minnesota Code 5-1 or 5-2) and 100% increase in T-wave inversion. |
| Evolving Q4: | An equivocal Q-code and no ST-segment elevation in previous study ECG or first ECG in event set of ECG(s) followed by a record with a diagnostic Q-code PLUS a major T-wave inversion (Minnesota Code 9-2) and 100% increase in STE. |

Note: A significant Q-code change requires ≥50% increase in event Q/R ratio or ≥1-mm initial R-wave amplitude decrease in event ECG compared with corresponding lead(s) of baseline ECG.

### TABLE 3. Positive ECG

Includes:

(a) Evolving ST elevation alone

- Evolving STE 1: No 9-2 in previous ECG or first ECG in event set of ECG(s) and 9-2 in at least 2 leads of a following event ECG with 100% increase in STE in both leads
- Evolving STE 2: 9-2 in previous ECG or first ECG in event set of ECG(s) with 100% increase in STE in at least 2 leads
- Evolving STE 3: 9-2 and no 5-1 or 5-2 in previous ECG in first ECG in event set of ECGs and appearance of 5-1 or 5-2 with 100% increase in T-wave inversions in at least 2 leads
- Evolving STE R1: Reversal of evolving STE 1
- Evolving STE R2: Reversal of evolving STE 2

OR

(b) Evolving equivocal Q-wave plus evolving ST-T depression/inversion

- Evolving Q5: No Q-code and neither 4-1-X nor 4-2 in previous study ECG or first ECG in event set of ECG(s) followed by a record with an equivocal Q-code PLUS 4-1-X or 4-2 and 100% increase in ST depression
- Evolving Q6: No Q-code and neither 5-1 nor 5-2 in previous study ECG or first ECG in event set of ECG(s) followed by a record with an equivocal Q-code PLUS 5-1 or 5-2 100% increase in T-wave inversion
- Evolving Q7: No Q-code and no 9-2 in previous study ECG or first ECG in event set of ECG(s) followed by a record with an equivocal Q-code PLUS a 9-2 and 100% increase in STE

OR

(c) New left bundle-branch block (code 7-1-1, with the QRS duration increasing by at least 20 ms from less than 120 ms to ≥120 ms)

STE indicates ST elevation.

In the case of a single ECG available only from a possible event hospital admission, a probable MI can be classified if compared with the previous study ECG, there is a new appearance of a diagnostic Q-wave (=MC 1-1-1 through 1-2-5 plus 1-2-7 or any code 1-3-X in the previous ECG and the event ECG has any code 1-1-X or presence of 9-2 in at least 2 leads).
TABLE 4. Nonspecific ECG: Evolution of Minor ST-T Depression/Inversion Alone or Minor Q-Wave Evolution Alone

(a) Evolving non-STE non-Q-wave pattern MI
- Evolving ST-T1: Either 4-0 (no 4-code), 4-4, or 4-3 in previous ECG or first ECG in event set of ECG(s) followed by a record with 4-2 or 4-1-2 or 4-1-1 and 100% increase in ST segment depression
- Evolving ST-T2: Either 4-2 or 4-1-2 in previous ECG or first ECG in event set of ECG(s) followed by a record with 4-1-1 and 100% increase in ST segment depression
- Evolving ST-T3: Either 5-0, 5-4, or 5-3 in previous ECG or first ECG in event set of ECG(s) followed by a record with 5-2 or 5-1 and 100% increase in T wave inversion
- Evolving ST-T4: Code 5-2 in previous ECG or first ECG in event set of ECG(s) followed by a record with 5-1 and 100% increase in T wave inversion
- Evolving ST-T5: Code 4-1-1 in previous ECG or first ECG in event set of ECG(s) followed by a record with 4-1-1 and 100% increase in ST depression
- Evolving ST-T6: Code 5-1 in previous ECG or first ECG in event set of ECG(s) followed by a record with 5-1 with 100% increase in T wave inversion
- Evolving ST-T7: Code 5-2 in previous ECG or first ECG in event set of ECG(s) followed by a record with 5-2 with 100% increase in T wave inversion
- Evolving ST-T R1 through ST-T R7 = the reverse of ST-T1 to ST-T7, respectively

OR

(b) Evolving minor Q wave alone
- No Q code in previous study ECG or event ECG, followed by an event ECG with an equivocal Q-code (1-2-8 or any 1-3 code)

C. Nonspecific ECG: See Table 4
D. ECG negative for ischemia: Normal ECGs or findings other than those described in Tables 2 through 4

Postmortem Consistent With Acute MI
Postmortem findings consistent with acute MI are a cardiac pathology consistent with recent coronary occlusion or MI ≤28 days old.

Case Classifications for CHD
I. Nonfatal events
   A. Define MI
      1. Evolving diagnostic ECG, or
      2. Diagnostic biomarkers
   B. Probable MI
      1. Positive ECG findings plus cardiac symptoms or signs plus missing biomarkers, or
      2. Positive ECG findings plus equivocal biomarkers
   C. Possible MI
      1. Equivocal biomarkers plus nonspecific ECG findings, or
      2. Equivocal biomarkers plus cardiac symptoms or signs, or
      3. Missing biomarkers plus positive ECG
   D. Unrecognized MI
      1. Appearance, in a nonacute setting, of a new diagnostic Q wave with or without ST-T wave depression, or ST elevation (Q1 through Q7 in Tables 2 and 3)
   E. Medical procedure-related event
      1. Cardiac events after (up to 28 days) a medical procedure (eg, general surgery) with criteria for definite, probable, and possible MI identical to those described above (I.A–C)
      2. May be reported separately as procedure-related cardiac events or combined with overall event rates
      3. If the medical procedure was performed for the treatment of acute ischemia (eg, angioplasty, coronary bypass surgery), an event should be classified as described above (I.A–C) and not considered procedure-related

F. Unstable angina pectoris
   1. New cardiac symptoms and positive ECG findings with normal biomarkers
   2. Changing symptom pattern and positive ECG findings with normal biomarkers
   G. Stable angina pectoris
   1. Cardiac symptoms in a pattern that remains constant in presentation, frequency, character, and duration over time

II. Fatal events (hospitalized patients)
   A. Define fatal MI
      1. Death within 28 days of hospital admission in MI cases defined in I.A
      2. Postmortem findings consistent with MI within 28 days
   B. Probable fatal MI
      1. Death within 28 days of hospital admission in cases defined in I.B
      2. Death within 6 hours of hospital admission with cardiac symptoms and/or signs. Other confirmatory data (biomarkers, ECG) are absent or not diagnostic.
   C. Possible fatal coronary event
      1. Death within 28 days of hospital admission in cases defined in I.C, I.F, and I.G
      2. Postmortem findings show old infarct and/or ≥50% atherosclerotic narrowing of coronary arteries

Ischemic CHD in Developing Countries
The emerging epidemic of ischemic CHD and MI in developing countries requires increased awareness and surveillance in those areas. Definition of the scope, trends, magnitude, and outcomes associated with this epidemic is essential for prevention, diagnosis, and treatment. However, these scarce resource settings in which diagnostic methods are costly, of varied reliability, or at times unavailable. There may be large differences between urban hospitals and rural facilities. Finally, advanced therapies for MI, such as emergency angioplasty or bypass surgery, although practiced in
some settings, are infrequently available, making the newest diagnostic techniques less critical.

Despite these limitations, it is essential to document the prevalence and trends in CHD in developing countries as the epidemic emerges and resource needs grow. The following definitions are recommended:

**Ischemic CHD**

I. Nonfatal events
   A. Criteria for definite, probable, and possible MI identical to those described above. This includes cardiac symptom/sign, biomarker, and ECG findings. Although limitations of available data may result in fewer definite and probable cases, the inclusion of possible cases in the data will allow tracking of overall trends. The use of criteria similar to those in developed countries will also allow comparisons, even though diagnostic testing will be used with different frequency. Older biomarkers, such as total LDH, LDH isoenzymes, and AST (SGOT), are not recommended for criteria. They may be used, however, if more recent markers are unavailable.
   B. Unrecognized MI (as above)
   C. Procedure-related MI (as above)
   D. Unstable angina pectoris (as above)
   E. Stable angina pectoris (as above)

II. Fatal events (in-hospital cases)
   Criteria are identical to those described above. Verbal autopsy, which involves interviews with witnesses and relatives by health workers, may be substituted for traditional postmortem.16

III. Fatal events (out-of-hospital cases)
   Criteria as below. A verbal autopsy may be used as evidence except for category II.C.2.

**Out-of-Hospital Ischemic CHD Death**

Out-of-hospital CHD death is a major public health burden, accounting for 50% to 75% of all fatal cardiovascular disease events in countries in which it is documented.17 It is often unexpected and affects all age, sex, and ethnic groups. Although the immediate mechanism of death is ventricular fibrillation or asystole, the underlying cause is commonly ischemic CHD. Other causes, including nonischemic forms of cardiac disease, are less common.

Classification of out-of-hospital CHD death is usually deficient because of its sudden onset, lack of information from the victim, lack of witnesses, and low autopsy rates. These factors limit the accuracy and extent of classification. Prospective epidemiological studies can provide more pre-event information and may collect relevant data from witnesses and other sources but may still face limited data on the circumstances surrounding the death. At the national level, the only source of information is the death certificate, which usually includes site of death.

Much recent research focuses on arrhythmogenesis and the immediate control of malignant cardiac rhythms. Traditional emphasis on the “suddenness” of the out-of-hospital cardiac event has led to restricted definitions (eg, death within 1 hour of symptom onset) that may exclude most out-of-hospital events. The definitions below broadly consider out-of-hospital death as a general category, with subcategories based on site of death, presumptive cause, and timing. For data collection purposes, the following should be included:

I. Site
   A. Before transport to medical facility (eg, home, work site, street)
   B. During transport (eg, ambulance, car)
   C. Pronounced dead in emergency department and not admitted to hospital

II. Cause
   A. Death certification, based on WHO methods for assigning causation, is the primary source
   1. Validated by postmortem examination, if available
   2. Enhanced by other information, if available (See II.B and II.C below)
   B. Interviews with witnesses and family members
   C. Medical history from healthcare records and physicians

III. Timing
   A. One hour and/or 24 hours since last seen or known to be alive
   B. Other time categories based on research needs

**Classification of Cause of Out-of-Hospital Death (Hierarchical)**

I. Definite fatal MI: Documented definite or probable MI in the previous 28 days and no evidence of a noncoronary cause of death, or autopsy evidence of recent coronary occlusion or MI <28 days old

II. Definite fatal CHD: (1) A history of CHD and/or documented cardiac pain within 72 hours before death and (2) no evidence of a noncoronary cause of death or (3) autopsy evidence of chronic CHD, including coronary atherosclerosis and myocardial scarring

III. Possible fatal CHD: An ICD code (underlying cause) for CHD death (ICD 9: 410 to 414, 427.5, 429.2, and 799; ICD 10: I20 to I25 and I46) and no evidence of a noncoronary cause of death

IV. Cardiac death: When death certificates are the only source of information: ICD 9: 390 to 398, 402, 404 to 429; ICD 10: I00 to I09, I11, I13, I20 to I25, I27, I30 to I52

V. Non-CHD death: Evidence of a noncoronary cause of death

VI. Unclassifiable: Insufficient information to determine whether the death was a CHD death (at any certainty level) or a noncardiac death

**Measuring Trends in Ischemic CHD**

Measurement of ischemic CHD trends in populations is important for several reasons. Among these are whether incidence and prevalence of disease are rising or falling, whether acute care is used appropriately, whether primary and secondary prevention guidelines are applied appropriately, to plan distribution of resources and the provision of medical services, and to detect the effect of changes in disease patterns and treatment. Mortality alone is less adequate for monitoring because of the lack of sensitivity to incidence and changing case fatality for CHD events.
Advances in diagnostic technology, in this case new biomarkers, along with changes in disease presentation make measuring and interpreting trends difficult. Only now is there a realization that changes in cardiac enzyme testing during the 1980s and 1990s presented obstacles to accurate trend estimation. A shift from transaminases and dehydrogenases to isoenzymes of the latter and then to CK, CK-MB, and CK-MB mass improved test precision, resulting in greater sensitivity and specificity in the diagnosis of MI over time and better case classification. Therefore, the trends in incidence and recurrence estimated during this period in areas in which surveillance existed are difficult to interpret without appropriate adjustment.

The advent of even more sensitive and specific measures of myocardial cell damage, troponins, has significant implications for trend analysis (Table 5). Accumulating data suggest that the more sensitive troponin test results in greater rates of MI diagnosis than older markers. Milder and smaller MI will be detected, cases that were earlier classified as nonischemic causes of troponin elevation are documented in many settings. The importance of small troponin increases is confirmed by their use as a sole diagnostic criterion for MI that the more sensitive troponin test results in greater rates of MI diagnosis than older markers. Milder and smaller MI will be detected, cases that were earlier classified as nonischemic causes of troponin elevation are documented in many settings. The importance of small troponin increases is confirmed by their use as a sole diagnostic criterion for MI.

The use of troponin in the United States and elsewhere gradually increased from 1995 onward. However, older biomarkers are still used for MI diagnosis in many hospitals throughout the world. This gradual increase has led to a period in which a changing proportion of CHD is diagnosed by the new, more sensitive biomarkers. That proportion is largely unknown. In addition, there are several generations of troponin assays with different normal ranges and numerous manufacturers. Performance of the assays differs appreciably, and the field is gradually evolving, with improving quality of measurement. It will be some years before these tests are standardized worldwide or even within developed countries.

Although the gradual spread of new diagnostic technology creates a period of transition, the need for accurate trend data remains. Administrative databases and retrospective studies are particularly vulnerable, because they must rely on available data. They are further compromised by changing insurance reimbursement rules based on diagnosis. More accurately interpret recent trends in CHD, the following are recommended:

I. Comparison studies of these recommendations with previous studies and other MI criteria should be performed. This should include different observers who test, evaluate, and validate the current recommendations for observer variation, completeness, and consistency.

II. Surveillance research documenting trends should use prospective methods when possible. These should include overlap methods in which old and new biomarkers are measured simultaneously to determine the effect of the new diagnostic tests. These data will allow the development of adjustment methods for trend analysis. They should be described in the literature to better understand the effects of the biomarker transition.

III. Sentinel research centers should be established to monitor other indicators of acute CHD, including:

A. ECG trends:
   1. Q-wave infarction
   2. ST-elevation and T-wave changes on admission ECG
   3. Trends in infarct size on the basis of biomarkers or hemodynamic or clinical indicators

D. Trends in infarct size on the basis of biomarkers or hemodynamic or clinical indicators

E. Simultaneous use of older biomarkers

### Table 5. Studies of CK-MB and Troponin Comparison

<table>
<thead>
<tr>
<th>Reference</th>
<th>n</th>
<th>Characteristics</th>
<th>Positive CK-MB, n (%)</th>
<th>Positive Troponin, n (%)</th>
<th>% Troponin, Troponin/CK-MB</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>801</td>
<td>Acute myocardial ischemia admissions</td>
<td>216 (27)</td>
<td>289 (36)</td>
<td>+34%</td>
</tr>
<tr>
<td>19</td>
<td>292</td>
<td>Possible myocardial ischemia in emergency ward</td>
<td>15 (5)</td>
<td>34 (12)</td>
<td>+127%</td>
</tr>
<tr>
<td>*</td>
<td>14,777</td>
<td>MI discharge diagnosis plus biomarker, ECG, and pain algorithm</td>
<td>4157 (28)</td>
<td>4661 (32)</td>
<td>+12%</td>
</tr>
<tr>
<td>33</td>
<td>1719</td>
<td>All ACS admissions</td>
<td>373 (22)</td>
<td>430 (25)</td>
<td>+15%</td>
</tr>
<tr>
<td>34</td>
<td>80</td>
<td>All ACS admissions except with diagnostic ECG of MI</td>
<td>23 (29)</td>
<td>32 (40)</td>
<td>+39%</td>
</tr>
<tr>
<td>35</td>
<td>798</td>
<td>ACS admits to cardiology service</td>
<td>189 (23)</td>
<td>228 (28)</td>
<td>(21%)</td>
</tr>
</tbody>
</table>

ACS indicates acute coronary syndrome.

*Personal communication (e-mail), ARIC Executive Committee (August 27, 2003).*
IV. Administrative databases and retrospective studies seeking to determine incidence from prevalence and trends may consider adjustment factors before and after troponin-derived research studies. However, the use of adjustment factors must consider several important caveats, including:

A. The variability in the use and spread of the new biomarkers in hospitals
B. The evolution of the methodology and quality control for new biomarkers
C. The relative paucity of overlap studies using new and old biomarkers simultaneously
D. The sensitivity of available adjustment factors to selection characteristics for patient entry (Table 5)

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