Creatine Kinase and Creatine Kinase B-Subunit Activity in Serum in Cases of Suspected Myocardial Infarction

W. Gerhardt, J. Waldenström, M. Hörder, S. Hofvendahl, R. Billström, R. Ljungdahl, H. Berning, and P. Bagger

We evaluated a diagnostic strategy by studying 481 patients suspected of having had an acute myocardial infarction; the prevalence of infarction by independent criteria was 0.43. This strategy is based on the sequential application of: (a) clinical criteria; (b) total creatine kinase determinations in two serum samples drawn within 10 to 20 h of the onset of acute symptoms; and (c) creatine kinase B-subunit (S-CK B) determinations after immunoinhibition with antibodies to creatine kinase M-subunit in the reaction medium in all samples found to have increased total creatine kinase activity. Discrimination limits of 150 U/L total creatine kinase for women and 200 U/L for men gave a diagnostic sensitivity of 0.99. Activities less than these limits in samples identified 98% of the 274 non-infarct cases (posterior probability of a negative result of 0.99) within 20 h. Subsequent determination of S-CK B in 282 patients who were positive by the discrimination limits for total creatine kinase verified myocardial infarction in 99% of 207 cases for which S-CK B exceeded the discrimination limit of 12 U/L. The strategy excluded 98% of all non-infarct cases at a posterior probability of 0.99.

Additional Keyphrases: enzyme activity - immunoinhibition - isoenzymes - electrophoresis, agarose gel

Rational utilization of enzyme tests, such as measurement of S-CK and S-CK B subunit activity, for the early exclusion and diagnosis of AMI requires a strictly defined diagnostic strategy based on sampling in the time interval in which S-CK and S-CK B are most likely to be increased. Several studies have demonstrated that this is 10 to 20 h after the onset of acute symptoms (1–7). It follows that earlier emergency determinations of these enzymes carry a considerable risk of falsely negative results (8), and in fact, may instill a false feeling of security in the clinician, leading to erroneous decisions. Because AMI can be definitely excluded by means of these enzyme tests only in the 10- to 20-h interval after onset of symptoms, it is rational to obtain samples only within this time period.

On the other hand, S-CK and S-CK B may be increased in some patients earlier than 10 h after onset of symptoms. However, it would appear irresponsible to deny a patient adequate care until a laboratory verification of an AMI has been obtained, especially because the risk of dangerous arrhythmias is greater during the initial phase of an AMI. Under Scandinavian hospital policy a patient suspected of having an AMI is rapidly transferred to the best possible monitoring facility, preferably a coronary care unit, and remains under supervision until an AMI can be definitely disproved.

We have previously described an analytically specific routine method for S-CK B determinations with use of the Scandinavian-recommended (SCE) CK reagent and complete immunoinhibition of CK M-subunit activity (9). As a working group of the Scandinavian Committee on Enzymes we here evaluate a three-stage diagnostic strategy comprising the sequential application of clinical criteria, 10- to 20-h S-CK, and S-CK B measured in all S-CK positive samples from 481 AMI-suspect patients.

Materials and Methods

Patients

We studied a total of 481 AMI-suspect patients admitted to our emergency ward and coronary care unit during 1977–1978 and part of 1979. The patients included in the study all presented with symptoms indicative of their having had an AMI (10) within the previous 24 h. Most had localized or irradiating chest pain lasting more than 20 min and often characterized by nonresponsiveness to nitroglycerin. Some presented with pulmonary edema or cardiogenic shock but no chest pain. A few patients had atypical symptoms, including cardiac failure, cardiac arrest, or severe arrhythmias with or without a history of previous coronary disease, indicating the possibility of an AMI.

Of the AMI suspect patients 85% were transferred to the coronary care unit upon admission. The prevalence of AMI (by independent criteria, see below) in this group was 0.47. For various reasons, such as only very slight suspicion of AMI, other severe complicating disease, or extreme old age, about 15% of the cases remained in the general-observation ward. The prevalence of AMI in this group was 0.15. The prevalence of AMI in the total patient population was thus 0.43.

Sampling

S-CK and S-CK B: From each patient two specimens were drawn within 10 to 20 h after onset of the acute symptoms, preferably after 10 and after 16 h. The samples were centrifuged within 1 h of collection. Samples not analyzed within about 2 h after centrifugation were stored at −80 °C until the next series was run.

S-ASAT, S-ALAT, and S-LD: Specimens for use in these assays were drawn daily for three days after admission.
Methods

S-CK and S-CK B were determined with use of the jointly developed CK method of the Scandinavian (SCE) and German Societies of Clinical Chemistry (11, 12) with and without the immunoinhibitor, antibody to creatine kinase M-subunit (9), at a reaction temperature of 37 °C (11, 9). Discrimination limits for S-CK were 150 U/L for women, 200 U/L for men (9). S-CK B results were corrected for residual adenylate kinase (EC 2.7.4.3) activity in serum as previously described (9, 13). The results were reported as S-CK B subunit activity and were not multiplied by the factor two for expressing them as "CK MB" activity (9, 13). The discrimination limit for S-CK B was 12 U/L (4, 8). Qualitative isoenzyme electrophoreses were carried out in agarose gel for CK isoenzyme identification (by fluorescence, 14) and LD isoenzyme estimation (by tetrazolium stain, 15) at or around peak S-CK or peak S-LD activity, respectively.

S-ASAT, S-ALAT, and S-LD activities were determined at 37 °C according to the SCE methodology (16), upper reference limits being 40, 40, and 450 U/L, respectively.

No "stat" analyses were done. The two prerequisites 10- to 20-h samples from each patient were determined batchwise in the laboratory. S-CK series were run three times a day at about 0600 and 0900 hours and at about 1500 hours. The results were reported to a decision-making clinician for the morning and afternoon rounds. S-CK B series were run every morning, and results were available during the morning round.

Classification into AMI and Not-AMI

Independently of the S-CK and S-CK B results, patients were classified into AMI and not-AMI groups according to the WHO diagnostic criteria for myocardial infarction: the patient's typical or atypical history, changes in the standard 12-lead electrocardiogram, and S-ASAT and S-LD isoenzyme data (10). All doubtful cases were further studied by CK isoenzyme electrophoresis (14).

Evaluation Terminology

Because no internationally accepted terminology yet exists, we define the concepts we used as follows:

For a quantitative analysis the likelihood or "odds" for AMI is a function of the numerical test value (17). The quantitative S-CK and S-CK B data were plotted in histograms, in classes of 50 and 12 U/L, respectively. We define odds for AMI as the fraction of patients having AMI out of all patients within each class of enzyme values. Odds for not-AMI is defined as the fraction of patients not having AMI out of all cases within the same classes of enzyme values. These odds may vary between 0.0 and 1.0. Because odds for AMI and odds for not-AMI are complementary fractions, they can be represented by a single "odds" line in histograms. The discriminator values were selected by guidance of the odds functions in the histograms (see relevant Figures below).

The enzyme data were evaluated retrospectively. Consequently, for the dichotomized data we have substituted posterior probability for the suggestive term "predictive value" (18). Mathematically, the two fractions are identical. The concepts we used were defined as follows:

- **diagnostic sensitivity**: the fraction of true-positive results out of all AMI patients.
- **diagnostic specificity**: the fraction of true-negative results out of all not-AMI patients,
- **posterior probability of positive results** (PP<sub>pos</sub>): the fraction of true positives out of all positive results,
- **posterior probability of negative results** (PP<sub>neg</sub>): the fraction of true negatives out of all negative results.

Results

Temporal Characteristics of S-CK and S-CK B

Examination of several enzyme time-curves from AMI patients showed that both S-CK and S-CK B approach their respective peak activities within 10 to 20 h after onset of the acute symptoms (cf. 1-9).

We then calculated the likelihood of increased S-CK and S-CK B as a function of time after the acute episode in 55 AMI cases in which the time of onset of symptoms could be established within one-half hour (Figure 1). The likelihood of obtaining a false-negative result 2 to 4 h, and 4 to 6 h after onset of the acute symptoms was 70% and 25%, respectively. Even after 6 to 8 h the likelihood of a false negative was at least 5%.

In contrast, all 55 AMI cases had increased S-CK and S-CK B activities by 10 to 20 h after onset of acute symptoms. From this we concluded that absence of increased S-CK or S-CK B activity in both the 10- to 20-h samples would exclude AMI.

Fig. 2. 10- to 20-h S-CK in 300 AMI-suspect men (AMI prevalence 0.44)

The histograms in Figs. 2, 5, and 6 are all plotted in the same manner: Upper panel: the frequency of AMI patients (ordinate) plotted as a function of enzyme activity in classes of 50 U/L (S-CK) or 12 U/L (S-CK B) (abscissa). Lower panel: The frequency of not-AMI patients plotted as a function of enzyme activity. Evaluation as quantitative tests: odds for AMI (ordinate) are plotted as a function of enzyme activity (abscissa), the fraction AMI patients/all patients within each class of enzyme activity. Odds for AMI and odds for not-AMI are represented by a single "odds" line (heavy line). Evaluation as dichotomous tests: The selected discriminator is shown as a vertical, dashed line. Sensitivity and specificity are given in the legends. PP<sub>pos</sub> and PP<sub>neg</sub> are given in the legends.

The histogram in Fig. 2 presents the higher of the two 10- to 20-h S-CK results in 300 men with suspected AMI (prevalence of AMI 0.44). With the shown discriminator at 200 U/L, PP<sub>pos</sub> was 0.99, PP<sub>pos</sub> 0.70.
with a very high degree of certainty. Consequently, we restricted sampling to two samples within this time interval, preferably at about 10 and about 16 h after the acute episode. Sampling outside of this time interval was discouraged.

10- to 20-h S-CK, Men

S-CK determinations were requested for 300 AMI-suspect men (AMI prevalence 0.44). The higher of the two 10- to 20-h values was plotted in the histogram (Figure 2), and the “odds” line was inserted in the graph.

Odds for AMI showed an erratic course with increasing S-CK, fluctuating between 0.5 and 1.0 depending on the sporadic occurrence of not-AMI patients with increased S-CK from extracardial causes. Odds for not-AMI increased to 0.96-1.00 for S-CK <200 U/L; i.e., if both 10- to 20-h S-CK values were <200 U/L, the likelihood of not-AMI was between 0.96 and 1.00.

Consequently, we selected a discriminator value of 200 U/L for men to obtain a high diagnostic sensitivity of 0.99—at the cost of a low (0.67) diagnostic specificity.

Conclusion: At an AMI prevalence of 0.44 and a discriminator value of 200 U/L, both S-CK results being negative identified 67% of all male not-AMIs, with a posterior probability of 0.99. All the positive results, including 31% false positives, required subsequent discrimination between AMI and not-AMI by S-CK B determination on these samples the following morning.

10- to 20-h S-CK, Women

S-CK determinations were requested for 181 AMI-suspect women (AMI prevalence 0.41). Odds for not-AMI increased to 0.97-1.0 for values below 150 U/L (histogram in ref. 8). Consequently, we selected a discriminator value of 150 U/L, giving a diagnostic sensitivity of 0.99—at the cost of a low (0.71) specificity.

Conclusion: At a prevalence of AMI of 0.41 and a discriminator value of 150 U/L, both S-CK results being negative identified 71% of all not-AMI in women with a posterior probability of 0.99. All the positive results, including the 29% false positives, required subsequent S-CK B determinations.

10- to 20-h S-CK, Both Sexes

In summary, 187 (68% of all the not-AMI cases) were designated as not-AMI by two negative 10- to 20-h S-CK with a PP_neg of 0.99. In addition, we had two false-negative patients.

The other 292 patients (61% of all AMI-suspects) were S-CK positives, with a PP_pos of 0.70. This, consequently, was the prevalence of AMI in the group on which S-CK B was subsequently determined.

S-CK B Discrimination Limit

S-CK B was studied as a function of time in 36 patients with angina pectoris classified as not-AMI by the independent WHO criteria above plus negative scintigraphy. The “worst case” day-to-day analytical imprecision (9) is shown in Figure 3 at 9 U/L ± 15% (CV). Close to the discrimination limit it is 12 U/L ± 10% (CV) (9). The major part of the observed fluctuations at the lowest activity may be ascribed to signal noise. The two highest values ± 2 SD did not reach the discriminator value, 12 U/L. Re-examination of this patient did not reveal any indications of graver symptoms than the others. Observe the distance from the upper reference limit to the discrimination value. Even in the “worst case” situation there is a very small probability of an analytical error’s causing a not-AMI value to fall above the discriminator.

The respective merits of an “absolute” discriminator in U/L vs a “relative” S-CK B expressed as a fraction of total S-CK are still being discussed (5). In Figure 4 we have plotted S-CK B activity vs total S-CK in 104 AMI-suspect patients, 93 classified as AMI and 11 cases of electrophoretically verified (14, 19, 20) S-macro BB (IgG-CK BB complexes) in not-AMI patients. The relative discriminator, 3% S-CK B (=6% CK MB), is drawn. With our analytically specific S-CK B method corrected for sample residual adenylate kinase activity (9), the 3% discriminator resulted in 7% false negatives out of these 93 AMI cases. In contrast, our selected discriminator of 12 U/L gave no false negatives or false positives in this population.

Recognition criteria for "macro" BB. In 10 of the 11 cases (90%) with “macro" BB, S-CK B constituted between 12% and 57% of total S-CK activity. These 10 cases fulfilled the previously established recognition criteria for “macro" BB (8, 14): a relatively high and stationary S-CK B activity in the two 10- to 20-h samples, constituting more than 10% of a normal or slightly increased total S-CK activity. About 90% of our cases were readily identified by both clinicians and the laboratory by the recognition criteria given above. S-"macro" BB
may be estimated to contribute 0.5% false positives (see Discussion).

The definition of a positive S-CK B result was thus extended: S-CK B activity >12 U/L in the absence of criteria for S-"macro" BB activity.

10- to 20-h S-CK B

On the basis of cost–benefit considerations, one of two strategies had to be selected: A, S-CK B determinations on samples from all patients (481 AMI-suspects) or B, S-CK B determinations only on S-CK-positive samples (292, or 61% of all AMI-suspects).

We present the data from both strategies so that the reader may evaluate the respective consequences.

S-CK B in strategy A (Figure 5). Odds for AMI increased to 0.84–1.00 with S-CK activities >12 U/L, and odds for not-AMI to 1.00 at S-CK <12 U/L. In accordance with previous studies (4) we selected a discriminator value of 12 U/L.

Diagnostic sensitivity was 0.99 (206 true pos./207 AMI)

Diagnostic specificity: A total of 19 not-AMI cases (bottom half of Figure 5) showed S-CK B activities >12 U/L, which would give a diagnostic specificity of 0.93 (255 true neg./274 not-AMI). However, 11 of these had S-"macro" BB (prevalence 2.3% in the total population). Ten of these were directly recognized by the criteria demonstrated above and could thus be transferred to the S-CK B-negative group (Figure 5). The remaining eight false positives were distributed as follows: two cases of cardiac arrest with transitory S-CK BB from brain ischemia; one case of a cardiac myoma with a fatal cerebellar hematoma, one Prinzmetal variant angiina, considered a borderline case against AMI; and four cases of shock with transitory S-CK BB.

The diagnostic specificity was thus calculated as 0.97 (265 true neg./274 not-AMI). PP_{pos} was 0.96 and PP_{neg} was 0.99.

S-CK B in strategy B (Figure 6). Odds for AMI increased to 0.91–1.00 for >12 U/L and odds for not-AMI to 1.00 for <12 U/L.

Fig. 5. S-CK B histogram for strategy A: S-CK B in samples from all 481 AMI-suspect patients (prevalence of AMI 0.43)

The higher S-CK B value in each case has been plotted. Of the 207 AMI cases, two false-negative S-CK results are marked on top of column 2 (12–24 U/L); with one false-negative S-CK B result, diagnostic sensitivity was 0.99. Of the 274 not-AMI cases, 19 had S-CK B activity greater than the discriminator 12 U/L; 11 were S-"macro" BB, of which 10 (broken-line boxes) fulfilled the recognition criteria given in the text and were transferred to the true negatives, leaving one S-"macro" BB as a false-positive. Eight false positives had S-CK BB (see text), giving a total of nine false positives and a diagnostic specificity of 0.97. PP_{pos} was 0.96, PP_{neg} 0.99

Fig. 6. S-CK B histogram for strategy B: S-CK B in samples from 292 cases with increased S-CK (prevalence of AMI 0.70)

The higher S-CK B value in each case has been plotted. Of the total of 207 AMI cases the two false-negative S-CK (Fig. 5) did not appear in strategy B. With one false-negative out of the remaining 205 AMI cases, diagnostic sensitivity for S-CK B was 0.99. S-CK B was determined on 87 not-AMI cases with increased S-CK.

Of the nine false-positive S-CK B (Fig. 5) only five with increased S-CK remained, giving a diagnostic specificity of 0.94. PP_{pos} was 0.99, PP_{neg} was 0.98

Diagnostic sensitivity was 0.99 (204 true pos./207 AMI); two of the total of 207 AMI cases were false S-CK negatives.

Diagnostic specificity: Of the initial total of 19 not-AMI with S-CK B >12 U/L, only eight who also had increased S-CK appeared in strategy B. These eight cases were: four S-"macro" BB and four of the S-CK MB and BB described above. Three of the four "macro" BB cases were directly identified by the recognition criteria, leaving one false-positive "macro" BB.

As compared with strategy A the number of false positives was thus reduced from nine to five (Figures 5 and 6). However, the number of true negatives was reduced even more, because only 61% of all not-AMI cases showed increased S-CK. Consequently, the diagnostic specificity became lower than for strategy A, viz., 0.94 (82 true neg./87 not-AMI with increased S-CK). PP_{pos} was 0.98, PP_{neg} was 0.98.

The performance of strategy B, calculated on the total material of 481 AMI-suspects, prevalence of AMI 0.43, is summarized in Table 1.

Function of the Selected Diagnostic Strategy in Suspected AMI

The selected diagnostic strategy is based on the sequential application of (a) clinical criteria, (b) S-CK on two 10- to 20-h

Table 1. Performance of Strategy B Calculated on the Total Population of 481 AMI Suspects (Prevalence of AMI 0.43)

<table>
<thead>
<tr>
<th>AMI</th>
<th>No. negative</th>
<th>No. positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>207 AMI</td>
<td>3</td>
<td>204</td>
</tr>
<tr>
<td>274 not-AMI</td>
<td>269</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total 481</th>
<th>PP_{neg} =</th>
<th>PP_{pos} =</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.99</td>
<td>0.98</td>
<td></td>
</tr>
</tbody>
</table>
The prevalence of AMI at each stage is shown in the left column, the fraction of all not-AMI excluded at each stage, in the right column. The inserted figures correspond to those actually found in the described population of 481 AMI-suspects. The results for any other prevalence of AMI in the initial group of AMI-suspects may easily be calculated by setting the number of patients at, e.g., 1000 and inserting the local prevalence. For instance, a prevalence of 0.20 in the initial group would give 200 AMI, of which 198 (99%) would be true S-CK-positives, and 800 not-AMI, of which 400 (50%) would be false S-CK-positives. Accordingly, prevalence of AMI in the "S-CK B" group would be 0.48 (198 AMI/408 AMI suspects).

Discussion

Material and Biological Sources of Error

The present study was concerned exclusively with the differential diagnosis of AMI vs not-AMI. The material contained only AMI-suspect patients. Consequently, certain categories of patients with S-CK BB or extracardial S-CK MB described in other types of materials did not appear as sources of error in this study. A survey of such other conditions may be found in ref. 21. The reader should be reminded that some early studies (22, 23) describing S-CK MB in some acute conditions were done with a glutathione-activated, one-vial immuno-inhibition test (CK MB1-test), which did not contain the adenylate kinase inhibitor P1,P2-dil(adenosine-5')pentaphosphate (24-28) and had no possibility of a separate determination of sample residual adenylate kinase activity (9, 13, 29). Accordingly, the results of such studies should be interpreted with great caution. Some of these early studies appear to be a main reason for the German selection of the relative discriminator value of 6% CK MB (21-28).

In contrast, we have observed no false-positive S-CK B results attributable to skeletal muscle CK MB with our residual adenylate kinase-corrected S-CK B assay in the present material. In other materials we have observed S-CK B activities exceeding 12 U/L in patients suffering from various collagenoses, severe skeletal muscle damage, and CO-intoxication, among other conditions.

The correction for sample residual adenylate kinase allows S-CK B to be determined in hemolyzed samples.

The main source of false positives, S-"macro BB," occurs at a prevalence of about 3% of all patients (Stein, personal communication) and in as many as 5% of elderly women. However, about 90% of such cases can be readily detected by the recognition criteria described. We further identified all "macro" BB cases by isoenzyme electrophoresis. Most can also be identified by the simple thermostability test described by Bohner et al. (30), viz., S-CK B determination before and after heating the serum at 45 °C for 20 min. Residual S-CK B activity >55% indicates the presence of thermostable "macro" BB activity.

In the present study only four of the 11 observed S-"macro" cases had increased S-CK, and consequently were detected by S-CK B determination in the selected strategy. Three of them were identified by the criteria above. Consequently, occurrence of S-"macro" BB as a source of error was less than 0.5% (8).

Evaluation of Diagnostic Strategy in Cases of Suspect AMI

S-CK and S-CK B are quantitative analyses. Odds express the likelihood of AMI or not-AMI as a function of the numerical test values in the particular material investigated (see also 17). They are thus retrospective estimates. Whether these are prospectively applicable to a new material depends on the similarity between the two materials.

To emphasize the nature of estimates, we have chosen to call these likelihood fractions "odds." The odds line confirms, e.g., in the case of S-CK B, the clinician's intuitive feeling that a highly increased value is more significant than a slightly increased one, and contradicts it, e.g., in the case of S-CK for men in which odds for AMI fluctuate with increasing S-CK, depending on the sporadic occurrence of not-AMI cases with high S-CK values from extracardial causes.

In the present study we have used the odds functions as guidelines for selecting discriminator values for S-CK and S-CK B.

Selection of a discriminator value transforms the quantitative test into a dichotomous test: negatives and positives. This permits calculation of diagnostic sensitivity and specificity and of the posterior probabilities, PPpos. and PPneg (18). Sequential application of tests entails an increase in prevalence and specificity and a decrease in sensitivity (31).

This has been utilized in the selected three-stage, sequential diagnostic strategy (Figure 7) as follows:

1. From patients admitted to the emergency ward of the hospital the admitting physician selects a group as AMI-suspects. The prevalence of AMI in this initial group depends exclusively on this physician's decision criteria. An experienced cardiologist may note a higher prevalence of AMI in this group than a novice.

The admitting physician establishes the time of onset of acute symptoms as accurately as possible and requests that two S-CK samples be collected about 10 and about 16 h after this time. In the case of admission later than 10 h after onset
of symptoms, one sample is taken at admission and the next before 20 h. A practical aid in implementing this system has been the use of preprinted "AMI Enzyme Forms" containing instructions for the physician and spaces for 10- and 16-h S-CK activities other enzyme data, electrocardiograph results, and other details (this form may be requested from the authors).

In our material the prevalence of AMI in the initial group was 0.43. Recalculation of the data in the flow diagram for any other prevalence is described in the legend to Figure 7.

2. Prevalence of AMI in the S-CK groups was 0.44 for men and 0.41 for women. Consideration of odds in 10- to 20-h S-CK led to the conclusion that no matter which discriminator value one might select, S-CK remained a poor tool for verification of AMI. In contrast, S-CK appeared to be an excellent tool for the early exclusion of AMI. Accordingly, we selected a low discriminator value to obtain a high diagnostic sensitivity, at the cost of a low diagnostic specificity. We could therefore exclude 68% of all the not-AMI with a PP_{pos} of 0.99 within 20 h after onset of symptoms, in contrast to our earlier routine procedure, which required two to three days of observation with daily aminotransferase assays.

3. PP_{pos} in the 292 S-CK positives was 0.70, which thus was the prevalence of AMI in the group in which S-CK B was determined (see Figure 7). The diagnostic specificity increased from 0.68 in the CK (only) group to 0.94 in the CK B group, and was 0.98 for the strategy calculated on the total data (Table 1). Sensitivity decreased very slightly from 0.990 to 0.996, owing to the loss of three AMI cases that were false negatives: two of them in the S-CK determinations, the last one in the S-CK B determination (Table 1).

As the patients with suspected AMI were taken through the stages of the selected diagnostic strategy, the prevalence of AMI increased from 0.43 to 0.70. A prevalence of AMI as low as 0.20 in the initial AMI-suspect group will still increase to 0.45 at the S-CK B stage. Posterior probabilities (and "predictive values") are highly dependent on prevalence (18); in other words, S-CK B results from a future application of the selected diagnostic strategy on a similar material of AMI suspects from an emergency ward with a prevalence of AMI as low as 0.20 will function with the same reliability as if S-CK B had been directly applied to a population from a coronary care unit, where the prevalence of AMI is about 0.5.

Today many methods for S-CK and S-CK B (MB) are used, each with its analytical sensitivity, specificity, and reference range (surveys in 22, 33). In contrast, the establishment of the SCE in 1970 has led to the implementation of SCE-recommended methods in more than 90% of medium and large-sized Scandinavian hospital laboratories (34). This has resulted in considerable improvement in interlaboratory variation (34).

The present study is a first attempt to recommend measures to improve a rational utilization of enzyme data in clinical practice.

References


