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Detection of acute myocardial infarction using the 12-lead ECG plus inverted leads versus the 16-lead ECG (with additional posterior and right-sided chest electrodes)

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Short title: 24-lead and 16-lead ECG in the diagnosis of MI

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SUMMARY

Background: The electrocardiographic (ECG) diagnosis of acute myocardial infarction (MI) should be improved. This might be done either by regarding all 24 aspects (both positive and negative leads), or a subset hereof (for example 19-lead ECG), of the conventional 12-lead ECG or by using additional electrodes. The purpose of the study was to investigate the accuracy of the different ECG methods in diagnosing acute ST-elevation MI.

Methods: The study population consisted of 479 patients admitted to Lund University Hospital with acute chest pain. One conventional ECG plus leads V4R, V5R, V8, and V9 were recorded for each patient within 24 hours of admittance. Biochemical markers were used as the "gold standard" for diagnosis of MI. We measured ST-segment elevations in the 12-lead, 16-lead, and 24-lead postadmission ECGs as well as in the 12-lead, 19-lead and 24-lead admission ECGs.

Results: The sensitivity for detecting acute MI was 28% for the postadmission 12-lead ECG, 33% for the 16-lead ECG, and 37% for the 24-lead ECG. The specificities were 97%, 93%, and 95%, respectively. For admission ECGs, the sensitivity was 33% for the 12-lead ECG, 45% for the 19-lead ECG and 49% for the 24-lead ECG, with specificities of 97%, 96%, and 94%, respectively.

Conclusions: The sensitivity for detecting acute MI was higher for the 16-, 19- and 24-lead ECGs than for the conventional 12-lead ECGs. Their specificity, however, was slightly lower. If increased sensitivity for detecting MI is desired, the 24-lead or 19-lead should be used since no additional electrodes are required.

Key words: 16-lead ECG, 19-lead ECG, 24-lead ECG, acute myocardial infarction, acute coronary syndrome

INTRODUCTION

Conventional 12-lead electrocardiography (ECG) is the most widely used method to analyze ECG changes in the diagnosis of acute myocardial infarction (MI). Anterior MI due to occlusion of the left anterior descending coronary artery is diagnosed with high certainty with this method (sensitivity 90%, specificity 95%), whereas diagnosis of MI due to occlusion of the right coronary artery or the circumflex coronary artery is less certain (sensitivity 53%, specificity 98%) (Blanke *et al.*, 1984).

Acute transmural ischemia caused by occlusion of a coronary artery produces an epicardial injury current that can be detected as a deviation of the ST segment toward the involved region (Holland & Brooks, 1977). Traditionally, the ECG diagnosis of acute MI is established by ST-segment elevation. However, acute occlusion of the circumflex artery is typically represented by ST-segment depression in leads V1-V3 (Shah et al., 1997), but guidelines recommend that patients with ST-segment depression should have this finding on "two consecutive ECGs at least several hours apart" and should have "imaging studies performed to confirm the presence of infarction" before thrombolytic treatment is administered (Alpert *et al.*, 2000). It has been debated whether patients with acute coronary syndrome benefit from an early invasive treatment or not, but the prospective, randomized FRISC II study (FRISC II, Lancet 1999) concluded that early invasive approach

should be the preferred strategy in patients with unstable coronary artery disease who have signs of ischemia on ECG or raised biochemical markers of myocardial infarction.

To observe ST-segment elevation in patients with acute posterior MI, one would have to either apply posterior thoracic electrodes or consider a "24-lead ECG," in which negative counterparts of all standard leads are also regarded (Figure 1) (Pahlm-Webb et al., 2002; Sadanandan et al., 2003; Wagner et al., 2006). The 24-lead ECG has the potential to identify the ST-segment elevation typical of acute coronary occlusion in patients for whom the 12-lead ECG shows only ST-segment depression. One alternative to the 24-lead ECG is to invert only some of the leads, which has been suggested by Perron et al (Wagner et al., 2006; Perron A, personal communication)]. In their study, the 19-lead ECG, with leads −V1, −V2, −V3, −aVL, −I, +aVR, and −III in addition to the standard 12 leads, had the highest sensitivity without a large decrease in specificity.

Leads V7-V9 have been shown to diagnose true posterior MI in some instances when standard leads cannot (Zalenski et al., 1993; Casas et al., 1997; Matetzky et al., 1999). Zalenski et al. (1993) found that the addition of leads V4R, V8, and V9 in patients with a provisional diagnosis of MI or unstable angina increased the sensitivity of ST-segment elevation detection from 47.1% to 58.8%, with no loss of specificity. In a study of low-risk patients with chest pain suggestive of acute coronary syndromes, however, posterior leads were not found to increase the detection rate of ischemia (Ganim et al., 2004).

Right ventricular infarction rarely occurs in isolation but often occurs with inferior MI (Andersen et al., 1987a). Its recognition has important therapeutic and prognostic implications. ST-segment elevation in right-sided chest leads (V3R to V5R) has very high sensitivity, specificity, and positive predictive value for detection of right ventricular involvement (Braat et al., 1983; Robalino et al., 1989; Andersen et al., 1990). Further, patients with mid-level or proximal occlusion of the right coronary artery often show ST-segment elevation in leads V4R and V5R as well as ST-segment depression in leads I, aVL, and V5-V6, and such patients often are not considered to have MI based on the 12-lead ECG (Liu et al., 2005).

The ECG diagnosis of acute MI clearly can be improved, especially when the right ventricle or the posterior wall of the left ventricle is involved. This could be achieved either by assessing 19 or 24 leads of the ECG or by using additional electrodes. The purposes of the present study were to investigate the performances of the different ECG methods (16-lead, 19-lead, and 24-lead ECG) in diagnosing acute ST-segment elevation MI and to compare their performances with that of the 12-lead ECG.

METHODS

Study population

The study population consisted of 575 consecutive patients admitted to Lund University Hospital from May 2004 to March 2005 with acute chest pain. Using a 16-lead digital ECG recorder (HP XLi, Philips Medical Systems, Thousand Oaks, CA, USA), 1 12-lead ECG plus leads V4R, V5R, V8, and V9 were recorded simultaneously on each patient within 24 hours after admission (postadmission ECGs). The ECGs were recorded while patients were in the cardiac catheterization laboratory, the intermediate-care unit, or the coronary-care unit. Patients with a pacemaker, preexcitation, left

ventricular hypertrophy, or left or right bundle branch block were excluded from the study due to the potential for confounding results. Left ventricular hypertrophy was estimated using the Cornell product (Molloy *et al.*, 1992). After exclusions, the study population consisted of 479 patients.

In a second analysis, only patients with acute MI established by biochemical markers who did not receive acute thrombolysis or percutaneous coronary intervention were considered (in total, 43 patients). In a third analysis, all 12-lead, 19-lead, and 24-lead admission ECGs from routine clinical admissions were analyzed. Only patients with a digitally stored ECG available were considered for this analysis. In this subpopulation, 377 patients remained after exclusions (the same exclusion criteria as above).

The study was performed in accordance with the principles of the Declaration of Helsinki, and written informed consent was obtained from each subject before enrolment.

ECG analysis

The 12-lead ECG was regarded as indicative of acute MI if it showed new or presumed new ST-segment elevation at the J point in 2 or more spatially contiguous leads with thresholds of 0.2 mV in leads V1, V2, or V3 or 0.1 mV in other leads. Contiguity in the transverse plane was defined by the lead sequence V1–V6 and, in the frontal plane, by the lead sequence aVL, I, −aVR, II, aVF, and III (Alpert *et al.*, 2000). This panoramic sequence is recommended by a Joint European Society of Cardiology/American College of Cardiology Committee and is also used routinely in Sweden. The 16-lead ECG was regarded as indicative of acute MI if the 12-lead ECG fulfilled the criteria listed above or if ST-segment elevation >0.05 mV was present in at least 1 of the following leads: V4R, V5R, V8, or

V9 (Andersen *et al.*, 1987b). The 24-lead ECG was regarded as indicative of acute MI if there was new or presumed new ST-segment elevation at the J point in 2 or more spatially contiguous leads with cutoff points as described above for standard leads. The ST-segment elevation thresholds for the inverted leads were set at 0.05 mV in leads −V2 and −V3 and 0.1 mV in the other leads, based upon observations made in normal subjects (Macfarlane, 1989). The 19-lead ECG (standard 12-lead ECG plus −V1, −V2, −V3, −aVL, −I, +aVR, and −III) was regarded as indicative of acute MI on the same basis as for 24-lead ECG.

Biochemical markers

Blood samples for creatine kinase-MB isoenzyme (CK-MB) and troponin T (TnT) were obtained as clinically indicated. The thresholds for biochemical markers for diagnosis of acute MI were CK-MB >10 µg/L in 1 sample or >5 µg/L in 2 consecutive samples and TnT >0.05 µg/L in 2 consecutive samples, with TnT levels taking precedence over CK-MB results. An evolution in biochemical markers was required (i.e. not persistent elevation). Three patients lacked TnT assay results; CK-MB alone was used to identify MI in these patients.

Statistical methods

The sensitivity and specificity were calculated for the 12-lead ECG (admission and postadmission ECGs), 16-lead ECG (postadmission ECGs), 19-lead ECG (admission ECGs), and 24-lead ECG (admission and postadmission ECGs), using the TnT/CK-MB results as the gold standard. The McNemar test (a variation of the χ^2 test adapted for matched proportions) was applied to examine the observed differences in sensitivity and specificity between the sets of ECGs. Sensitivity and specificity were tested in

pairs. The level of significance was established at p<0.05. Positive predictive values [true positive/ (true positive + false positive)] for the different ECGs at a hypothesized prevalence at 0.2 were also calculated. All statistical analyses were performed using SPSS for Windows, version 11.5 (SPSS Inc, Chicago, IL, USA).

RESULTS

All patients

Study population

The study population consisted of 479 patients. Of these, 242 had acute MI and 237 had no acute MI, according to biochemical markers. Of the 242 MI patients, 163 (67%) were male. The mean age was 67.8 years (range, 18 to 94 years). All but 31 patients (87%) had undergone coronary angiography either before or after the postadmission ECG. Two hundred and five patients underwent either percutaneous coronary intervention or bypass surgery (85%).

Of the 237 non-MI patients, 133 (56%) were male, and the mean age was 63.4 years (range, 23 to 90 years). Of the non-MI patients, 43 (18%) were examined angiographically, and 30 patients (13%) underwent either percutaneous coronary intervention or bypass surgery because of significant stenosis.

Sensitivity and specificity

For the 12-lead ECG, the sensitivity for detecting acute MI was 28.1%. For the 16-lead ECG, the sensitivity was 33.1%; and for the 24-lead ECG, 36.8%. The specificity for detecting acute MI was 97.0% for the 12-lead ECG, 93.2% for the 16-lead ECG, and 94.5% for the 24-lead ECG (Figure 2).

The difference in sensitivity was statistically significant between the 12-lead and 16-lead ECGs (p=0.013) and between the 12-lead and 24-lead ECGs (p<0.001), but not between the 16-lead and 24-lead ECGs (p=0.054). For specificity, the same pattern was noted; the difference was statistically significant between the 12-lead and 16-lead ECGs (p=0.004) and between the 12-lead and 24-lead ECGs (p=0.031), but not between the 16-lead and 24-lead ECGs (p=0.581). The positive predictive value was 71% for the 12-lead ECG, 55% for the 16-lead ECG, and 63% for the 24-lead ECG.

As indicated in Figure 3A, 12 additional patients with MI were identified using the 16-lead ECG compared with the 12-lead ECG, as were 21 additional patients using the 24-lead ECG compared with the 12-lead ECG. Of these, 4 patients fulfilled the criteria for MI on both the 24-lead and 16-lead ECGs. Thus, 17 patients were not identified as having MI when considering only the 16-lead ECG, and 8 patients were missed when considering only the 24-lead ECG. Among the 8 patients with MI on the 16-lead ECG but not on the 24-lead ECG, 6 showed maximum ST-segment elevation in lead V4R, and 2 showed maximum ST-segment elevation in lead V8.

Among the non-MI patients, 1 of the patients with negative 12-lead ECG results fulfilled the criteria for MI on both the 16-lead and 24-lead ECGs. In all, 21 non-MI patients had ST-segment deviation on at least 1 of the ECGs. Figure 3B shows the distribution of these patients. The discharge diagnoses for these patients, according to the records, were angina pectoris (n=8), unspecified chest pain (n=8), and panic syndrome, acute subendocardial MI, heart failure, post-MI syndrome, and observation for suspected MI (n=1 for each).

The lead showing the maximum ST-segment elevation for patients with MI on the 24-lead ECG but not on the 12-lead ECG was most often −V4,

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followed by lead −V2 (Figure 4). For the non-MI patients who showed MI on the 24-lead ECG but not on the 12-lead ECG, leads −V4 and −V5 showed the maximum ST-segment elevation. For patients either with or without MI who showed MI on the 16-lead ECG but not on the 12-lead ECG, lead V4R most often showed the maximum ST-segment elevation (Figure 5).

MI patients who did not receive reperfusion therapy

In total, 43 patients with elevated biochemical markers did not receive acute MI treatment (thrombolysis or acute percutaneous coronary intervention). Of these, 4 patients had ST-segment elevation on 12-lead ECG, but 2 of them were considered to have pericarditis (young men with typical clinical course) and were excluded from the following analysis.

The sensitivity for detecting acute MI in these patients was 4.9% for the 12-lead ECG (2 of 41 patients), 17.1% for the 16-lead ECG (7 of 41 patients), and 26.8% for the 24-lead ECG (11 of 41 patients). Considering only the 14 patients with a CK-MB >50 μg/L (n=14), the sensitivity for the 12-lead ECG was 7.1%; for the 16-lead ECG, 14.3%; and for the 24-lead ECG, 35.7%. Patients with a CK-MB >100 μg/L also were analyzed (n=8); the sensitivity was 0% for the 12-lead ECG, 12.5% for the 16-lead ECG, and 37.5% for the 24-lead ECG.

Of the 3 patients with CK-MB >100 μg/L and ST-segment elevation on the 24-lead ECG, 1 had maximum ST-segment elevation in lead −V2, 1 in lead −V4, and 1 in leads −V2 and −V3 equally. The patient with ST-segment elevation on the 16-lead ECG had maximum ST-segment elevation in lead V4R. Figure 6 shows the chest leads from 1 patient with acute MI according to biochemical markers who did not receive percutaneous coronary intervention or thrombolysis. This patient did not have ST-segment elevation on the 12-lead ECG, but only on the 16-lead and 24-lead ECGs. No ST-segment elevation was observed in the limb leads.

Patients with admission ECG

This subpopulation consisted of 377 patients. In all, 166 of these patients had acute MI according to biochemical markers. Of the 166 patients, 113 (68%) were male and 53 (32%) were female. Of the 211 patients without MI, 121 (57%) were male and 90 (43%) were female.

For the 12-lead ECG, the sensitivity for detecting acute MI was 32.5%. For the 19-lead ECG, the sensitivity was 44.6%; and for the 24-lead ECG, 48.8%. The specificity for detecting acute MI was 97.2% for the 12-lead ECG, 96.2% for the 19-lead ECG, and 93.5% for the 24-lead ECG (figure 2).

The difference in sensitivity was statistically significant between all sets tested (12-lead vs. 19-lead ECGs: p<0.001; 12-lead vs. 24-lead ECG: p<0.001; 19-lead vs. 24-lead ECG: p=0.016). For specificity, there was no statistical difference between the 12-lead and the 19-lead ECG (p=0.50) and between the 19-lead and the 24-lead ECG (p=0.063). For the 12-lead vs. the 24-lead ECG, the difference was statistically significant (p=0.016). The positive predictive value at a hypothesized prevalence of 0.2 was 75% for the 12-lead ECG, 75% for the 19-lead ECG, and 66% for the 24-lead ECG.

DISCUSSION

In this study, the sensitivity to detect acute MI was significantly higher for the 16-lead and 24-lead ECG tracings compared with the 12-lead ECG. Specificity, however, was significantly

higher for the 12-lead ECG than for the 16-lead or 24-lead ECG. The sensitivity for diagnosing acute MI for was very low for all 3 methods, probably because most ECGs were recorded after reperfusion therapy. In some cases, the ST-segment elevation was already normalized. When only admission ECGs were analyzed, sensitivity was higher for the 19-lead and 24-lead ECG compared to the 12-lead ECG, while specificity was slightly lower for the 24-lead compared with the 12-lead ECG, but not compared with the 19-lead ECG. Positive predictive values were equal for 12-lead and 19-lead ECGs in the admission group, otherwise higher for the 12-lead ECGs. These results indicate that especially the 19-lead ECG might be an attractive alternative to the 12-lead ECG, since it is equally likely that a patient with a "positive" ECG has acute coronary syndrome, but the sensitivity for the method is higher.

In patients with elevated biochemical markers who did not receive acute reperfusion therapy, sensitivity was much higher for the 24-lead ECG than for the 12-lead ECG (27% versus 5%, respectively). Even when considering patients with large infarctions (CK-MB >50 or >100 μg/L), who would probably benefit from acute thrombolysis or percutaneous coronary intervention, more MI patients were detected with the 24-lead ECG compared with the 12-lead ECG. Thus some patients whose MIs are detectable only by 24-lead ECG are perhaps being overlooked for potentially beneficial reperfusion therapy.

In a recent study by Martin et al. (Wagner *et al*., 2006; Martin T, personal communication) the abilities of the 12-lead ECG and the 24-lead ECG to detect acute MI (determined by magnetic resonance imaging) were investigated. They found that the sensitivity increased from 50% with the 12-lead ECG to 84% with the 24-lead

ECG, whereas the specificity decreased only slightly, from 97% to 93%. No additional leads were used in that study for comparison, but 24-lead ECG appears to be a promising alternative for enhancing the sensitivity for detecting acute MI. The present study showed no difference in sensitivity or specificity between the 16-lead and 24-lead ECGs, although there were trends towards higher sensitivity and specificity for the 24-lead ECG compared with the 16-lead ECG. Given that the 24-lead ECG requires no additional electrodes, this method is also simpler than 16-lead ECG and should therefore be considered as an add-on to analysis of the 12-lead ECG. On the other hand, the present study might have underestimated the true sensitivity of the 16-lead ECG in detecting MI, because ST-segment elevation in right-sided chest leads is usually an early and transient phenomenon in right ventricular infarction (Braat *et al*., 1983; Klein *et al*., 1983). Leads V4R and V5R might be of greater value in emergency situations than indicated in the present study. In addition, 8 of the patients with acute MI in the current study were detected only by the 16-lead ECG and not by the 12-lead or 24-lead ECGs.

Of interest is that the leads with maximum ST-segment elevation in the 24-lead ECG in patients who did not have acute MI were −V4 and −V5 (−V4 and −V6 for admission ECGs). In a recent study, Perron et al. (Wagner *et al*., 2006; Perron A, personal communication) sequentially added inverted leads to the 12-lead ECG in patients who underwent prolonged percutaneous coronary intervention in order to identify a cutpoint at which the sensitivity for acute ischemia/infarction was significantly increased without a significant decrease in specificity. They concluded that a 19-lead ECG—with leads −V1, −V2, −V3, −aVL, −I, +aVR,

and −III in addition to the standard 12 leads—had the highest sensitivity (78%) without a large decrease in specificity (to 93%). In the present study, we found higher positive predictive value for 19-lead ECG compared with 24-lead ECG. ST-segment depression in lead V5 is known to have the highest sensitivity for detecting ischemia during exercise tests (Viik *et al.*, 1997). Some of the patients in the present study who had ST-segment elevation in leads −V4, −V5, and −V6, but not elevated biochemical markers, thus might have had ischemia but not acute MI. Some of these patients also might have had left ventricular hypertrophy not detected by Cornell voltage. Of note, maximum ST-segment elevation occurred most often in lead V4R in patients with MI, but this lead also showed maximum ST-segment elevation most often in patients without MI.

In clinical routine, it is common to distinguish ST elevation MI from non-ST elevation MI. All ST deviations during chest pain are probably not equivalent (as indicated for example by the false positive rates in inverted V4-V5). It is also questionable whether all ST deviation during acute coronary syndrome is the same. The VANQWISH trial (Boden *et al.*, 1998), for example, showed raised mortality with an invasive approach early after non-Q-wave MI. However, many observational studies and the randomized TIMI IIIb trial (de Feyter *et al.*, 1985; Anderson *et al.*, 1995; McCullough *et al.*, 1998) have shown shorter hospital stays, fewer readmissions, less ischemia, and fewer symptoms with an early invasive approach. In the prospective FRISC II study (FRISC II, Lancet 1999), patients were randomly assigned to an early invasive or non-invasive treatment strategy. They found a decrease in the composite endpoint of death or MI in the invasive group compared with the

non-invasive group. They concluded that early invasive approach should be the preferred strategy in most patients with unstable coronary artery disease who have signs of ischemia on ECG or raised biochemical markers of myocardial damage.

In the present study, ST-segment elevation was the only ECG variable used to detect MI. Instead of using inverted leads in the 24-lead ECG, however, it is equally possible to use ST-segment deviation (that is, both ST-segment elevation and depression) in the 12 standard leads with the addition of lead −aVR instead of lead +aVR. Experienced cardiologists might in practice recognize posterior MI due to characteristic ST depression in V1-V3. In the present study, however, we deliberately did not compare the ST elevations in the 12-lead ECG in addition to ST depression in V1-V3 with the expanded lead sets. Experienced cardiologists might for a long time have regarded ST depression in lead V1-V3 as posterior MI, but still the presence of ST depression has not received the same status in international guidelines. We therefore wanted to investigate the performances of the 19-lead or the 24-lead ECG, since this might be a pedagogic approach to better understand the relation between the ECG and the cardiac physiology.

Limitations of the study

For logistical reasons, 16-lead ECGs were not recorded on admission. It would have been preferable to record these study ECGs before treatment, to increase the likelihood of observing higher sensitivities for detecting acute MI.

CONCLUSION

Sensitivity for detecting acute MI is higher for 16-lead and 24-lead ECGs compared with the conventional 12-lead

ECG. Specificity, however, decreases slightly when using the 16-lead or 24-lead ECG. To increase the sensitivity for detecting MI, clinicians might wish to consider the use of the 24-lead ECG or a subset thereof, such as the 19-lead ECG, since no additional electrodes are required beyond those of the conventional 12-lead ECG.

ACKNOWLEDGMENTS

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FIGURE LEGENDS

Figure 1. Contiguous leads in the 24-lead ECG. The figure shows frontal and transverse views of acute posterior myocardial infarction. Note the ST-segment depression in leads V1–V5 and the ST-segment elevation in the corresponding inverted leads. Reprinted from (Pahlm-Web *et al*., 2002), with permission.

Figure 2. Receiver-operator characteristic diagram of sensitivity and specificity of the postadmission and the admission ECGs.

Figure 3. Venn diagrams of (A) patients with acute MI (biochemical markers) and signs of acute MI on at least one ECG, and (B) patients without acute MI but with ST-segment deviation on ECG.

Figure 4. Proportions of patients with maximum ST-segment elevation in inverted leads, among patients who were diagnosed as having MI on 24-lead ECG but not on standard 12-lead ECG.

Figure 5. Proportions of patients with maximum ST-segment elevation in leads V4R, V5R, V8, or V9, among patients who were diagnosed as having MI on 16-lead ECG but not on standard 12-lead ECG.

Figure 6. Chest leads (standard, inverted, and leads V4R, V5R, V8, and V9) from a patient with acute MI who did not show ST-segment elevation on the 12-lead ECG, but only on the 16-lead and 24-lead ECGs.
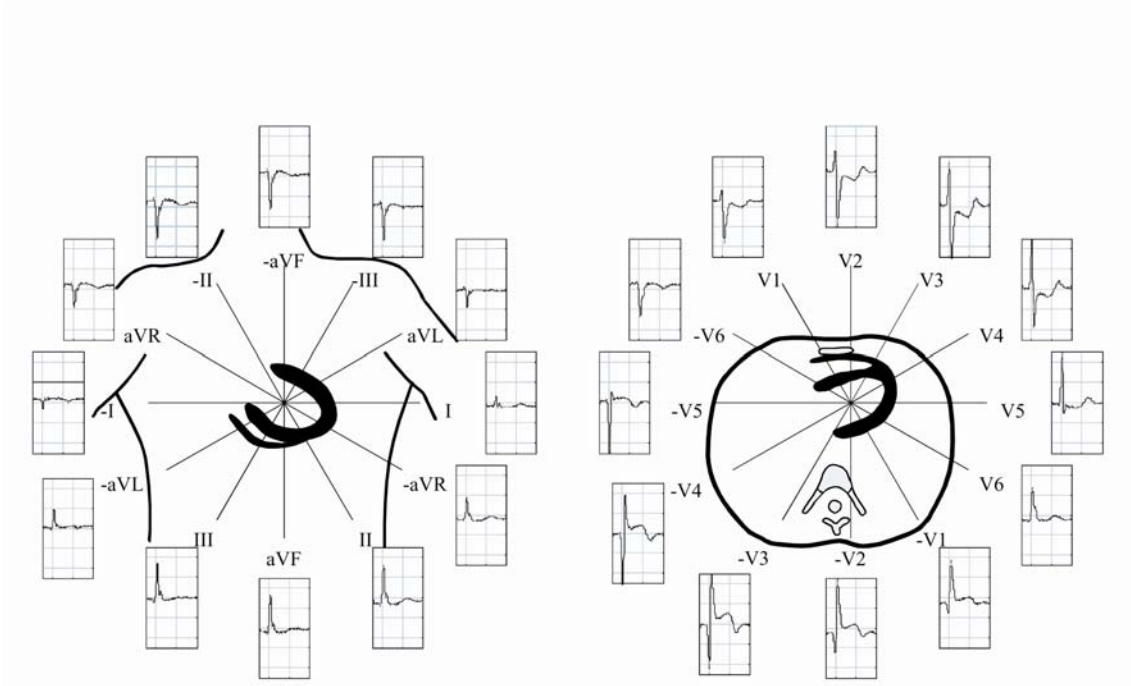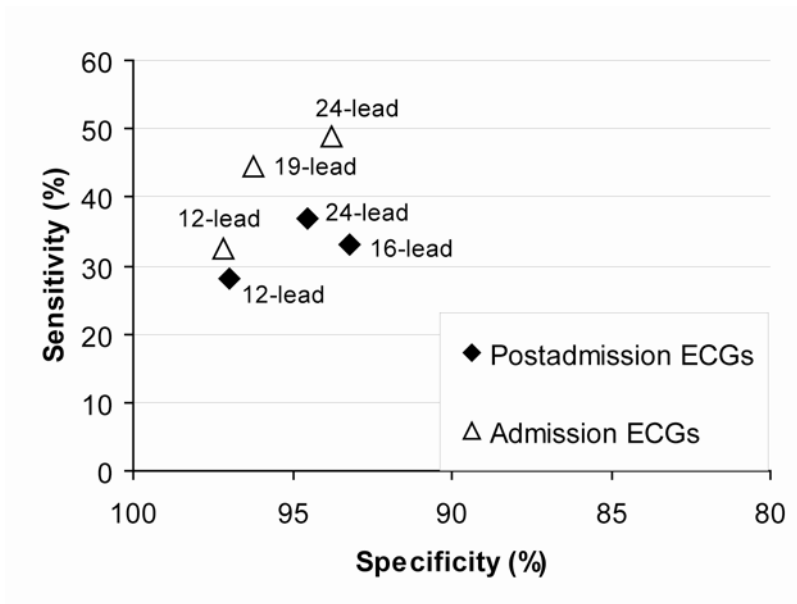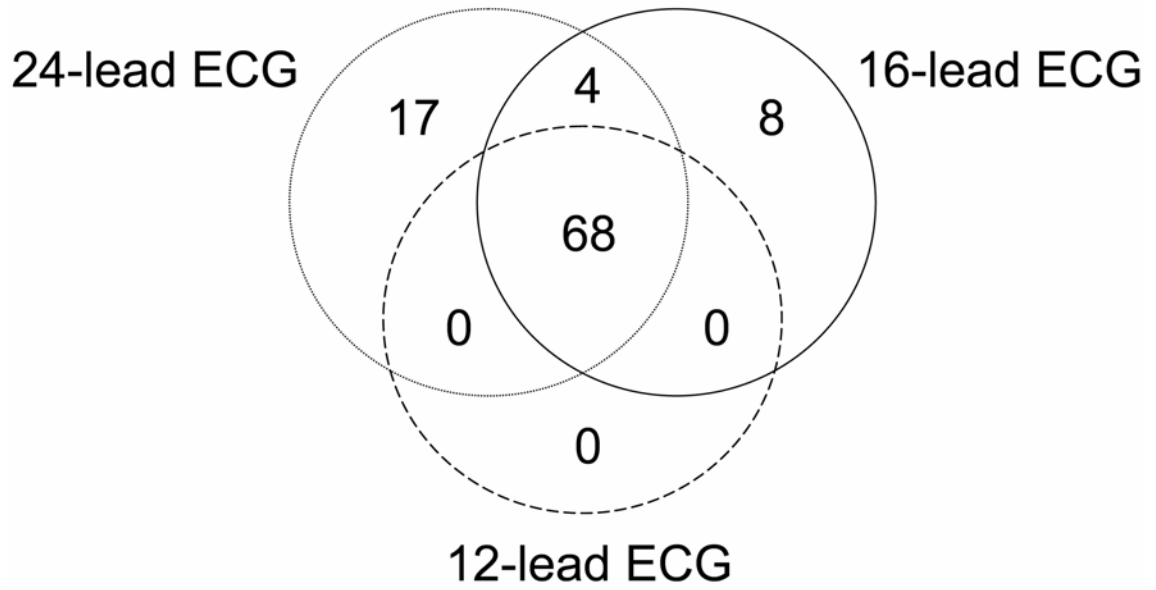
Figure 1

Figure 2

A

24-lead ECG

16-lead ECG

17

4

8

68

0

0

0
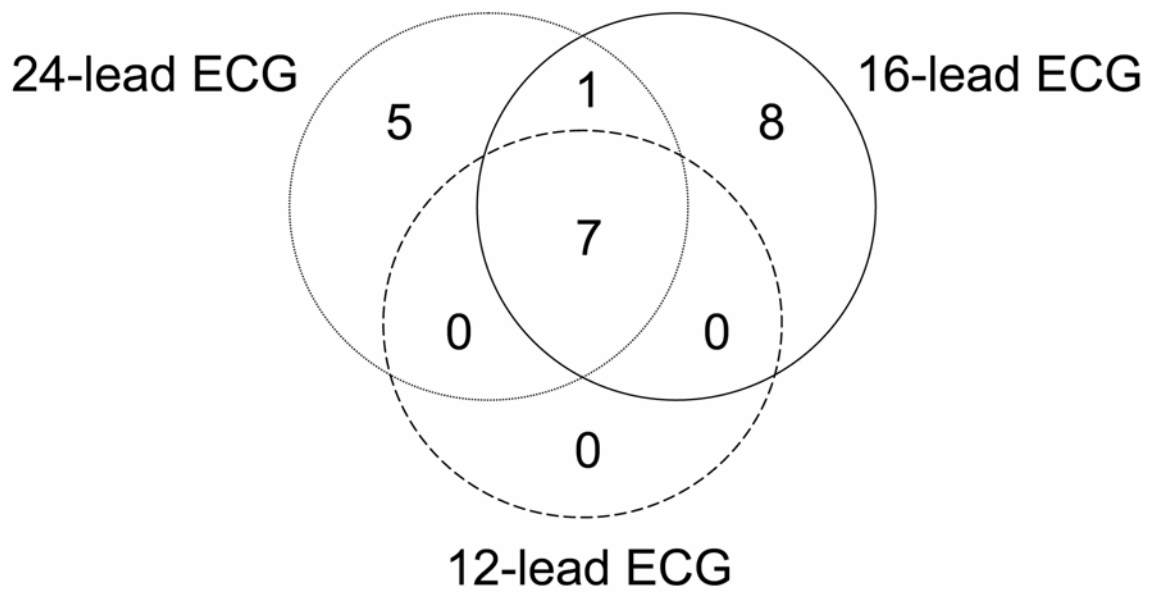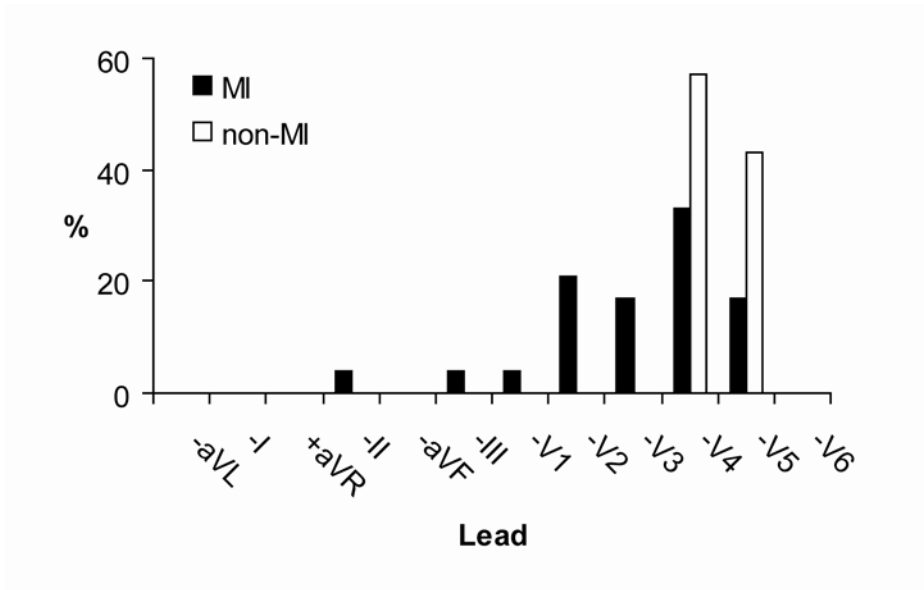
12-lead ECG

B

24-lead ECG

16-lead ECG

5

1

8

7

0

0

0

12-lead ECG

Figure 3

Figure 4

Figure 5

Figure 6