realized. Were the current study repeated today, in laboratories that have sought to use the anatomical and pathological rationale that I have suggested above should drive the carotid intima–media thickness protocol, I believe the use of the composite measure would be strongly confirmed.

Finally, when considering what segments should be studied in evaluating the effectiveness of a drug in retarding the progression of carotid intima–media thickness (and presumably atherosclerosis), a similar argument can be made. A number of very successful multicentre clinical trials have used a carotid intima–media thickness outcome variable including data from all three anatomical segments that were acquired from a complete circumferential scan of each carotid artery[4–6]. In principle, a drug may well be expected to have different effects on retarding progression of carotid intima–media thickness in different anatomical segments. Thus limiting studies to the common carotid artery only (far wall only even) or to only one of the other two segments accessible to ultrasonic imaging may do a relatively poor job of determining if a drug is effective. Drugs that are truly effective overall may be found to be ineffective due to limiting the study to only one of the three accessible sites. Furthermore, effectiveness of a drug in the common carotid artery alone may be due to dilatory effects of the drug on the arterial diameter and be incorrectly interpreted as effective in retarding progression of intima–media thickness.

In summary, from a scientific perspective, it makes good sense to measure carotid intima–media thickness in all three segments accessible to high resolution B-mode ultrasound. It requires developing greater skill within and more effort from the individuals charged with obtaining the B-mode images of artery walls needed to make these measurements. This will require more time to train and certify these individuals. If one does not take this approach, one will do a relatively poor job of characterizing the level of risk within an individual as well as be led to incorrect evaluations of therapeutic agents. One could reasonably say that too many of our diagnostic tests with medical ultrasound are not done in a particularly reliable manner. How often do we even have a valid quality control/quality assurance programme in place to really know the reliability of our tests? We owe it to the potential of carotid intima–media thickness that it not go the way of other diagnostic tests due to a lack of resolve to do it the right way and to commit the necessary level of resources to the task.

W. A. RILEY
Wake Forest University School of Medicine, Winston-Salem, NC, U.S.A.

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Continuous multilead ST-segment monitoring should be a part of the clinical routine

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Acute coronary syndrome is a dynamic condition, often with repeated episodes of myocardial ischaemia due to waxing and waning thrombus, intermittent vasoconstriction and downstream platelet embolization. However, in clinical practice, there are few objective methods that are able to reflect the dynamic nature of this condition. A standard 12-lead ECG, especially if obtained both during and after chest pain, is one method to assess these patients. However,
since 70–90% of all ischaemia detectable with ECG is silent, even repeated registrations during chest pain will often be insufficient to detect all episodes of ST-segment changes.

The value of ST-segment recording with Holter in patients with unstable angina or non ST-elevation myocardial infarction has been examined in several studies during the last two decades. The presence of transient ST-segment deviations has been related to a higher rate of left main or multivessel disease[1,2] and subsequent death, myocardial infarction, severe angina and need for revascularization[1–4]. The number of patients identified as having transient ST-segment changes has varied widely (15%–66%), mainly because of the use of different inclusion criteria. During short- as well as long-term follow-up, occurrence of transient ST-segment changes has been an independent predictor of outcome in multivariate models including both clinical background variables, recurrence of chest pain and ECG on admission[2–4].

However, the major disadvantage with Holter-recording is the off-line analysis, which means that the information obtained may reflect events occurring several hours or days earlier. This has probably contributed to the limited use of Holter monitoring in clinical practice. Another problem with Holter recordings is the use of 2–3 leads, which may result in missed episodes of ischaemia[5,6].

On-line multilead ST-segment monitoring with continuous 12-lead ECG or vectorcardiography was introduced during the late 1980s[7,8]. It has been found useful for detection of ischaemia during and after percutaneous coronary intervention[8,9], and for identification of successful reperfusion and a better outcome after thrombolytic treatment in patients with ST-elevation myocardial infarction[10–12]. In patients with unstable angina or non ST-elevation myocardial infarction, both continuous 12-lead ECG and vectorcardiography have been shown to improve detection of ischaemia and to be of incremental prognostic value in relation to both ECG on admission and troponin T[13–16]. In addition, the methods have been shown to be useful in identifying responders to[17–19], and predicting the effect of antithrombotic treatment[20].

In the article published in this issue, Drew and co-workers provide additional data on the usefulness of the continuous 12-lead ECG[21]. The study included a broad population, from patients with ST-elevation myocardial infarction to patients with stable coronary artery disease admitted for cardiac catheterization. Among these patients, 20% had episodes of transient ST-segment deviation, of which only 23% were accompanied by chest pain. When patients were divided according to the initial standard 12-lead ECG into those with initial ST-segment elevations and those with initial ST-segment depressions or T-wave inversions, the incidence of transient ST-segment deviations were about 30% in both groups, which is in accordance with previous studies including similar patients[15,19,20,22,23]. Interestingly, 15% of the patients without signs of ischaemia on the initial ECG subsequently had transient ST-segment changes during monitoring. This points out a serious limitation with a single 12-lead ECG and stresses the importance of ST-segment monitoring. Moreover, when added to a logistic regression model including signs of ischaemia on the initial ECG, occurrence of transient ST-deviations came out as an independent predictor of outcome, which is well in line with previous observations[2–4,13,15]. Unfortunately, the authors have not stated clearly how they separated transient ST-deviations predicting the event from those occurring during the event. Furthermore, it would have been interesting to know the prognostic value of ST-deviations in relation to diagnosis, since one can assume that most events were in the group with acute coronary syndrome.

Most previous studies have considered episodes of ST-segment depression and ST-segment elevation as one group. In the present study, Drew and co-workers found that transient ST-segment elevations to be of shorter duration, had higher ST-magnitude changes and were more often associated with chest pain. Moreover, patients with episodes of ST-elevation more often had single vessel disease, whereas those with episodes of ST-segment depressions more often had three-vessel disease. Thus, the direction of the transient ST-segment deviation may have therapeutic implications.

Despite all clinical evidence and the fact that on-line multilead ST-monitoring was introduced in the late 1980s, the use of this method in clinical practice, outside Scandinavia, is still very limited. However, there are several reasons to believe that the use will increase. Multilead monitoring has the ability to detect ischaemia that does not necessarily result in myocardial necrosis, and to reflect the dynamic nature of myocardial ischaemia and coronary thrombosis. Hence, multilead monitoring has the capacity to reflect the response to pharmacological treatment[17–19]. No other method can provide this information immediately on-line. These features will be valuable in the future when the management of acute coronary syndrome becomes more tailored and targeting treatment for the individual patient more important.

However, there is a need for further studies in this field. Most reports emanate from a few centres. Both continuous 12-lead ECG and vectorcardiography
need to be validated in terms of inter-observer agreement and with clinicians from several different centres. Another problem is the lack of standardization. The criteria used during analysis is often poorly described and different definitions of a transient ST-segment deviation have been used. In the present study by Drew et al., a transient ischaemic episode was defined as an ST-segment deviation of $\geq 200$ $\mu$V in at least one lead or $\geq 100$ $\mu$V in at least two leads, whereas other studies have defined an ischaemic episode as an ST-deviation of $\geq 100$ $\mu$V in at least one lead\[6,13,17,20,22\]. Furthermore, Drew and co-workers considered a shift of ST-level in a positive direction in leads with ‘fixed’ ST-segment depressions as an ischaemic episode. Not all would agree on this definition and this may to some extent explain the rather high rate of patients with transient ST-segment elevation in the group with ST-segment depressions or T-wave inversions in the initial ECG. However, few reports describe ST-segment changes in detail. Several reports also lack information on how ST-segment changes due to changes in body position have been judged. Although most changes are obvious, they certainly can imitate ischaemia and cause problems in the analysis.

Although more studies are needed, we believe we have enough data to recommend using continuous multilead ST-segment monitoring as a routine when assessing patients with acute coronary syndrome. So far, there have been no ‘head to head’ comparisons between continuous 12-lead ECG and continuous vectorcardiography. However, the methods have many similarities and seem to identify the same patients when used in the same population\[29\]. Today, the major divisor is between repeated single ECG recordings and on-line continuous multilead monitoring, not between the different systems. We now need studies randomizing patients to continuous multilead monitoring in order to provide the final evidence that the use of this technology also improves some measure of outcome.

T. JERNBERG
B. LINDAHL
L. WALLENTIN
Department of Cardiology,
Cardiothoracic Center,
University Hospital,
Uppsala, Sweden

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‘The heart doesn’t pump, the kidneys suck’

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The amusing quote that serves as a headline for this editorial was told to me some years ago by a nephrology colleague of mine. Of course, he was trying to be humorous but perhaps there was a bit of truth in what he said. The heart and the kidneys are closely allied in maintaining the body’s circulatory homeostasis, a fact well known to physiologists for many decades. Of course, the circulation of the blood depends upon the heart’s ability to pump, but blood pressure regulation is a major component of normal renal function. Thus, the partnership between the heart and the kidneys is a critical one for normal circulatory function.

In this issue Sørenson et al. report their observations on more than 6000 patients with previous myocardial infarction[1]. The authors were interested in determining whether abnormal renal function affected mortality following a myocardial infarct. Their analysis clearly demonstrated that abnormal renal function increased the likelihood of dying after a myocardial infarction. However, mild renal impairment had no influence on prognosis after statistical adjustment was made for other variables that affect post-myocardial infarction prognosis.

Only patients with creatinine clearances less than or equal to 40 ml·min$^{-1}$ had an increased risk of dying following an myocardial infarction.

Sørenson and co-workers are not the first investigators to observe that abnormal renal function affects long-term prognosis in patients with arteriosclerotic coronary artery disease. Aronow prospectively studied approximately 1400 elderly patients with a variety of chronic illnesses. Most of these elderly patients also had atherosclerotic heart disease. He noted that patients with hypertension and/or diabetes mellitus combined with an increased serum creatinine had a markedly increased likelihood of developing a new coronary event during 3-5 years of follow-up[2]. Similarly, Johannes et al. observed in the HOPE trial (Heart Outcomes and Prevention Evaluation) that patients with pre-existing vascular disease or diabetes mellitus combined with one other coronary risk factor had a significantly increased risk for a subsequent coronary event if they had mild, moderate, or severe renal insufficiency[3]. ACE inhibitor therapy with ramipril reduced the coronary event rate irrespective of whether the patients had renal insufficiency or not. And finally, it has been known for decades that abnormal renal function markedly worsens prognosis in patients hospitalized with acute myocardial infarction[4].

What could be the mechanism by which renal insufficiency worsens prognosis in patients with

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