Onon-invasive investigation of regional dysfunction in acute myocardial infarction.

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**Document Version**
Publisher's PDF, also known as Version of record

**Publication date:**
1979

**Link to publication in University of Groningen/UMCG research database**

*Citation for published version (APA):*
SUMMARY

This thesis describes a non-invasive method by which important data can be obtained about the pump function of the heart in patients with acute myocardial infarction. Disturbance in the pump function of the heart is an increasingly important cause of death and disability in patients with coronary artery disease. Arrhythmias are another important cause of death in these patients, but lately it has been possible to treat these more and more effectively, because more effective drugs and better methods of investigation are available. In chapter 1 this matter is discussed and several methods are described by which the pump function of the heart can be investigated. In patients with acute myocardial infarction it is important to obtain data on global dysfunction: the functional disturbance in the left ventricle as a whole, and regional dysfunction: the functional disturbance in a part of the left ventricular wall. Left ventricular function can be studied during the different periods of the cardiac cycle, i.e. the phases of contraction, relaxation and filling. Invasive investigation (cardiac catheterisation) generally gives extensive and reliable results. In the last one or two decades, the development of several non-invasive methods applicable to infarct patients has received increasing attention, because these methods are not harmful to the patient and are easily repeatable. Our investigation was made by registering an echocardiogram (of the left ventricular posterior wall, the mitral valve and the left atrium), an apexcardiogram and a carotid pulse tracing, together with an electrocardiogram and a phonocardiogram. Patients with recent myocardial infarction were examined. The examination has been carried out three times: in the first week, in the second week and two months after the infarction.

In chapter 2 a description is given of the way in which the pump function of the left ventricle gets disturbed as a result of regional dysfunction. Regional dysfunction occurs because of localized stenoses in the coronary arteries, which reduce the circulation of a part of the myocardium. It consists of an abnormal way of contraction and relaxation in which a part of the ventricular wall lags behind the rest. Normally during relaxation an elastic recoil takes place, causing the rapid filling with blood from the left atrium. Two thirds of the ventricular filling normally takes place this way. The remaining third
part of the filling is mainly caused by atrial contraction. Incoordinate contraction, occurring in regional dysfunction, causes a change in the shape of the left ventricle, which disappears during relaxation (see fig. 1). This change in the shape of the left ventricle takes place during isovolumic relaxation and causes a movement of the left ventricular posterior wall, which does not occur in normal cases. The movement of the left ventricular posterior wall echo during isovolumic relaxation is expressed as a fraction of its total movement. This fraction is called IR-ratio and is introduced here as a non-invasive index of incoordinate relaxation. This in turn is related to the size of the infarct. The relaxation disorder also causes a disturbance in early diastolic filling. Left atrial contraction therefore has to contribute relatively more to ventricular filling.

Chapter 3 discusses the way in which information about cardiac function may be obtained from the apexcardiogram. For this purpose, a study was made in which simultaneous recordings were made of left ventricular pressure and apexcardiogram during cardiac catheterisation. The study was made in 19 patients with different diagnoses. It appears that the apexcardiogram is similar to the left ventricular pressure curve (or wall stress curve), especially during the isovolumic periods. From the isovolumic parts of the apexcardiogram, indices can be obtained of contraction velocity and relaxation velocity, which agree to similar indices obtained from the left ventricular pressure curve. There is some agreement between the index of contraction velocity and \( \frac{dp}{dt} \) (max). Further, the ratio of the a-wave amplitude and total amplitude of the apexcardiogram \( (a/H\text{-ratio}) \) is related to the left ventricular pressure rise during atrial contraction.

Chapter 4 gives the data of the 17 infarct patients and the 28 normal individuals, as well as the methods of the investigation. From the carotid pulse tracing, the electrocardiogram and the phonocardiogram, systolic time intervals were measured, including left ventricular ejection time and the total electromechanical systole. Also the ratio of pre-ejection time and ejection time was calculated. From the recording of the apexcardiogram, the indices of contraction velocity and relaxation velocity, mentioned in chapter 3, and the \( a/H\text{-ratio} \) were measured. From the mitral valve echocardiogram, the onset of flow, at the beginning of rapid filling and again at the beginning of filling during systole. In chapter 4, the data of the 17 infarct patients and the 28 normal individuals, as well as the methods of the investigation, are presented. From the carotid pulse tracing, the electrocardiogram and the phonocardiogram, systolic time intervals were measured, including left ventricular ejection time and the total electromechanical systole. Also the ratio of pre-ejection time and ejection time was calculated. From the recording of the apexcardiogram, the indices of contraction velocity and relaxation velocity, mentioned in chapter 3, and the \( a/H\text{-ratio} \) were measured. From the mitral valve echocardiogram, the onset of flow, at the beginning of rapid filling and again at the beginning of filling during systole.
The beginning of atrial systole, was determined. From the echocardiogram of the left atrium, the filling pattern of the left ventricle in terms of atrial diameter changes was obtained. From the left atrial diameter change during atrial systole and the $a/H$-ratio of the apexcardiogram, an index of left ventricular end-diastolic compliance, the compliance-ratio, was calculated. From the echocardiogram of the left ventricular posterior wall, the above mentioned IR-ratio was measured. The IR-ratio was compared with the maximum concentration of the enzyme lactodehydrogenase, 1st and 2nd fraction ($LDH_{1+2 \text{ max}}$) in the plasma, which is a measure of infarct size. Finally, a reproducibility study was made to test the reliability of the measurements by means of repeated independent observation.

Chapter 5 presents the results of the investigation. From the investigation of the normal individuals it appears that measurements related to relaxation are age-dependent: indices related to relaxation velocity and volume changes during rapid filling are lower with increasing age. In infarct patients the IR-ratio in the first week after infarction correlates with infarct size as expressed by $LDH_{1+2 \text{ max}}$. Measurements related to relaxation, isovolumic phase and rapid filling appeared to correlate quite well with the IR-ratio. Measurements related to atrial contraction correlated better with $LDH_{1+2 \text{ max}}$ than with the IR-ratio. Indices related to ventricular systole showed hardly any relation to the IR-ratio or $LDH_{1+2 \text{ max}}$.

In chapter 6 the results of the investigation are discussed. It appears that regional dysfunction in myocardial infarction can indeed be described as proposed in chapter 2. Myocardial infarction causes incoordinate contraction and incoordinate relaxation. Incoordinate contraction causes a decrease in the stroke volume and increase in the end-systolic volume. Incoordinate relaxation causes a disturbance in rapid filling. In this way, left atrial volume preceding left atrial contraction is larger than normal. This results in an increased force of left atrial contraction, which in turn may cause an increase in the end-diastolic volume. Via the Starling mechanism of the left ventricle, this may increase the stroke volume, thus compensating for the consequences of incoordinate contraction. As diastolic compliance is also less than normal, especially in the early stage, this does not always occur.
From the reproducibility study it can be concluded that the measurement of the IR-ratio is sufficiently reliable and sensitive. It may therefore be used as an index of infarct size in the early stage of acute myocardial infarction. Further investigation should be made to establish the value of the IR-ratio in monitoring patients with acute myocardial infarction and in the control of their therapy with e.g. betablocking agents, calcium antagonists, vasodilating drugs and catecholamines.

In summary, the following conclusions can be made:
1. The IR-ratio appears to be a new, useful and quite easily obtainable and reliable non-invasive index of regional dysfunction in the early stage of myocardial infarction.
2. From an apexcardiogram indices of velocity of contraction and relaxation may be derived, which reflect global ventricular function. However, the measurement of these indices may be too complicated for clinical use.
3. Congestive heart failure caused by myocardial infarction is not only due to decreased left ventricular diastolic compliance but also due to incoordinate relaxation, which in turn is closely related to incoordinate contraction. The relaxation disorder causes a disturbance in initial rapid filling, which may be compensated by increased atrial activity.
4. Relaxation should be considered as an important function of the left ventricular myocardium, lasting from the end of systole till the end of rapid filling. In normal persons the rate and extent of relaxation are related to age. Relaxation should be considered seperately from and next to diastolic compliance.
5. The most valid and sensitive indices of regional dysfunction can be derived from the relaxation period.