Relationship between Heart Rate and Ventricular Ectopic Rhythm in Acute Myocardial Infarction

W. Merx, K. W. Heinrich, W. Bleifeld and S. Effert

Department of Internal Medicine I (Director: Prof. Dr. S. Effert) Rheinisch-Westfälische Technische Hochschule Aachen

Summary. The study of 1313 time intervals from 146 patients with acute myocardial infarction showed that a linear relationship exists between the occurrence of ventricular ectopic beats or runs of ventricular ectopic beats and the heart rate. With faster heart rates, the percentage of these arrhythmias is decreased. Ventricular tachycardias and ventricular fibrillation or flutter were also seen most commonly in bradycardia, but there was a second maximum in frequencies exceeding 110/min. It is supposed that in cases with such tachycardial heart action severe heart failure and myocardial hypoxia are often present and that under these circumstances, only relatively few ventricular ectopic beats are necessary to trigger ventricular tachycardias or ventricular flutter/fibrillation.

These results underscore in clinical practice that acceleration of heart frequency is a reliable measure to suppress ventricular ectopic rhythm. Electrostimulation of the right atrium is recommended for this purpose, although a stable position for the electrode catheter within the atrium may be difficult to achieve.

Key words: Ventricular ectopic rhythm, arrhythmia, heart rate, bradycardia.


Schlüsselwörter: Ventriculäre ektope Rhythmusstörungen, Arrhythmien, Herzfrequenz, Bradykardie.

In general antiarrhythmic drugs are used to prevent ventricular tachyarrhythmias. These drugs have, however, proved a disappointment in many cases, because the negative side effects on contraction and conduction frequently necessitate a stop in the treatment. In some cases, when the slowing down of the excitation wave is relatively greater than the prolongation of the refractory time, it is indeed possible to provoke ventricular tachycardias and fibrillation by antiarrhythmic drugs.

In this situation, interest has recently been focused on heart rate. When the frequency is low it may provoke ventricular arrhythmias and, on the other hand, when the heart rate becomes faster, it can be relied upon to suppress ventricular ectopic rhythm.

For many years it has been known that ventricular tachyarrhythmias accompanying bradycardia in total atrioventricular block can be successfully prevented by electrostimulation of the heart [1–3]. Since Sowton, Leatham and Carson in 1964 [4] using high frequency electrostimulation, and in the same year Domany and Effert [5] using sympathomimetic amines, proved that, in the case of intact atrioventricular conduction, recurrent ventricular fibrillation can also be prevented by acceleration of heart frequency, the number of clinical reports on suppression of dangerous ventricular tachyarrhythmias by these means has grown steadily [6–15].

In view of these promising results we thought it useful to evaluate more closely the relationship between heart frequency and ventricular ectopic rhythm in patients with myocardial infarction.

Material and Method

This investigation was made possible by a monitor system which automatically recorded all arrhythmias. The details of this system have previously been reported [16–18]. Fig. 1 shows the set-up in our cardiac care unit with a bedside monitor and a central unit, to which an analogue magnetic tape recorder is connected. In addition to storage, this installation allows the automatic recording of arrhythmias with the aid of a special evaluation electronic system: If an abnormally early impulse occurs within the monitored time between two normal R-waves, or if no impulse occurs within a second monitored time, reaching from one normal R-wave to just behind the next following normal R-wave, an ECG recorder is started by the evaluation unit. The ECG is written out two seconds before the beginning of the arrhythmia since the relevant data is recorded between the recording head and the playback head of the magnetic tape recorder.
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Fig. 1. Principle of the monitoring system with equipment for long-term continuous monitoring. The bedside monitors are connected to the central unit, which stores the electrocardiograms from six patients. Arrhythmias are written out automatically and the ECG from analysis is performed with time-contraction.

Ventricular ectopic beats and runs of ventricular ectopic beats occurring within mean time intervals of three hours were counted and correlated with the corresponding mean heart rate in 146 patients with acute myocardial infarction. Altogether 1313 time intervals were studied. In this group only patients with sinus rhythm were included and the correlations were made only during the first three days of acute myocardial infarctions. Runs of ventricular ectopic beats are defined as sequences of two to ten ventricular ectopic beats, sequences of more than ten ventricular ectopic beats are defined as a ventricular tachycardia.

In order to investigate the dependence of ventricular tachycardias and ventricular fibrillation or flutter upon the preexisting heart frequency we studied 41 episodes of ventricular tachycardias in 32 patients and 31 episodes of ventricular fibrillation/flutter in 24 patients with acute myocardial infarction. The heart frequency with which these arrhythmias were calculated was calculated from the last 4 to 6 seconds before ventricular tachycardias or flutter/fibrillation arose. Patients who experienced atrial fibrillation or flutter instead of sinus rhythm were not excluded from this latter group.

Results

The second figure shows the relationship between ventricular ectopic beats and the frequency of sinus rhythm in 1313 examined time intervals from 146 patients. In the case of frequencies of up to 65/min, in a collection of 109 time intervals, 82% had at least one ventricular ectopic beat. Acceleration of heart rate was seen to be negatively correlated with the occurrence of ventricular ectopic beats and in the case of a sinus rate exceeding 125/min only 11% ventricular ectopic beats were observed. A linear correlation exists between heart rate and the frequency of ventricular ectopic beats and is highly significant ($r = 0.985$, $p < 0.001$). The regression line has a slope of $-0.90$ and therefore an acceleration of 10/min reduces the possibility that ventricular ectopic beats occur by 9%.

Fig. 3 shows the results obtained when the examined time intervals were not only grouped according to the presence or absence of ventricular ectopic beats, but were further divided according to the number of ventricular ectopic beats per hour. It can be seen that ventricular ectopic beats, in a number of more than 5/min, did not occur when the heart frequency exceeded 125/min.

The incidence of runs of ventricular ectopic beats was similar to ventricular ectopic beats becoming less frequent with higher rates of sinus rhythm, as is demonstrated in Fig. 4. It can be seen that 22% and