Fourier analysis of ventricular fibrillation of varied aetiology

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ABSTRACT

Fast Fourier Transform analysis was used to study ventricular fibrillation induced by several different methods in greyhounds anesthetized with sodium pentobarbitone. The dominant frequency at the body surface of ventricular fibrillation induced electrically in non-ischemic hearts was initially 9.9 ± 0.7 Hz, remained above 9 Hz for 70 sec and then rapidly fell to 5 Hz. The dominant frequency of ventricular fibrillation induced by acute occlusion (initially 12.3 ± 0.2 Hz), or by reperfusion (12.2 ± 0.4) of the anterior descending branch of the left coronary artery, showed a similar time-course. However ventricular fibrillation induced by administration of potassium (4.8 ± 0.8 Hz) or ouabain (7.1 ± 1.1 Hz) was significantly slower. There was little difference in the time-course of fibrillation in the non-ischemic heart recorded directly from the epicardium or from a surface lead. While the dominant frequency of fibrillation recorded from the endocardium of the heart was initially similar to that recorded at the body surface, there was no significant fall in frequency during the period of analysis. These findings may be of relevance to the poor response to DC countershock after prolonged ventricular fibrillation.

Keywords Ventricular fibrillation Myocardial ischemia Reperfusion Cardiac glycosides Potassium

Introduction

Ventricular fibrillation has been defined as "chaotic asynchronous fractionated activity of the heart" (1). It is generally considered to be due to irregular totally disorganized electrical activity of the ventricles. However direct electrophysiological measurements during ventricular fibrillation are extremely difficult to obtain due to the complexity of the arrhythmia. Epicardial recordings at the onset of reperfusion ventricular fibrillation initially showed that wavefronts were conducted at increasing speed from the area of reperfusion to the opposite side of the heart (2). However further analysis was impossible after only a few seconds, due to the variable pathways in the myocardium. Can we learn more about ventricular fibrillation from a study of the electrocardiogram at the body surface?

Fibrillation can develop in a number of clinical situations, in association with different cardiac pathologies. When cardiac arrest victims are treated in the community it is often difficult to elucidate the cause of ventricular fibrillation. However the electrophysiological mechanisms in these conditions may be quite different. Does this affect the properties of the ventricular fibrillation waveform at the body surface, and could this be used as a diagnostic test? Some reports of frequency analysis of ventricular fibrillation have shown that certain frequencies predominate (3-7). We report the use of Fast Fourier Transform analysis in the study of ventricular fibrillation due to a number of different aetiologies.

Methods

Greyhound dogs of both sexes (weight 21-35 kg) were anesthetized with an intravenous injection of sodium pentobarbitone (May and Baker, 30-35 mg/kg), intubated, and ventilated with room air (Palmer Ideal pump, tidal volume 12 ml/kg: rate 18/min). Arterial blood pressure was recorded (M4, Devices) by a pressure transducer (Consolidated Electrodynamics 4-327-L221) from a femoral artery cannula. Esophageal (or in the open-chest experiments, pericardial) temperature was measured by an electronic thermometer (Comark 1604-2).

In some experiments 1-2 endocardial electrode catheters (USCI or Elecath 6-7F) were passed via the right external jugular vein to the apex of the right ventricle under X-ray screening (Watson MX2).

Induction of fibrillation

Ventricular fibrillation, produced by a different method in each of the 7 groups, was recorded for at least 15 minutes. Blood samples were taken from the femoral artery for assay of plasma potassium during the control period, and at the onset of fibrillation, when ventilation was stopped. At the end of each experiment (except Group 3), the heart was excised and weighed.

Group 1. Electrically-induced ventricular fibrillation in normal hearts (5 dogs). A bipolar pacing catheter was positioned in the right ventricle and connected to an isolated stimulator (Devices 2533). One hour later ventricular fibrillation was induced by brief rapid stimulation of the heart (5 ms pulses, 10 V, 100 Hz).

In a further 3 dogs electrode catheters were positioned in the right ventricle, via the external jugular vein, and at the apex of the left

ventricle, via the common carotid artery. Fibrillation was induced electrically after a control period of 1 h.

Group 2. Potassium-induced ventricular fibrillation (5 dogs), produced by the administration of KCl (8-16 mmol) over 8 seconds through a catheter positioned in the pulmonary artery (8).

Group 3. Glycoside-induced ventricular fibrillation (5 dogs). Ouabain was administered i.v, initially 40 ug/kg body weight, followed 30 min later by 20 ug/kg, and then 10 ug/kg every 15 min until ventricular fibrillation occurred (9).

For the next 4 groups, thoracotomy was performed through the left 4th intercostal space, and a snare was passed around the main anterior descending branch of the left coronary artery (LAD).

Group 4. Electrically-induced ventricular fibrillation in the non-ischemic heart (5 dogs). The snare was not tightened, and there was no myocardial ischemia. Fibrillation was induced electrically using the right ventricular catheter as in Group 1.

Group 5. Spontaneous ischemic ventricular fibrillation (9 dogs), within 1 hour of proximal occlusion of the LAD.

Group 6. Reperfusion ventricular fibrillation (5 dogs). After the LAD had been occluded proximally for 1 hour without spontaneous fibrillation, the snare was released. Ventricular fibrillation occurred within 1 hour of reperfusion.

Group 7. Electrically induced ventricular fibrillation during myocardial ischemia (4 dogs).

Recording system

Lead II of the electrocardiogram was constantly monitored (Hellige

Multiskriptor EK 22 amplifier). In groups 1 and 2 the unipolar endocardial electrocardiogram (V lead) was recorded from the apex of the right ventricle, simultaneously with lead II. Similarly, in groups 4-7, the epicardial electrocardiogram was recorded from the potentially ischemic zone (V lead) through an electrode sutured to an area of left ventricular epicardium below the snare.

The ventricular fibrillation was sampled at 400 Hz (8 bit A-D conversion) and recorded in digital form on cassette tape (10). Replay of the tape through the A-D converter reconstituted the analogue signal. In 6 experiments a multichannel FM tape recorder (SE Laboratories) was used simultaneously, to record the electrocardiogram signal in analog form. Power spectral analyses using the FM tape-recorder and the digital cassette-tape system, gave similar peak frequencies for both lead II and endocardial records, with a highly significant correlation coefficient ($r = 0.99$; 6 experiments).

Frequency Analysis

Fast Fourier Transform analysis of the recorded ventricular fibrillation signals was performed using a Bruel and Kjaer Spectrum Analyzer (Type 2031), at a frequency range of 0-50 Hz to match the frequencies of interest and the frequency response of the recording system. This microprocessor-based system (68000) transforms data from the time domain to the frequency domain, giving a logarithmic plot of the root mean square power contributed by each frequency (the power spectrum). Individual time windows of 4 sec duration were analyzed, and the resulting power spectra were averaged as analyses of either 8 or 40 sec periods of fibrillation. The effects of discontinuities between the

windows were reduced by a Hanning weighting. The screen cursor indicated the frequency with the largest amplitude and the -3 dB bandwidth, and the spectrum was plotted (Bruel and Kjaer type 2308). Sine-waves (2 mV) amplified by the electrocardiograph, recorded on the cassette tape, replayed and analysed in this fashion showed a frequency response of 0.35-55 Hz for the complete system (-3 dB limits).

Results

Heart-rate increased significantly in Groups 3, 5, and 7 prior to the onset of fibrillation (Table 1). Blood pressure did not change significantly from the initial values, before the development of ventricular fibrillation, except in Group 6, where there was a fall after reperfusion.

Fibrillation in the non-ischemic heart

Early after the onset of fibrillation the main feature of the frequency analysis was a strong dominant frequency, with adjacent frequencies showing a much lower amplitude (Figure 1). Smaller repetitive increases in amplitude occurred at higher harmonics of the dominant frequency, but these harmonics were low in power compared to the fundamental. In records from both the body surface (lead II) and the epicardium, after fibrillation for 120-160 secs there was a slower dominant frequency (with little loss of power), and a loss of power at frequencies higher than 20 Hz, compared with the initial 0-40 secs of fibrillation.

Averaged power spectra, each of 2 consecutive 4 sec periods of ventricular fibrillation, were obtained sequentially throughout the first 2 mins of ventricular fibrillation in a group of non-ischemic hearts (Figure 2). The initial frequency of 9.3 Hz rose to a maximum of over 11 Hz at 40 sec, fell slowly to the initial value by 72 sec, and then fell rapidly to some 5 Hz in the next 16 sec. Similar data were obtained from the same continuous records when 10 consecutive 4 sec spectra of

ventricular fibrillation were averaged (Figure 3). The frequency rose slightly in the first min, but then fell to 4-5 Hz and little further change occurred over the third min. The bandwidth at -3 dB was initially narrow (Table 2), becoming widest at the same time as the rapid fall in frequency, and then narrowing. Such changes in bandwidth could indicate a general slurring of the waveform. However this is unlikely, as the amplitude of these different peak frequencies did not change dramatically over this period.

Effect of the aetiology of ventricular fibrillation

The administration of increasing doses of potassium chloride caused a bradycardia, followed by ventricular tachycardia and fibrillation. Potassium-induced asystole did not occur. Ventricular fibrillation developed after a dose of 8 mmol in 3 dogs, and 16 mmol in the other 2. The arterial potassium concentration at the time of ventricular fibrillation ranged from 10.6 to greater than 16 mM/l. From the start potassium-induced ventricular fibrillation had a low dominant frequency (4.8 ± 0.8 Hz), which fell to even lower levels over the next 2 min (Figure 3).

Ventricular fibrillation induced by toxic doses (106 ± 10 ug/kg) of the cardiac glycoside, ouabain (Group 3), also showed a low initial dominant frequency (7.1 ± 1.1 Hz) , which then fell rapidly (Figure 3).

The initial dominant frequency of electrically-induced fibrillation in non-ischemic hearts was slightly higher in dogs after thoracotomy (12.2 ± 0.6 Hz, Group 4; Figure 4) than in closed-chest dogs (9.9 ± 0.7 Hz, Group 1; $p < 0.05$; Figure 3). However after the first 40 sec there was no significant difference between Groups 1 and 4, and the pattern of

change in frequency with time appeared to be similar in the 2 groups.

In Groups 5-7, the ischemic hearts, the dominant frequency was initially some 12 Hz, falling after 120 sec to 5-6 Hz (Figure 4). Surprisingly, as the fibrillation developed on a different basis in each group, there were no significant differences in frequency. The dominant frequency of fibrillation showed a similar value and time course, whether it was due to ischemia, reperfusion, or electrical stimulation during ischemia. However the frequencies of all 3 types of fibrillation in the ischemic heart fell more quickly than in non-ischemic fibrillation induced after thoracotomy ($p < 0.05$).

During occlusion of the anterior descending branch of the left coronary artery with a snare (Group 5), ventricular fibrillation developed in 2 dogs at 2 min, and in the other 7 dogs at more than 9 mins after occlusion of the artery. The initial dominant frequency of the fibrillation showed no significant correlation with the distance of the ligature from the left main stem orifice ($r = 0.32$), or with the duration of ischemia before fibrillation developed ($r = -0.24$).

In a further 2 dogs with an intact thorax, ischemic ventricular fibrillation was induced by pledgets of Gelfoam, injected through a catheter in the anterior descending branch of the left coronary artery. The dominant frequencies of fibrillation (initially 11.0 and 12.3 Hz) did not differ from the open-chest animals in Group 5 .

When the ligature around the coronary artery was relaxed, ST-segment elevation rapidly disappeared from the epicardial electrogram. During reperfusion of ischemic myocardium (Group 6), ventricular tachycardia was often associated with a significant fall in arterial

blood pressure (Table 1). Fibrillation developed at a mean time of 18.4 ± 2.2 min after reperfusion. There was no correlation between the duration of reperfusion before ventricular fibrillation, and the initial dominant frequency of the fibrillation.

Epicardial recordings

In non-ischemic hearts after thoracotomy (Group 4), the initial ventricular fibrillation recorded from the left ventricular epicardium(10.7 ± 0.4 Hz) had a slightly lower dominant frequency than ventricular fibrillation recorded by lead II in the same dogs (12.2 ± 0.6 Hz; $p < 0.05$). No significant differences were apparent after the first 40 sec.

By contrast, 4 dogs with spontaneous ischemic fibrillation (Group 5) showed frequencies of ventricular fibrillation recorded from the epicardium overlying the ischemic zone that were initially much lower than fibrillation recorded in lead II (Figure 5; $p < 0.005$). After 80 sec, this difference was no longer present.

In 2 dogs in which spontaneous ischemic fibrillation did not occur after coronary artery occlusion, and in which ventricular fibrillation was electrically induced during myocardial ischemia (Group 7), the frequency of ventricular fibrillation recorded from the epicardium of the ischemic zone was similar to that in lead II at all times. In the reperfusion group (Group 6), epicardial ventricular fibrillation also had similar frequencies to fibrillation recorded in lead II.

Endocardial recordings

Fibrillation recorded from the right ventricular endocardium (electrically-induced in non-ischemic hearts; Group 1) did not show the

rapid fall in frequency seen after 70 sec in lead II (Figures 6-7). Instead the frequency remained high, and even after 5 min of ventricular fibrillation, the dominant frequency was still 11.1 ± 2.0 Hz (Figure 7).

Although the dominant frequencies of ventricular fibrillation recorded from lead II and from the endocardium were initially similar, the power spectra showed some differences (Figures 1 and 6). This is not surprising, as simple observation of the ECGs of the 2 types of fibrillation suggested that endocardial ventricular fibrillation was more regular and vigorous, sometimes resembling the pattern of ventricular flutter. The power spectra of endocardial ventricular fibrillation showed more prominent harmonics, and the higher frequencies fell away more gradually. In 3 dogs simultaneous recordings of fibrillation were made from the right and left ventricular endocardial surfaces. Maintenance of the high frequency of fibrillation was observed at both ventricular endocardial recording sites; there were no significant differences.

Even in fibrillation induced by the intravenous administration of potassium, a method which caused a very slow fibrillation in lead II, the frequency of endocardial ventricular fibrillation began at 8.3 ± 1.0 Hz. The frequency of endocardial fibrillation fell subsequently by only a small amount (7.6 ± 1.5 Hz after 3 min of fibrillation, $p < 0.05$). In this group (Group 2), the dominant frequency of fibrillation in the endocardium was significantly higher than that in lead II after the first 80 sec ($p < 0.05$). At 160-200 sec the dominant frequency was 7.6 ± 1.5 Hz in the endocardium, and 2.2 ± 0.8 Hz in lead II.

In contrast with the other aetiologies, the frequency of endocardial ventricular fibrillation recorded after glycoside

intoxication in one dog from Group 3, fell to low levels after the first 40 sec and remained low.

Plasma potassium

Table 3 shows the control and post-ventricular fibrillation plasma potassium values in each of the groups. Only in group 2 (potassium-induced ventricular fibrillation was there a significant rise in plasma potassium ($p < 0.001$). The other groups showed no significant difference in plasma potassium before and after ventricular fibrillation. Although ouabain caused a small rise in plasma potassium, this was not statistically significant.

Discussion

These frequency analyses have confirmed that most of the power of the electrocardiogram during fibrillation is concentrated in a narrow band of frequencies. With continued fibrillation in the non-ischemic or ischemic heart, the dominant frequency shifts to lower levels, without much widening of the band-width. With fibrillation after the administration of potassium or ouabain, distributed to the whole heart, there was a lower frequency of fibrillation than in the non-treated heart. Local ischemia or reperfusion did not cause any reduction in the frequency of fibrillation recorded in the limb lead electrocardiogram, compared with that in the non-ischemic heart.

Non-ischemic hearts

In the experiments in which periods of 8 seconds were analyzed, the dominant frequency did not fall gradually from the onset of the fibrillation, but instead remained high (> 9 Hz) for 1.2 min, fell rapidly over a short period of time to about 5 Hz, and then levelled out. The frequency of epicardial fibrillation also fell rapidly in the second minute of fibrillation, at a similar rate to that in limb lead II. However in these non-ischemic hearts fibrillation recorded from the interior of the heart persisted at an unchanged frequency for several minutes.

Intracellular action potentials recorded during ventricular fibrillation do not show such a high frequency as 10 Hz. In the first minute of ventricular fibrillation in the reperfused dog heart there is rapid activity at some 5-8 Hz (300-480 per min), with unstable membrane potentials in diastole, and low amplitude action potentials (11,12).

However the remarkably short refractory periods of action potentials arising during incomplete repolarization allow rates of stimulation up to 500-700 per min (8-12 Hz; 13). As fibrillation progresses there is a loss of diastolic membrane potential, action potential amplitude and overshoot (11,12). After some 5-10 minutes action potentials of slow channel type, sensitive to verapamil but not tetrodotoxin, develop in the sub-epicardium (11), and the frequency of electrical activity slows to 2.5 Hz. Sano (12) and Czernecka (14) found asynchrony between the action potentials of neighbouring fibers, local electrograms and the surface electrocardiogram during ventricular fibrillation. In contrast Hogencamp and co-workers (15) demonstrated some degree of synchrony between recordings from different fibers. The high frequency found in the present experiments may be a composite frequency, due to asynchronous, slower frequency activity in many different areas of the heart (6). Alternatively many of the rapid, low amplitude responses seen with intracellular micro-electrode recordings are of larger amplitude in adjacent tissue, and can be recorded at the body surface.

Multiple extracellular recordings have been used by several groups to map the complex activation patterns in ventricular arrhythmias and fibrillation (2,16,17). In a more recent report 3-dimensional mapping of 232 bipolar sites was used to study the electrophysiological mechanisms in reperfusion ventricular fibrillation in the cat heart (18). The transition from ventricular tachycardia to fibrillation was associated with acceleration of the tachycardia to 10-12 Hz, shortening of the recovery periods in the myocardium to 70-100 msec, slow and blocked conduction, and the development of multiple activation wavefronts and

multiple re-entrant pathways in the ventricular myocardium. These findings, based on the detailed analysis of single beats of fibrillation, are consistent with the frequency spectra recorded at the epicardium, endocardium or body surface in the present study over periods of 4-40 sec.

The cause of the fall in frequency after some 1.5-2.0 min of continued fibrillation is uncertain. Slow response action potentials in the myocardium appear after some 5 min of fibrillation (11). It is likely that some metabolic change is affecting the electrophysiological properties of the myocardium, as a consequence of the global myocardial ischemia and high oxygen consumption during fibrillation. Potassium accumulates in the extracellular space during fibrillation, but in the first 10 mins this occurs more slowly and to a smaller extent than during local ischemia (19,20). A rapid fall in myocardial pH, starting after some 30-45 sec has been recorded in previously non-ischemic myocardium during fibrillation (21,22). Activation of phosphorylase, with rapid glycogenolysis is prominent in the first minute of ventricular fibrillation (23). However after 2 min of fibrillation the rapid accumulation of lactate in the myocardium is associated with depressed phosphorylase activity (23). Other changes, observed to occur during regional ischemia, and probably occurring in fibrillation, include a fall in intracellular high energy phosphates (24,25), and an increase in intracellular sodium (26). In the non-ischemic hearts of mongrel dogs, cardiopulmonary bypass prevented the fall in the dominant frequency of electrically-induced fibrillation with time (7). Further studies of the biochemical changes occurring during ventricular fibrillation may give a

basis for the changes in frequency. Such studies may also give a more rational basis for the improved clinical management of fibrillation in the diseased heart, or during cardiac surgery.

Further evidence of a critical time during canine ventricular fibrillation was provided by Geuze et al (27). Some 60-90 secs after the onset of fibrillation, at a similar time to the rapid fall in frequency seen in our study, successful defibrillation and recovery were rare.

The dominant frequencies of fibrillation recorded from the right ventricular endocardium and from lead II were initially similar in these non-ischemic hearts (Group 1). However the endocardial frequencies of fibrillation remained high for over 3 mins, while they declined in recordings from the epicardium and lead II. This is in agreement with a previous study, in which direct bipolar recordings from the left ventricular endocardium showed that electrical activity continued in the endocardium for many minutes, even while fibrillatory activity in the myocardium and epicardium was falling to low levels (28). While the data from limb lead recordings in the present study are generally in agreement with those of a recent report (7), we did not find a similar time-course for the decline in frequency in the surface and endocardial leads during fibrillation (7). Indeed the present study showed that the time-courses of fibrillation recorded from lead II and from the endocardium were entirely different, in accord with Worley et al (28).

The resistance of the endocardium to ischemia is well recognized. Following transmural myocardial infarction, a thin rim of subendocardial tissue remains viable and may be implicated in the subsequent development of arrhythmias (29-31). Several factors may contribute to this resistance

to ischemia (29,32). Purkinje fibres have large stores of glycogen, little contractile force, and a low metabolic rate. Oxygen may be supplied by the blood in the ventricular cavities (29). Purkinje fibres are more tolerant of hyperkalaemia, hypoxia and acidosis than other myocardial cells (32-33). In our study the frequencies of endocardial fibrillation recorded from the right and left ventricles were similar, and declined at a similar rate over 6 min.

Fibrillation due to potassium and glycosides

High extracellular concentrations of potassium reduce the cellular resting membrane potential, inhibiting activity in the fast inward sodium channels, and thus promoting slow responses and reentry (34). The marked difference between the frequencies in hyperkalaemia (Group 2), and those in the non-ischemic (Groups 1) and ischemic hearts (Groups 4-6) may indicate that fast channel availability is required for the initially high frequencies of ventricular fibrillation. During potassium-induced ventricular fibrillation endocardial recordings showed a persistent, high frequency of fibrillation. This is in keeping with previous reports, which have indicated that Purkinje fibers are resistant to high potassium concentrations (32,33).

Glycosides are also known to reduce the resting membrane potential (35) and may cause ventricular fibrillation by reentry; other mechanisms such as delayed depolarizations may also be implicated. It is of interest that despite the rapid ventricular tachyarrhythmias prior to ventricular fibrillation in the glycoside group, the subsequent fibrillation was quite slow.

Fibrillation in the ischemic heart

It is surprising that the frequencies of ischemic ventricular fibrillation, reperfusion ventricular fibrillation, and electrically-induced ventricular fibrillation in the presence of ischemia were so similar. There is considerable evidence to suggest that the electrophysiological mechanisms giving rise to fibrillation may be different in the 3 situations. In comparison to ischemic arrhythmias, reperfusion arrhythmias are more severe, and occur more abruptly and more predictably (36). Drugs which prevent one form may be ineffective in the other. Furthermore in the acutely ischemic heart, fibrillation after both occlusion and reperfusion required twice as much energy for defibrillation as electrically-induced ventricular fibrillation despite similar masses of ischemic tissue (37). This suggests a difference in the underlying metabolic or pathological conditions. Nevertheless we have shown that these differences do not affect the dominant frequency of the ventricular fibrillation in the limb lead. However the frequency of fibrillation in the ischemic zone was reduced.

Ventricular fibrillation recorded from the epicardium of the ischemic zone was much slower if the fibrillation arose spontaneously (Group 5), than if the fibrillation was induced electrically (Group 7). Fibrillation recorded from lead II had a similar higher frequency in both Groups 5 and 7, comparable to that of electrically-induced fibrillation recorded from the epicardium in the ischemic heart (Group 7). This may reflect the more widespread electrophysiological upset required for the spontaneous initiation of ischemic fibrillation, extending up into the superficial layers of the epicardium, since this was a non-penetrating

surface electrode. Conduction block, in epicardial recordings from the ischemic zone, is an important factor in the development of ischemic ventricular fibrillation (38). Our results indicate that, during the first minute of fibrillation, the ischemic myocardium has a lower frequency of electrical activity than the previously well-perfused myocardium. This disparity then disappears as severe ischemia and slowing of the frequency of fibrillation develops throughout the heart.

Clinical relevance

Observations on the frequency of ventricular fibrillation are not new. Using cinephotography Wiggers (39) described 4 stages in the time-course of ventricular fibrillation based partly on changes in frequency, and noted that "ventricular fibrillation is never an asynchronous incoordinate contraction of ventricular fibres". At the outset of these studies it was expected that the dominant frequencies of fibrillation would be significantly different in different clinical conditions. However the present studies indicate that Fourier analysis of the fibrillation waveform is unlikely to be of diagnostic value in the community management of sudden collapse.

The fall in frequency with duration of fibrillation indicates some critical change in cardiac electrophysiology, which may be relevant to the success or failure of cardiac resuscitation. The continued high frequency of fibrillation in the endocardium may also be of importance in defibrillation failure after 80 seconds of fibrillation. A more thorough understanding of the biochemical and electrical events occurring in the heart during ventricular fibrillation should lead to significant advances in resuscitation techniques.

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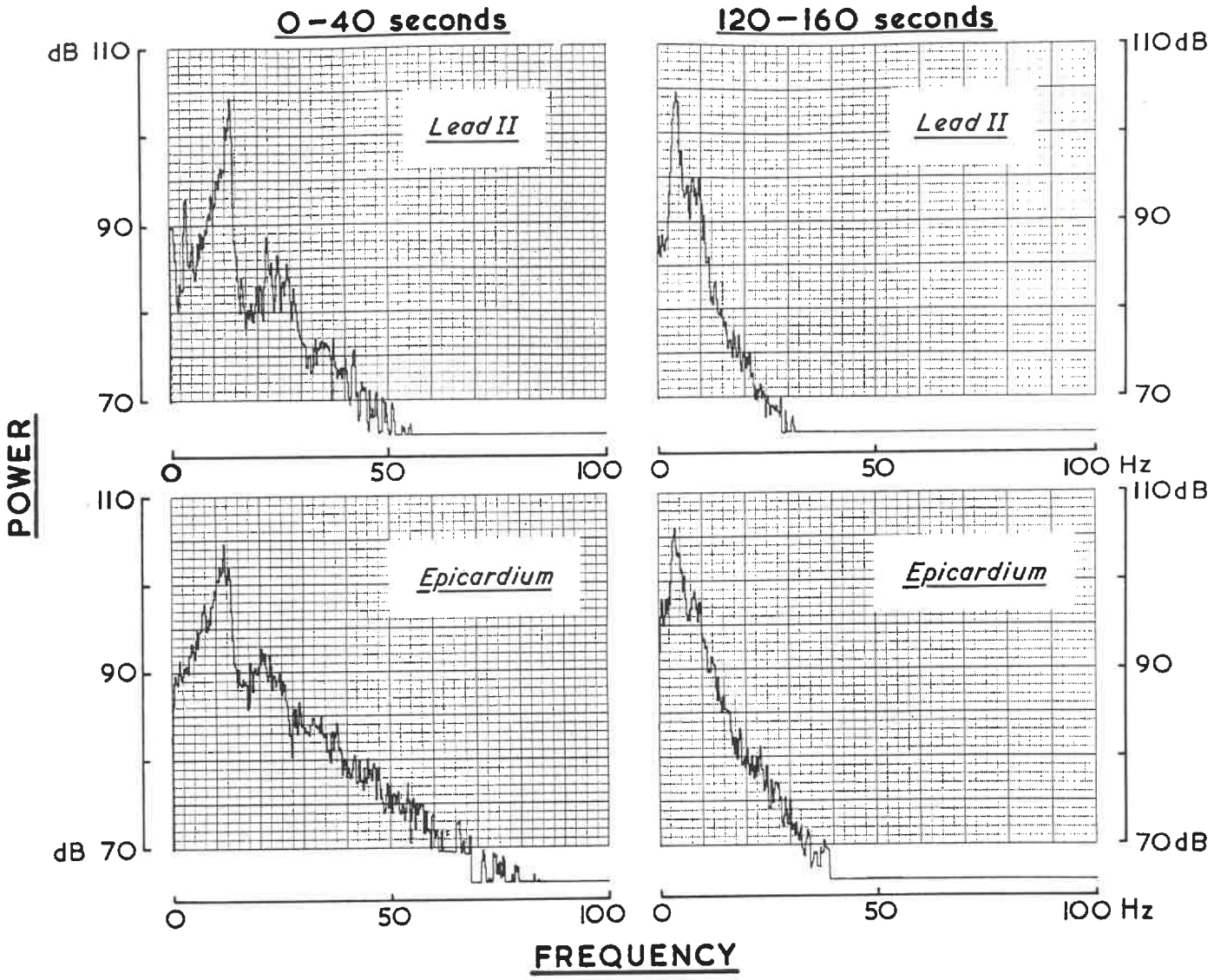

Figure 1. Power : frequency spectra of ventricular fibrillation show similar dominant frequencies in recordings from the body surface (Lead II) and the epicardium (unipolar lead connected to Wilson central terminal) in a non-ischemic heart. After fibrillation for 120-160 seconds the dominant frequency shifted to a slower frequency, with little loss of power. The contribution of higher frequencies was also reduced at this time, compared with the initial records (0-40 sec).

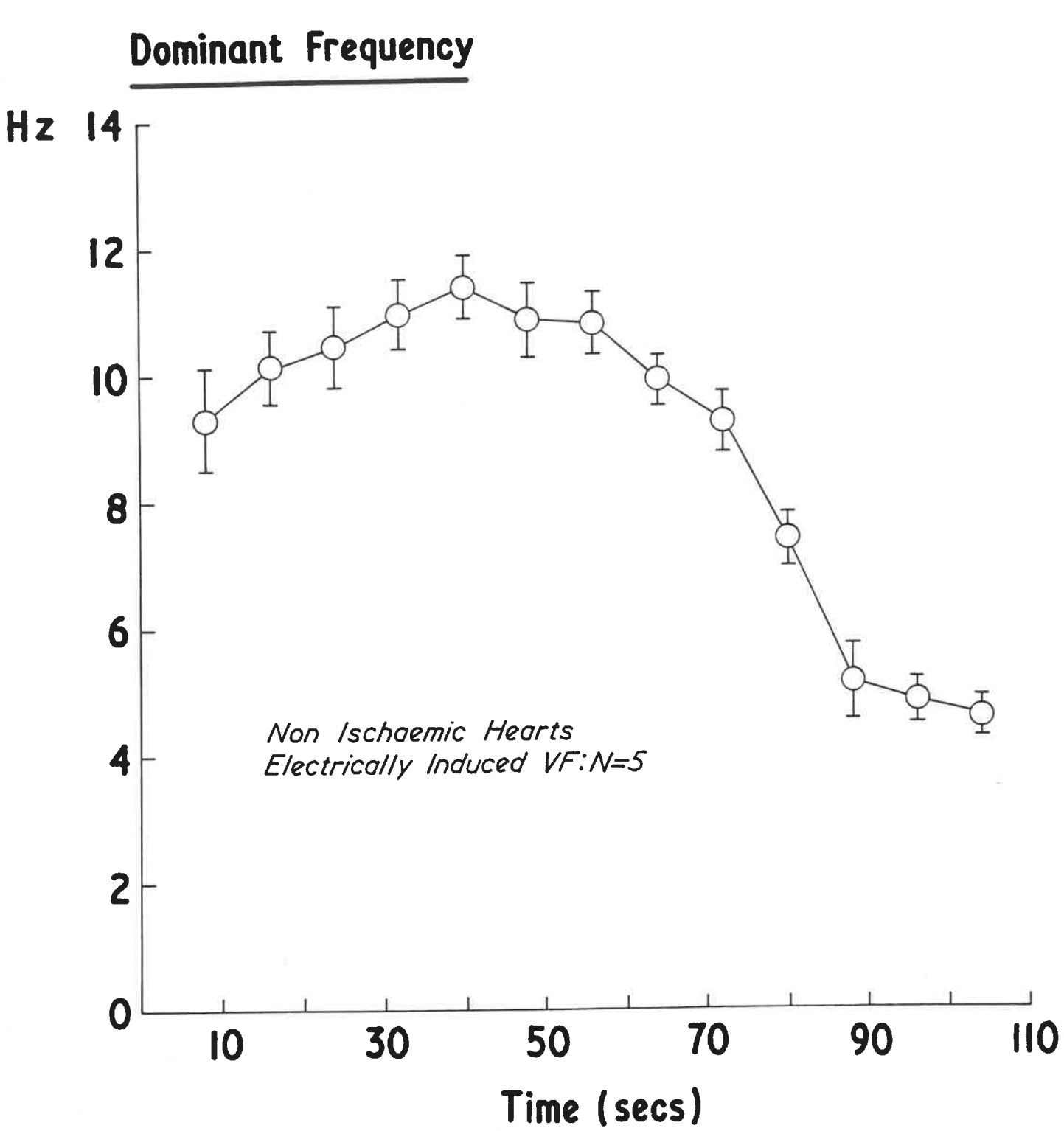

Figure 2. The change with time in the dominant frequency of 8 second periods of electrically-induced ventricular fibrillation recorded in lead II of the surface ECG in 5 non-ischemic hearts. The dominant frequency remained above 9 Hz for 72 sec, and then rapidly fell below 5 Hz in the next 16 sec.

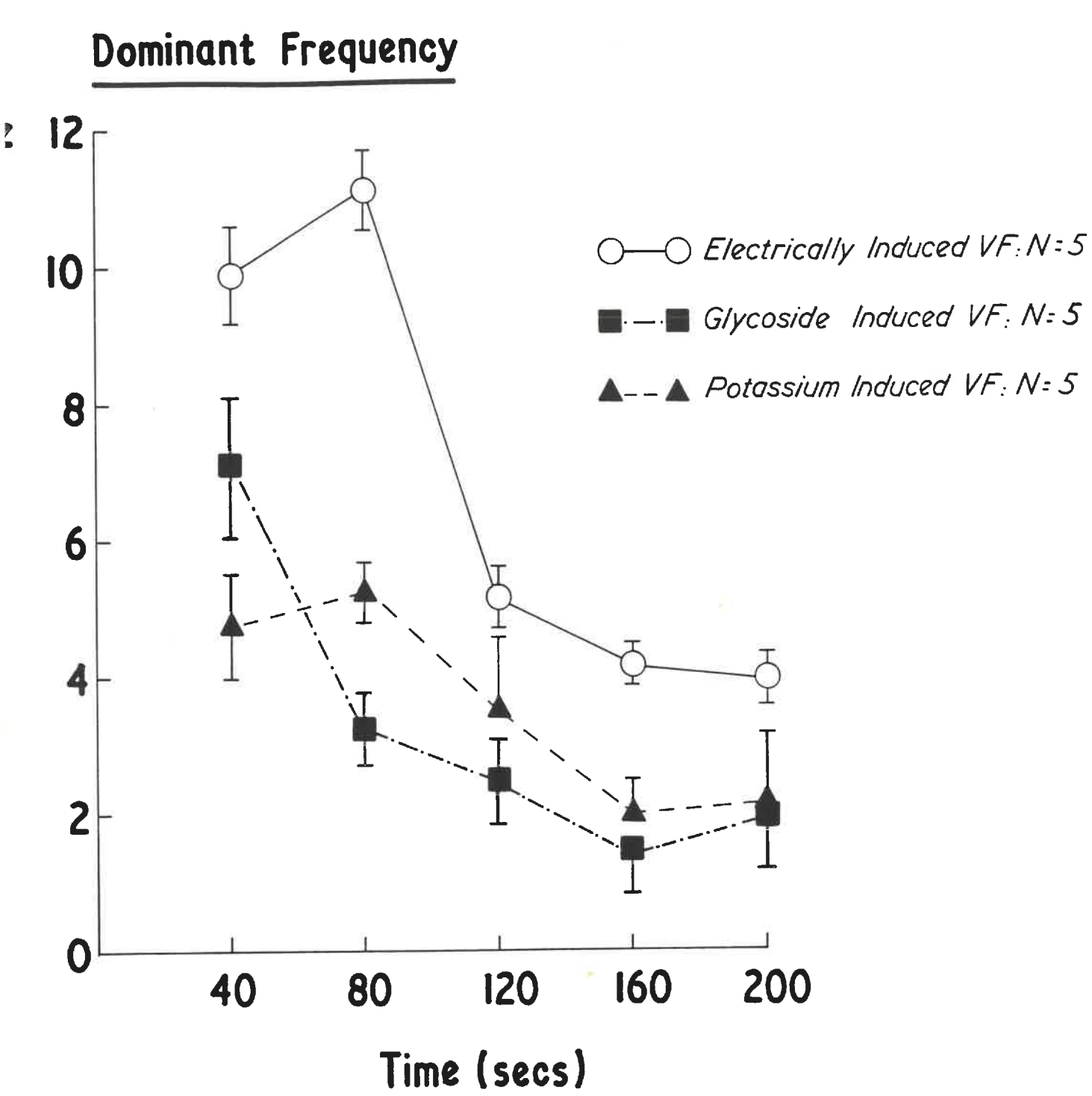

Figure 3. The change in the dominant frequency with time for ventricular fibrillation induced by the rapid injection of KCl into the pulmonary artery, by the slow intravenous administration of the cardiac glycoside, ouabain and by rapid electrical stimulation in non-ischemic hearts. At all times the dominant frequency after potassium or ouabain was significantly less than in electrically-induced fibrillation ($p<0.05$).

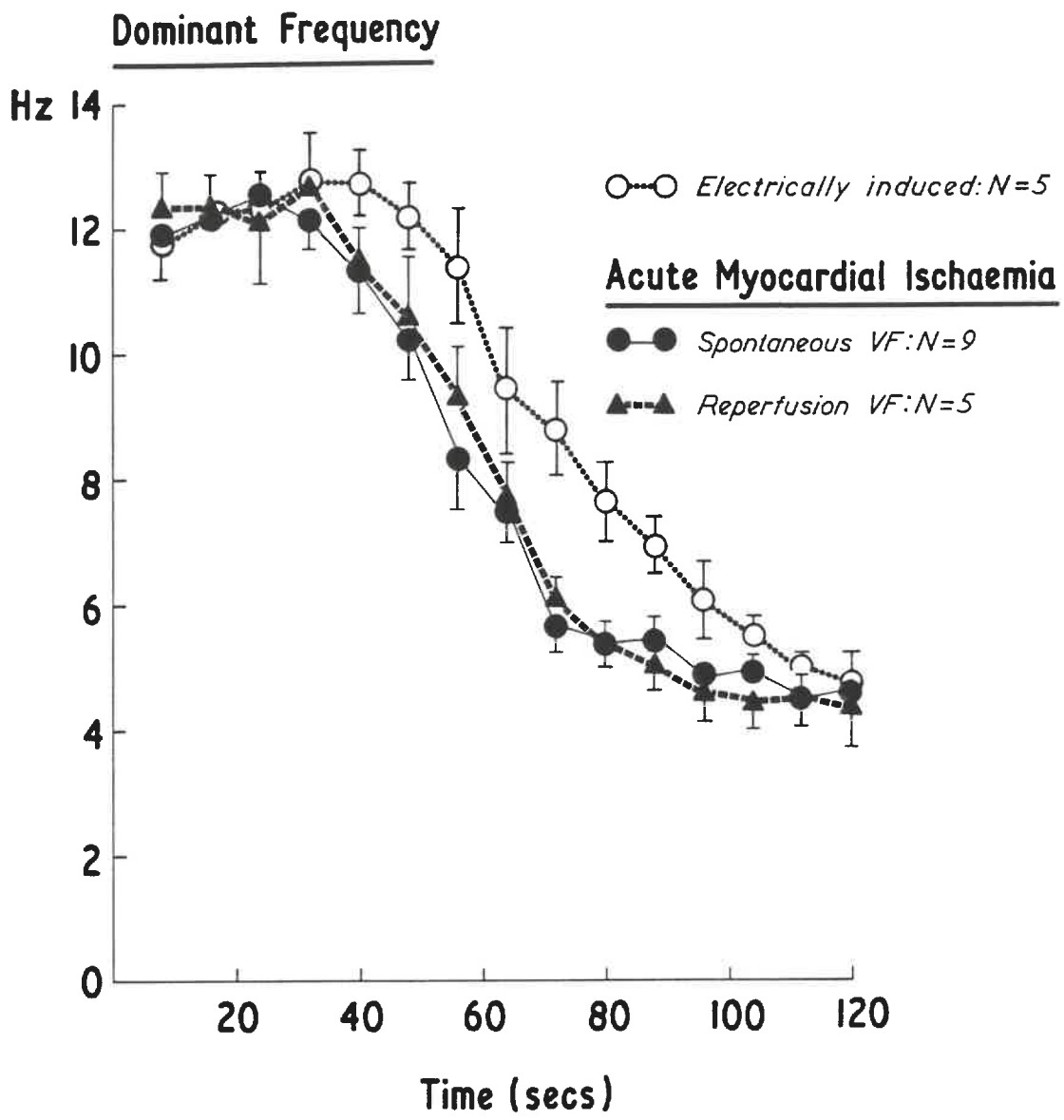

Figure 4. The time course of the dominant frequency of ventricular fibrillation resulting from acute occlusion of the anterior descending branch of the left coronary artery (9 hearts), from reperfusion of this artery (another 5 hearts), and from electrical stimulation of the heart in the presence of ischemia (4 hearts). There were no significant differences between the 3 groups at any time. Ventricular fibrillation after rapid electrical stimulation in dogs with a thoracotomy (5 non-ischemic hearts) showed a similar frequency to that in the ischemic hearts.

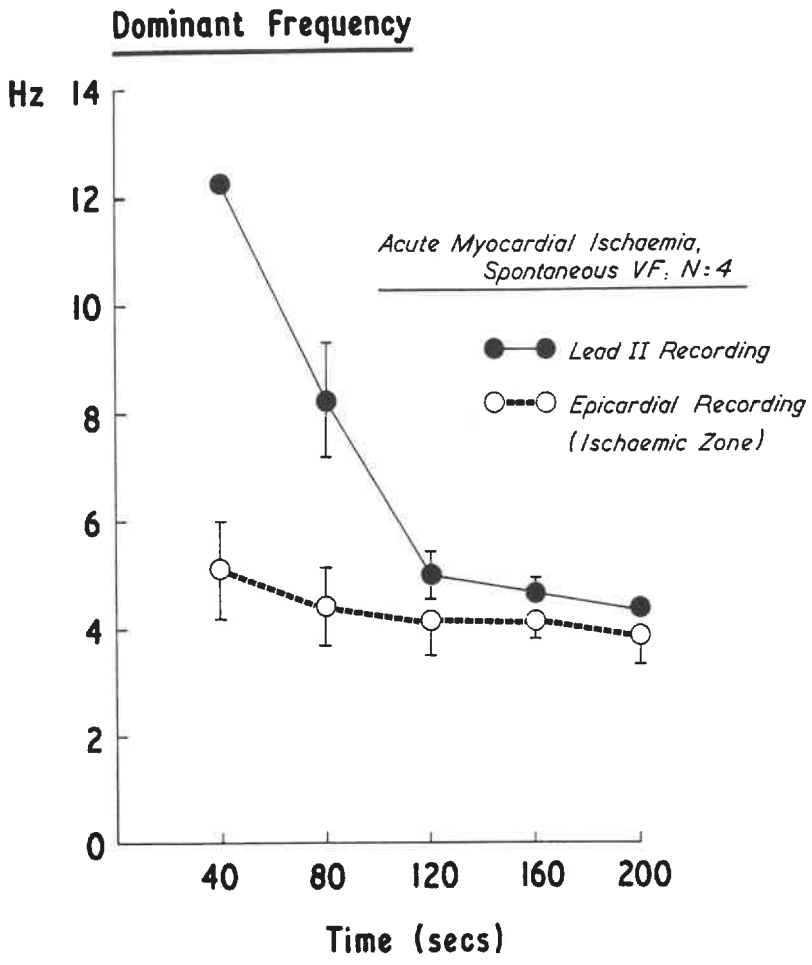

Figure 5. The dominant frequency of ventricular fibrillation in simultaneous recordings from the body surface (lead II) and the ischemic epicardium, after occlusion of the anterior descending branch of the left coronary artery in 4 hearts. A higher frequency was recorded initially at the body surface, but after 80 sec there was no significant difference in the dominant frequency at the 2 sites.

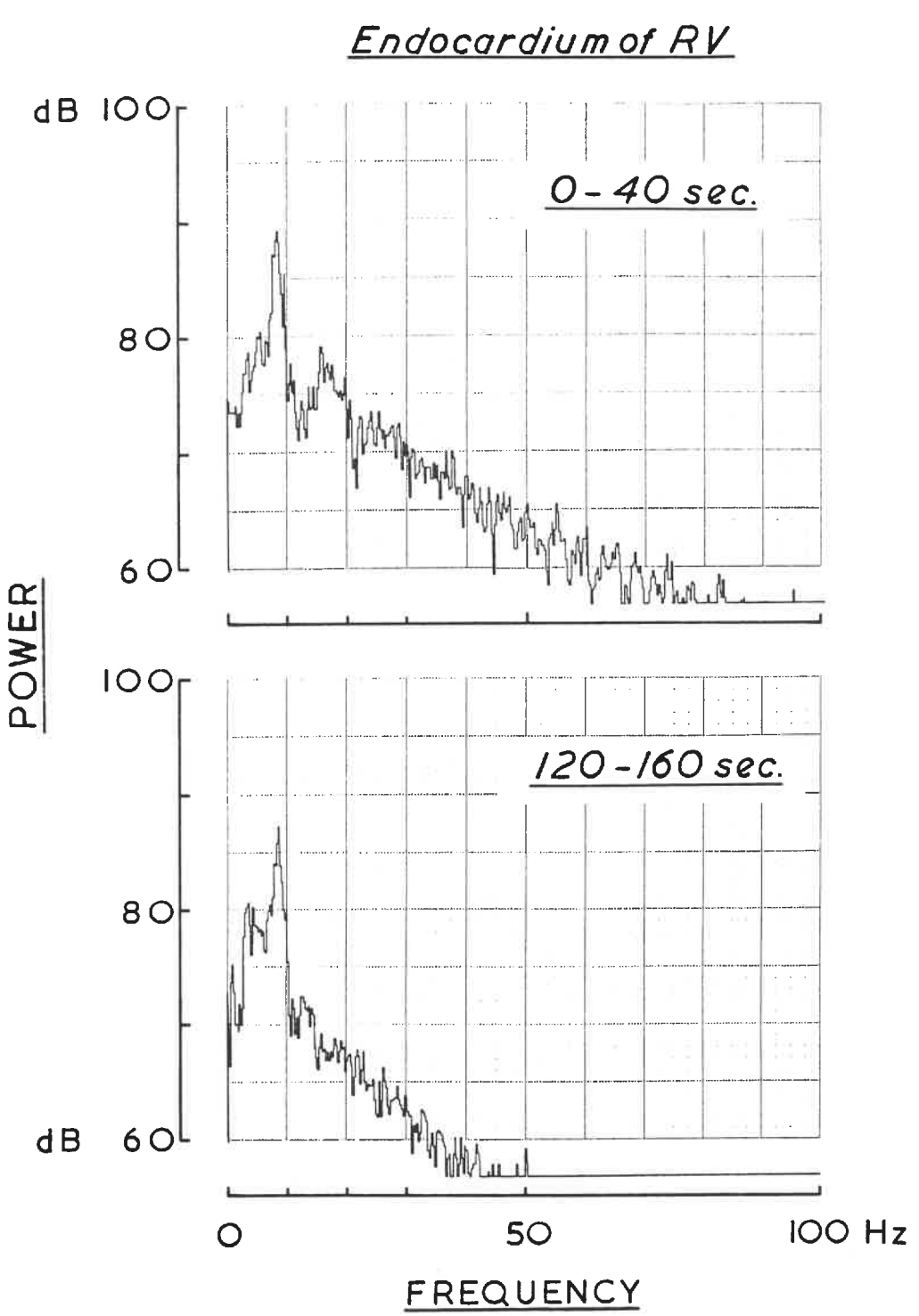

Figure 6. Power : frequency spectra of ventricular fibrillation recorded from the endocardium of the right ventricle on an FM tape recorder. As the fibrillation continued there was some fall in amplitude of the higher frequencies, but the dominant frequency remains high (8.8 Hz initially; 9.0 Hz at 120-160 sec).

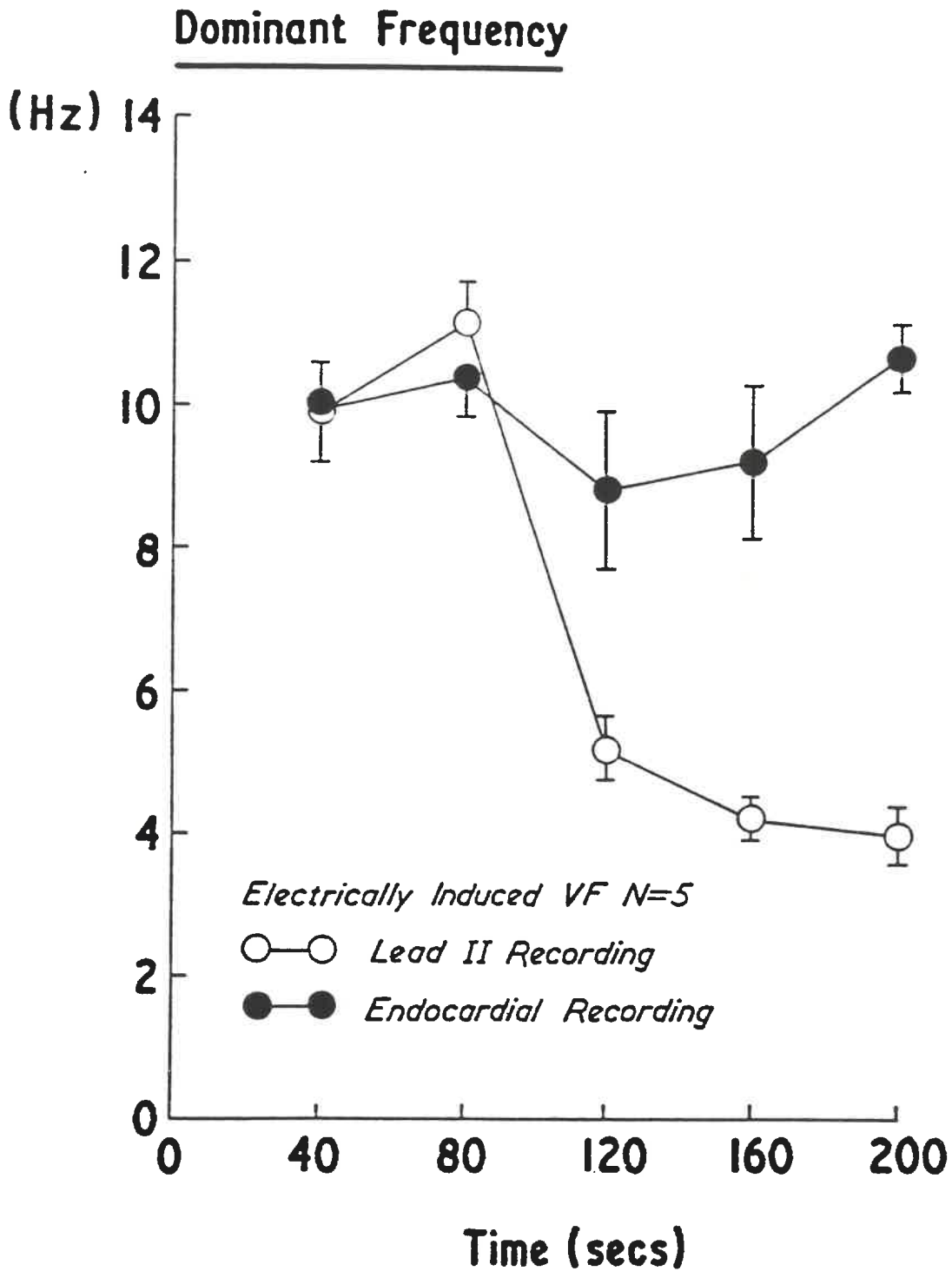

Figure 7. The time course of the dominant frequency for electrically-induced ventricular fibrillation in 5 non-ischemic hearts, in simultaneous recordings from lead II, and from a unipolar endocardial lead in the right ventricle. Although there was no significant difference initially, the Lead II recording at 100-200 sec showed a significantly slower dominant frequency than the endocardium.

	BODY WEIGHT KG	HEART WEIGHT G	VENTRICULAR RATE per min		MEAN ARTERIAL PRESSURE mm.Hg	
			Initial	Pre VF	Initial	Pre VF
Group 1 Non-ischemic electrical closed chest	25.3±1.0	271.6±12.1	110.2±12.8	130.9±18.4	108.6±5.8	122.4±11.5
Group 2 Potassium	29.2±1.4	319.0±12.9	124.2±16.3	145.8±15.2	123.4±12.4	142.7±10.1
Group 3 Ouabain	30.7±2.9	-	113.0±6.7	192.6±12.4*	123.4±14.4	149.3±28.5
Group 4 Non-ischemic electrical thoracotomy	26.9±0.7	323.1±16.5	155.8±6.8	155.8±7.5	125.8±15.5	125.6±5.0
Group 5 Ischemic Spontaneous VF	27.6±0.8	313.8±13.1	151.6±4.7	164.2±7.1*	132.3±5.8	132.5±8.8
Group 6 Reperfusion VF	28.6±1.5	291.3±13.7	150.2±9.7	203.0±43.5	117.7±2.9	96.3±3.5*
Group 7 Ischemic electrical	28.2±2.3	311.7±14.1	146.4±4.4	171.5±10.2*	127.5±9.6	128.0±9.5

* $p<0.05$

TABLE 1. Body weight, heart weight, ventricular rate and mean arterial pressure in the 7 groups of dogs. The significance of the differences from the initial values is shown. Body weight did not differ significantly between the groups. Heart weight in group 1 was significantly lower than in groups 2,4 and 5 ($p<0.05$).
The initial ventricular rate was significantly higher in groups 4-7 (thoracotomised animals) than in groups 1-3 (closed chest animals).

TABLE 2. The mean dominant frequency (±SEM) with -3dB limits of the first 40 seconds of ventricular fibrillation in Groups 1-7.

	Dominant Frequency	-3dB limits	
		Lower	Upper
Group 1 Non-ischaemic electrical closed chest	9.9±0.7	9.3±0.6	10.5±0.6
Group 2 Potassium	4.8±0.8	4.0±0.7	5.8±0.8
Group 3 Ouabain	7.1±1.1	6.5±1.2	7.5±1.1
Group 4 Non-ischaemic electrical thoracotomy	12.2±0.6	11.7±0.6	12.8±0.7
Group 5 Ischaemic spontaneous VF	12.3±0.2	11.6±0.3	12.8±0.3
Group 6 Reperfusion VF	12.2±0.4	11.4±0.3	12.8±0.4
Group 7 Ischaemic electrical	11.7±0.5	10.9±0.2	12.3±0.4

	PLASMA POTASSIUM (mmol/l)	
	Initial	Post VF
Group 1 Non-ischemic electrical closed chest	3.9±0.2	3.8±0.2
Group 2 Potassium	3.9±0.2	15.1±1.1**
Group 3 Ouabain	3.9±0.2	5.1±0.3
Group 4 Non-ischemic electrical thoracotomy	4.0±0.1	3.8±0.3
Group 5 Ischemic spontaneous VF	3.7±0.1	3.8±0.1
Group 6 Reperfusion VF	3.9±0.05	4.1±0.1
Group 7 Ischemic electrical	3.9±0.1	3.7±0.1

TABLE 3. Plasma potassium at the beginning of the initial period, and directly after the onset of ventricular fibrillation. The plasma potassium increased significantly in group 2 ($p<0.001$**).