THE USE OF THE MINIATURE ACCELEROMETER IN THE DETECTION OF ABNORMAL DIASTOLIC SKIN SURFACE MOVEMENT AT THE CARDIAC APEX

During the early diastolic (relaxation) phase of the cardiac cycle, blood from the left atrium flows rapidly into the left ventricular chamber (the rapid filling phase). The manner in which the ventricle receives this surge of blood depends on a number of factors such as the compliance of the ventricular muscle, the left ventricular end systolic pressure, etc. Pathologic variation of these factors is known to result in exaggerated movement of the heart and overlying skin surface during the filling phase. In a pamphlet called Examination of the Heart by the American Heart Association, the following appears:

> "During early diastole, the brief outward chest wall movements associated with a left ventricular gallop (VG, left ventricular filling sound or S_3) may occasionally be seen or felt, even when the vibrations are not audible. In children and young adults, the presence of an early diastolic ventricular filling sound (S_3) and movement may be a normal finding. On the other hand, the development of such a movement or sound (left ventricular gallop) in a patient with heart disease usually signifies ventricular failure. Patients with acute transmural myocardial infarction frequently develop a transient palpable and audible ventricular filling (S_3) sound, which reflects the acutely altered ventricular compliance..."

In addition to the abnormal response of the left ventricle to the early rapid

filling phase, another abnormal movement in late diastole can result from contraction of the left atrium which attempts to inject additional blood into the left ventricle. The pamphlet continues:

> "During late diastole, immediately prior to the first heart sound, the left atrial contribution to the apex impulse--referred to as the atrial impulse or "A" wave--may be detected. In most instances a palpable atrial impulse coincides with an audible atrial gallop (AG) sound or S_4... A palpable atrial impulse at the apex, with its associated sound, may be found in normal children with thin chest walls or even in some normal adults if the P-Q interval is long and if the circulation is hyperdynamic. The presence of such an atrial pulsation or sound may have no significance if there is no other evidence of heart disease. On the other hand, these findings may have considerable significance in certain circumstances. For example, in some patients with ischemic heart disease a palpable atrial impulse at the apex may develop or become more prominent during an attack of angina pectoris or even during exertion without chest pain. An atrial gallop and/or a palpable atrial impulse occurs very frequently in patients with acute transmural infarction and reflects the altered compliance of the left ventricle, with or without early ventricular failure..."

The frequency range of these diastolic vibrations is so low as to be almost subaudible to the human ear using the stethoscope. Indeed, these sounds are often extremely subtle and difficult to assess, even in the quietest of examination rooms. It has long been known that the vast majority of chest wall motion due to cardiac function is subaudible to the human ear. In

figure 1 below is shown the shaded portion of the skin surface vibration which falls above the threshold of audibility. While the vibratory information concerning valvular sounds and murmurs is well studied with the stethoscope, the diastolic vibrations giving rise to the third and fourth heart sounds (ventricular and atrial gallop sounds) exist predominantly within the subaudible portion of this graph. It is our feeling, based on these observations and on our own results to date, that the exaggerated skin surface motion associated with pathologic alterations of diastolic heart motion is quite poorly studied with the stethoscope and that the majority of the clinically relevant information concerning the phenomenon is presently missed by the physician using conventional techniques.

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Agress CM, Fields LG
New method for analyzing heart vibrations.
Am J Cardiol, Aug., 1959,
pp 184-190.

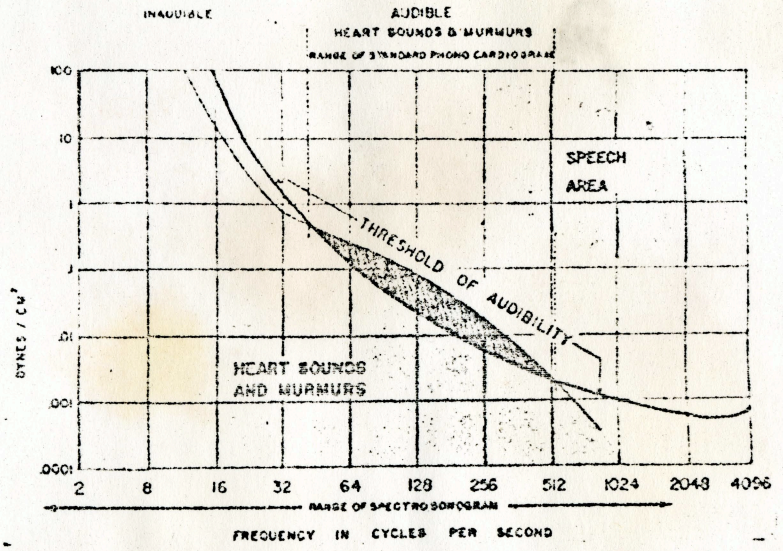


FIG. 1. This graph indicates an average threshold of audibility and shows how small a portion of the vibratory spectrum of the heart lies above the threshold of audibility.

The fact that the small percentage of the diastolic vibration which is audible has proven over the years to be an important diagnostic and prognostic sign is suggestive of the potential clinical value of the assessment of this

diastolic skin motion if it is monitored in its entirety. We have thus sought to utilize the miniature accelerometer as a sensor of two distinct parameters of skin surface motion; velocity and acceleration. In the normal subject, the diastolic skin surface velocity is of low frequencies and is no larger than the systolic portion of the velocity waveform. In figure 2 is shown the skin surface velocity tracing at the cardiac apex from a patient who is in congestive heart failure. An upward movement of the waveform corresponds to an outward velocity of the skin surface while a downward movement of the waveform corresponds to an inward velocity of the skin surface. Note that the relationship of the early diastolic rapid filling wave (RFW) to the systolic wave is markedly exaggerated. The third heart sound (S_3), which is the audible portion of this filling vibration, is a very obscure component in the accompanying phonocardiogram (PCG). In figure 3 is shown a velocity tracing from another patient who is in a more severe condition of congestive heart failure. Here the rapid filling wave (RFW) is by far the dominant feature of the skin surface velocity waveform at the apex. Since these patients were only accessible to our machine when they were able to be brought to the cardiac graphics laboratory, we were not able to study the changes of the waveform from one day to the next. In figure 4a and 4b is an example of a waveform obtained on different days as the patient improved. This patient was a 24 year old man who entered the hospital with myocarditis subsequent to a presumed viral infection. In figure 4a is shown the velocity tracing at the apex on a Friday afternoon when the patient's condition was serious. A rapid filling wave (RFW) is clear on this tracing. In figure 4b is shown the velocity tracing obtained at the same position on the chest wall on the following Monday morning after he had

received a weekend of treatment. Note that the ratio of rapid filling wave (RFW) to systolic wave has markedly reduced to the point that no clear RFW is seen on the tracing. These two tracings were recorded at different "gain" settings, which accounts for the overall change in the size of the signal. Nevertheless the ratio of diastolic to systolic motion has clearly reduced after treatment. The patient was much improved on the basis of his other physical signs as well. If a bedside skin surface velocity recorder had been used on this patient during this treatment period, a progressive and clear reduction of the relative size of the RFW to the systolic velocity would have been seen. It is our current impression that the relative size of the RFW to the systolic motion of the skin surface at the cardiac apex correlates directly to the state of congestive heart failure and that the time course of the patient's condition and the effects of therapeutic intervention can probably be monitored with the signal. A three year, $164,000 grant to study this possibility along with other considerations of the diastolic heart movement has recently been recommended for funding by the National Institute of Health (National Heart, Lung, and Blood Insittute). At the present time the only method of monitoring the mechanical function of the failing heart is with the Swan Ganz catheter, a painful and expensive technique which carries with it a certain finite mortality rate. (There are companies who sell portable gamma imagers, etc. but these are incredibly expensive). We anticipate that our research will establish the clinical utility of a simple, inexpensive, non-invasive monitor utilizing the miniature accelerometer to assess the mechanical aspects of heart function as a complement to the assessment of the electrical aspects as done with the EKG. This is especially important when it is considered

that the majority of deaths occurring in the coronary care unit are "mechanical" deaths (loss of contractile force resulting in congestive heart failure) and not "electrical deaths" (due to intractable arrhythmias). This loss of contractile force is totally unnoticed by the EKG, except to say that the signal goes flat when the heart finally stops beating.

The velocity signals of figures 2, 3 and 4a, 4b were obtained by electrical integration of the acceleration signal derived from the miniature accelerometer. In order to produce a reasonably stable signal of velocity, it was necessary to filter the integrated signal substantially. The result of this filtration is an inevitable distortion of the low frequency components of the signal with potential error in the assessment of the waveform. While we do not believe that the signals of figure 2, 3, and 4a, 4b are significantly distorted due to this filtration, it is difficult to determine the extent of distortion in the full range of patients, since some have very low heart rates with large low frequency systolic and diastolic skin movements. This distortion can be improved by the substitution of the simple filters used so far with a properly designed Butterworth or Chebyshev high pass filter. We have recently turned our interest to the study of the skin acceleration as a more stable parameter of skin surface motion. This reorientation was particularly important in the monitoring of skin motion after the exercise stress test, when respiration is greatly exaggerated. The velocity signal is totally unstable during this time and we believe that it is unrealistic to attempt to impose stability on the signal through filtration. The skin surface acceleration signal is, however, extremely stable against respiratory artifact. Also, the diastolic velocity exaggerations of figures 2, 3 and 4a, 4b would likewise be expressed

in the acceleration signal and is, we believe, a viable alternative to the velocity signal for the continuous bedside monitor in the coronary and intensive care unit. A decision will have to be made in the near future whether to concentrate solely on the skin surface acceleration signal for both the continuous monitoring and stress test applications of the device or to use the skin surface velocity signal in the continuous monitoring application.

Regardless of whether the velocity signal or the acceleration signal is used in the continuous monitoring application in the coronary care unit, the acceleration signal is certainly best for the exercise stress test application. We have studied the acceleration signal on approximately 24 patients in conjunction with the exercise stress test. This was done with the help of Dr. David Sheps, the director of the exercise stress test lab. We recorded a pre-exercise signal when the patient was reclining on the examination bed and a post-exercise signal immediately after the test and as soon as the patient could return to the bed and assume the reclining position. (Indicated on the recording as "supine", although actually with the head at about 20°). Initially, we also recorded a pre and post exercise acceleration signal while the patient was standing on the treadmill. In this way we were able to record the signal within ten seconds of termination of the treadmill walk. We found, however, that if abnormal diastolic accelerations were going to occur, they would persist for several minutes into the recovery period and in fact would reach a maximum of exaggeration at about three minutes after the termination of the walk while the patient was lying on the bed. We thus eliminated the recordings made while the patient was standing. In these tests I did not have the ability to display the waveform on the same paper as the EKG, since the machine was not

modified to accept the acceleration signal. Instead, the signal was recorded separately on an Electronics for Medicine VR6 recorder along with EKG lead V_5 at a paper speed of 50 mm/sec. The acceleration signal was AC coupled to the DC input channel of the VR6 with a high pass filter whose time constant was greater than 2 seconds. Also, the signal was passed through a low pass filter with a corner frequency of 150 Hz and a roll-off of 6 db/octave. Also, the DC input channel incorporated a low pass filter with a corner frequency of 250 Hz. I did not use the hybrid EKG-accelerometer clip. Instead, I attached the miniature accelerometer directly to the skin using double sided tape. It was placed as close as possible to the cardiac apex, usually between precordial EKG leads V_4 and V_3. Since the overall size of the acceleration waveform increased several fold as a result of the treadmill walk, the pre and post exercise signals are not recorded at the same "gain" setting. Therefore, this discussion applies strictly to the ratio of the diastolic to systolic amplitudes.

In figures 5a, 5b, and 5c are shown the waveform recorded from a patient with a known prior myocardial infarction (in 1976) and with increasing chest pain since January of this year. An upward movement of the signal corresponds to an acceleration of outward skin movement while a downward movement corresponds to a deceleration of outward movement. In figure 5a the pre-exercise waveform is essentially normal with an uneventful rapid filling wave (RFW) in comparison to the systolic portion. During the treadmill test the patient experienced chest pain and had 2.0 mm of ST segment depression in several EKG leads. His post exercise tracing is shown in figure 5b. Note here at three minutes into the recovery period that the rapid filling wave (RFW) has come to dominate the signal and is clearly abnormal. Nitroglycerin was given to the patient and another recording was made twelve minutes into the recovery period (figure 5c). At this time the EKG and the acceleration signal have returned to normal. It is significant that the attending physician did not

hear a third heart sound during the recovery period, although a clear abnormality was recorded by the accelerometer.

In figures 6a and 6b are shown another example which involved a different type of diastolic skin acceleration abnormality. This is a man who had a known infarction in 1972 and who now has angina pectoris. This man is also suspected of being in the initial stages of congestive heart failure. In figure 6a the pre-exercise tracing shows an unremarkable waveform. During the treadmill test the patient had chest pain and 1.5 mm of ST segment depression in two precordial EKG leads. The signal recorded approximately three minutes into the recovery period (figure 6b) shows a markedly abnormal diastolic movement. This exaggerated movement is highly sensitive to respiratory variation which we believe to be due to expiratory augmentation of left atrial filling and not simply to a change in the volume of air in the lungs. This movement is best considered as a summation of the rapid filling and left atrial contraction events, since at this high heart rate the two tend to occur simultaneously. This abnormality is actually an abrupt deceleration of outward movement and contains some relatively high frequency components. This is very different from the previous example of figure 5b which is initially an exaggerated acceleration of outward movement and which is composed of lower frequencies. The waveform is telling us something different about the hemodynamic alterations of this heart than in the example of figure 5. Our initial interpretation is that this is primarily an atrial contraction abnormality (correlated with an S_4) due to an elevated left ventricular end diastolic pressure, which is the result of an exercise induced loss of contractile force. If this is true it confirms the suspicion that the patient is entering exercise induced

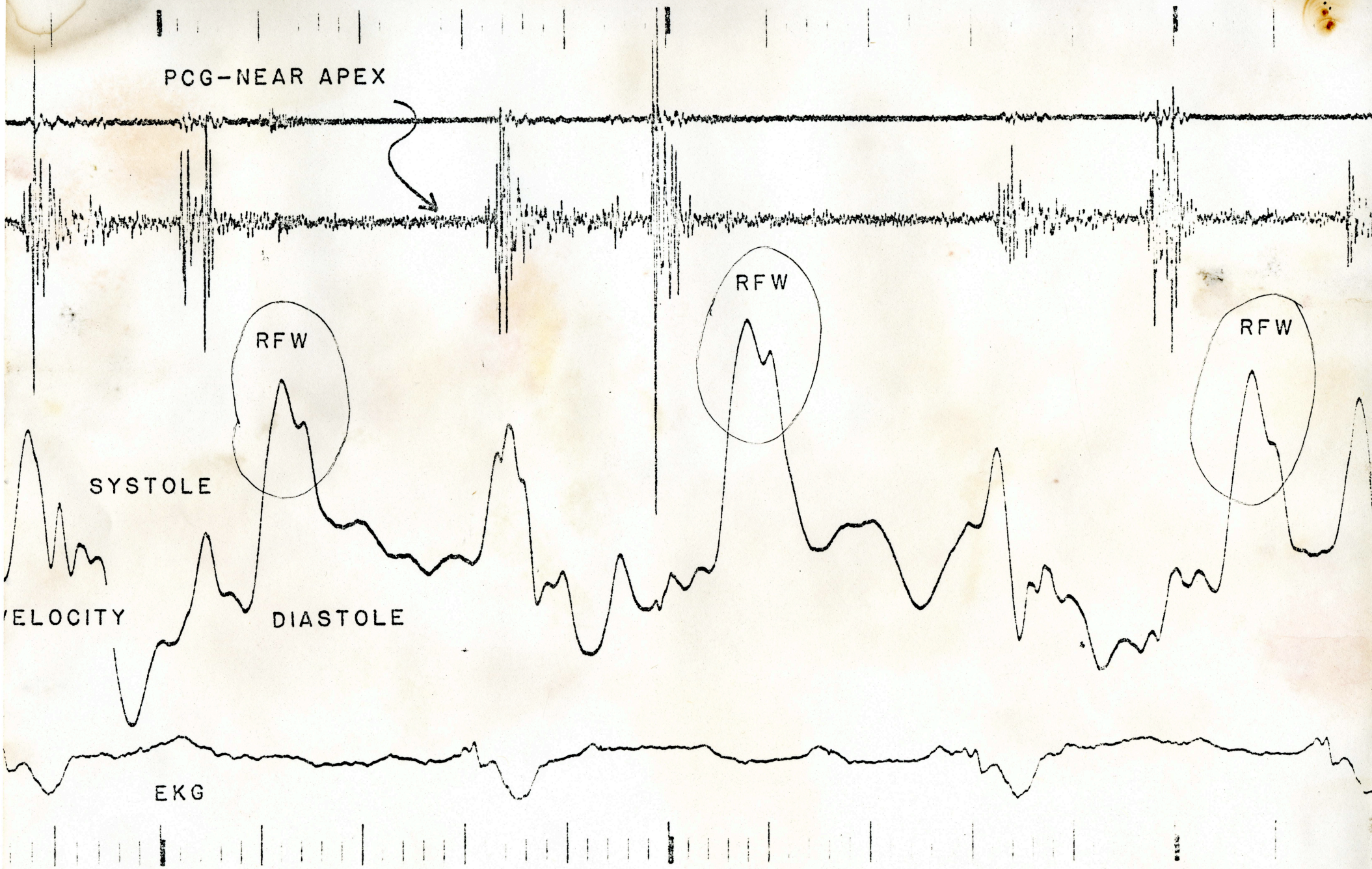
congestive heart failure and is clearly relevant to the assessment of the patient's condition. It appears on inspection of the accelerometer tracing that this diastolic vibration would in fact dominate the heart sounds as perceived by the stethoscope. If this is true, it would be possible for the physician to misinterpret this abnormal vibration as a normal first heart sound, since it comes quite close in timing to the onset of ventricular systole. The recording on stripchart paper along with the EKG shows this vibration to occur before the QRS complex and thus to be diastolic in nature.

In the final example is shown a case in which the EKG was marginally positive and in which the diastolic accelerations were totally normal. This is a man with a documented infarction ten days prior to the stress test. He completed stage three of the standard Bruce protocol with no chest pain or dyspnea. As seen in figure 7a and 7b there is no diastolic exaggeration either before or after the stress test. Our initial interpretation is that the man has no significant remaining live ischemic tissue subsequent to his infarction (no rapid filling wave) and that his heart suffers no significant loss of contractile force as a result of the stress test (no elevation of the left ventricular end systolic pressure so as to produce an exaggerated atrial contraction wave).

We are in no position at this point in our studies to make a definitive statement as to the meaning of the diastolic exaggeration, or lack thereof, in the acceleration signal in conjunction with the findings of the EKG test. It is possible that the acceleration signal will prove to be more sensitive and/or specific than the EKG as a predictor of ischemic heart disease. Whether or not this is true, we have found that the diastolic accelerations do not simply mimic the results of the EKG test and it is a tantalizing

field of investigation to find out exactly what the signal is telling us.

As mentioned above, it is well accepted that the development of a third or fourth heart sound (S_3 or S_4) as perceived with the stethoscope is relevant to the assessment of the patient undergoing the exercise stress test. The acceleration signal has already proven itself as a very sensitive and practical means of recording these conventional sounds on the EKG stripchart paper. Therefore, regardless of the results of our upcoming studies of the acceleration signal, the clinical value of the technique in expanding the capabilities of the EKG machine cannot be denied.



PAPER SPEED-100mm/sec

FIG. 2

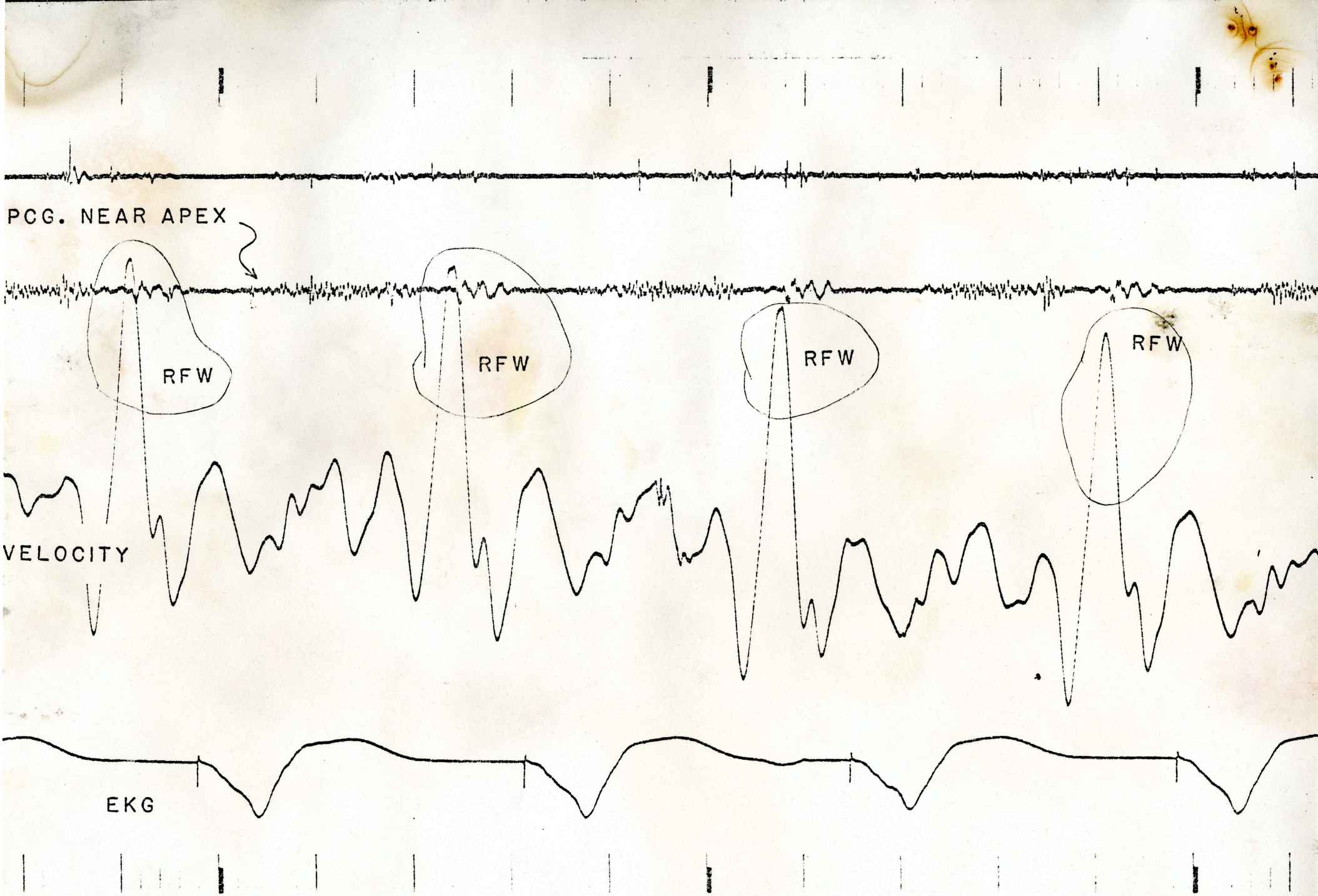


FIG. 3

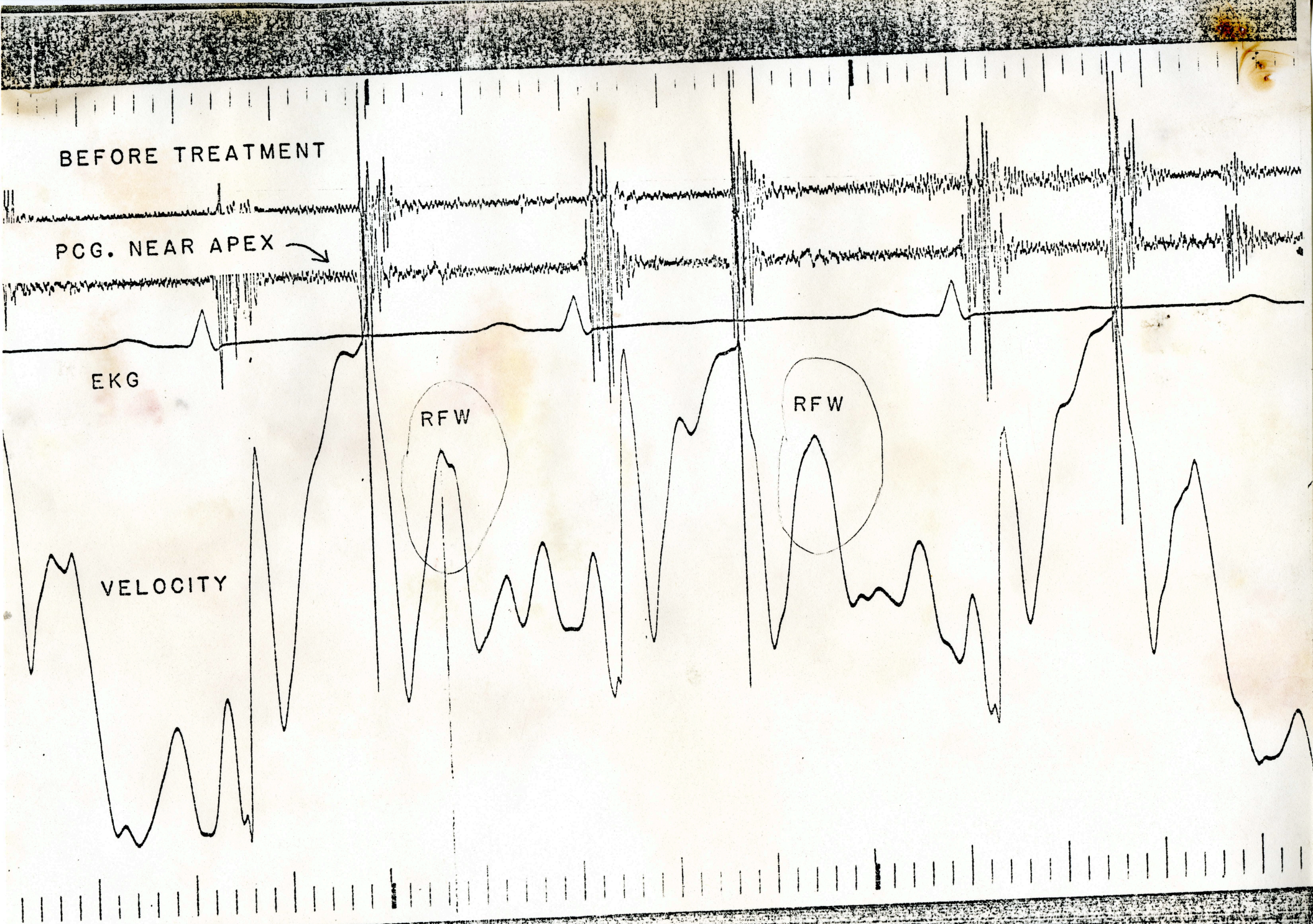


FIG. 4a

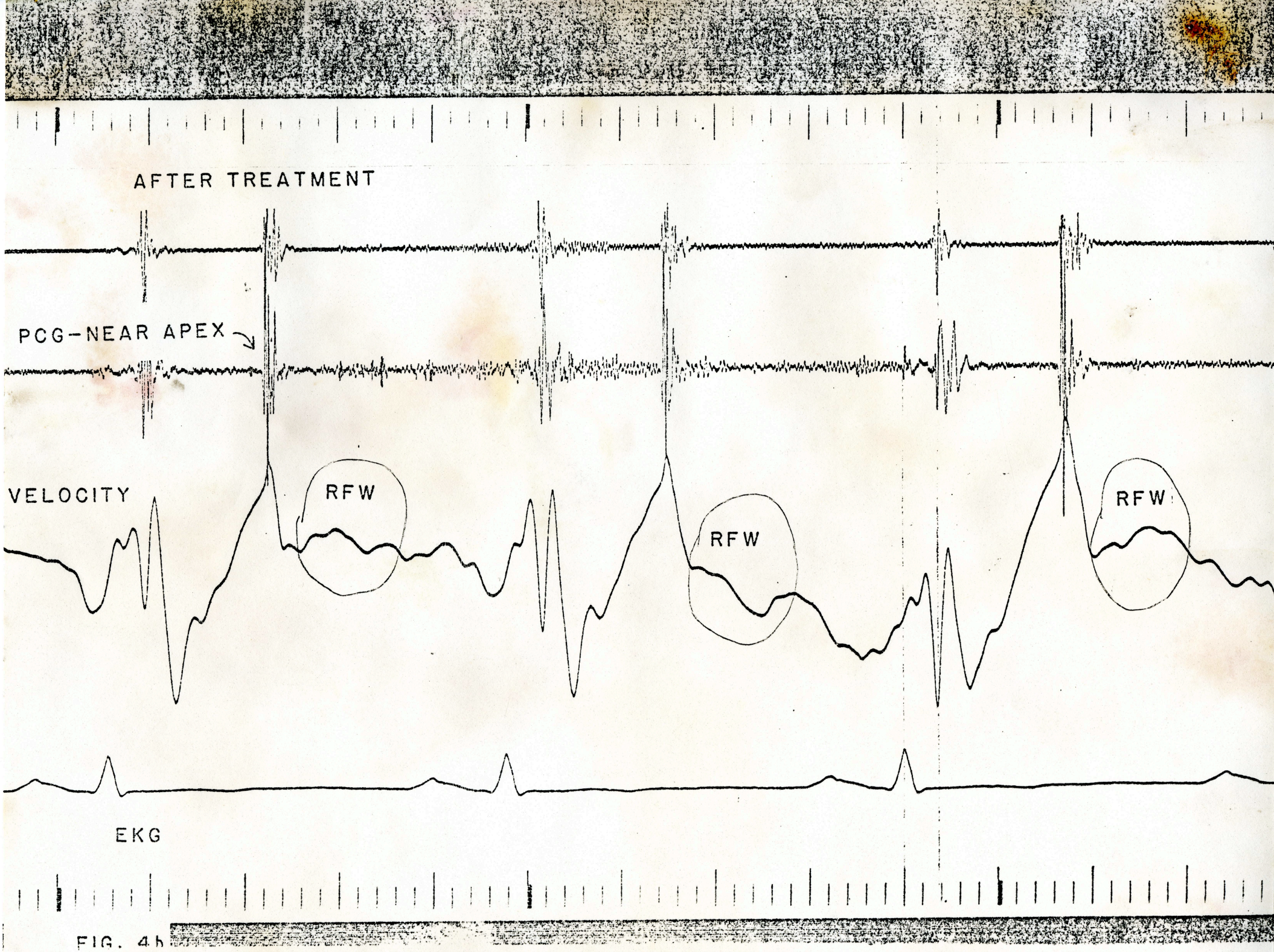


FIG. 4b

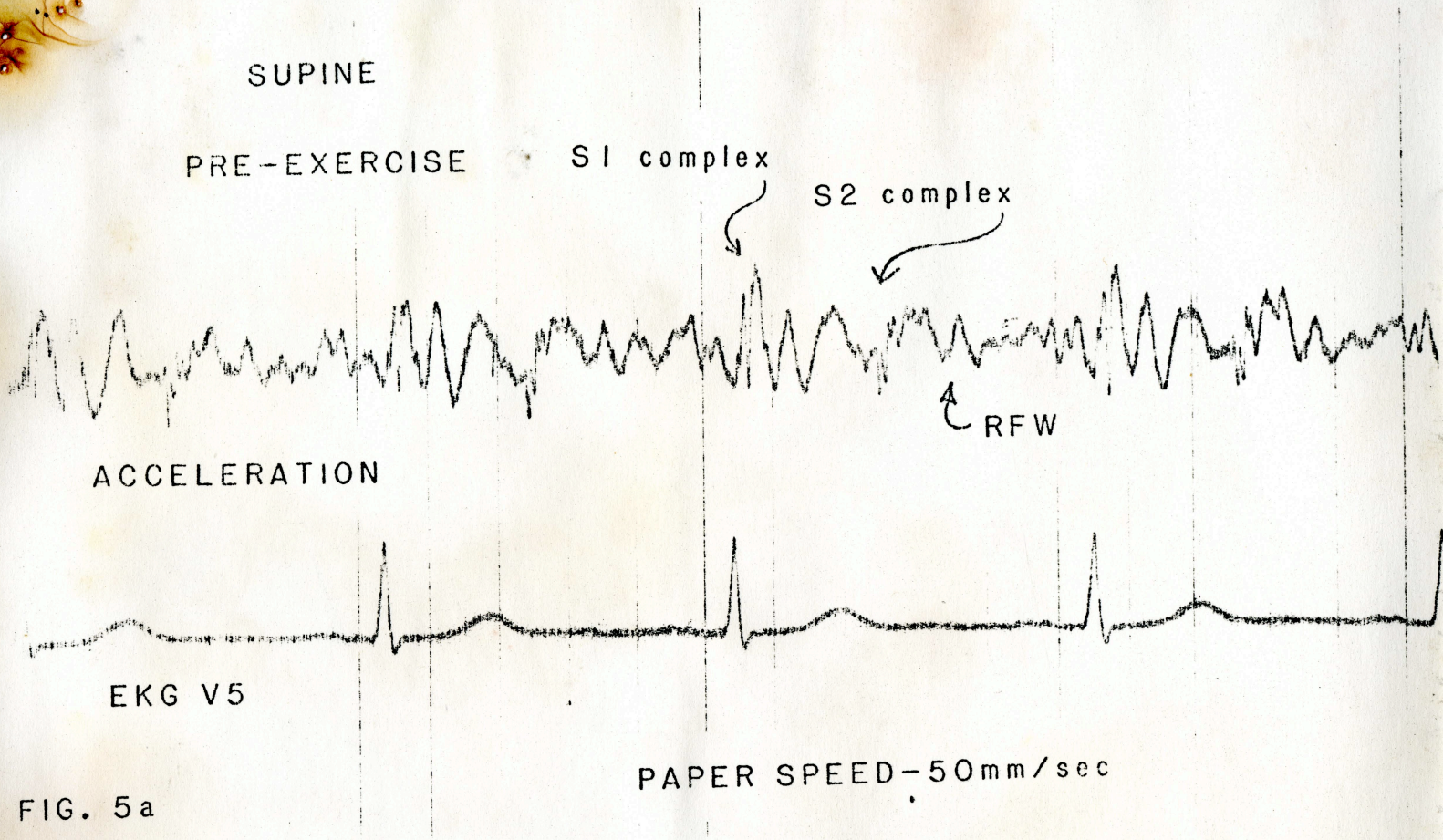

SUPINE
PRE-EXERCISE
S1 complex
S2 complex
RFW
ACCELERATION
EKG V5
PAPER SPEED-50mm/sec

FIG. 5a

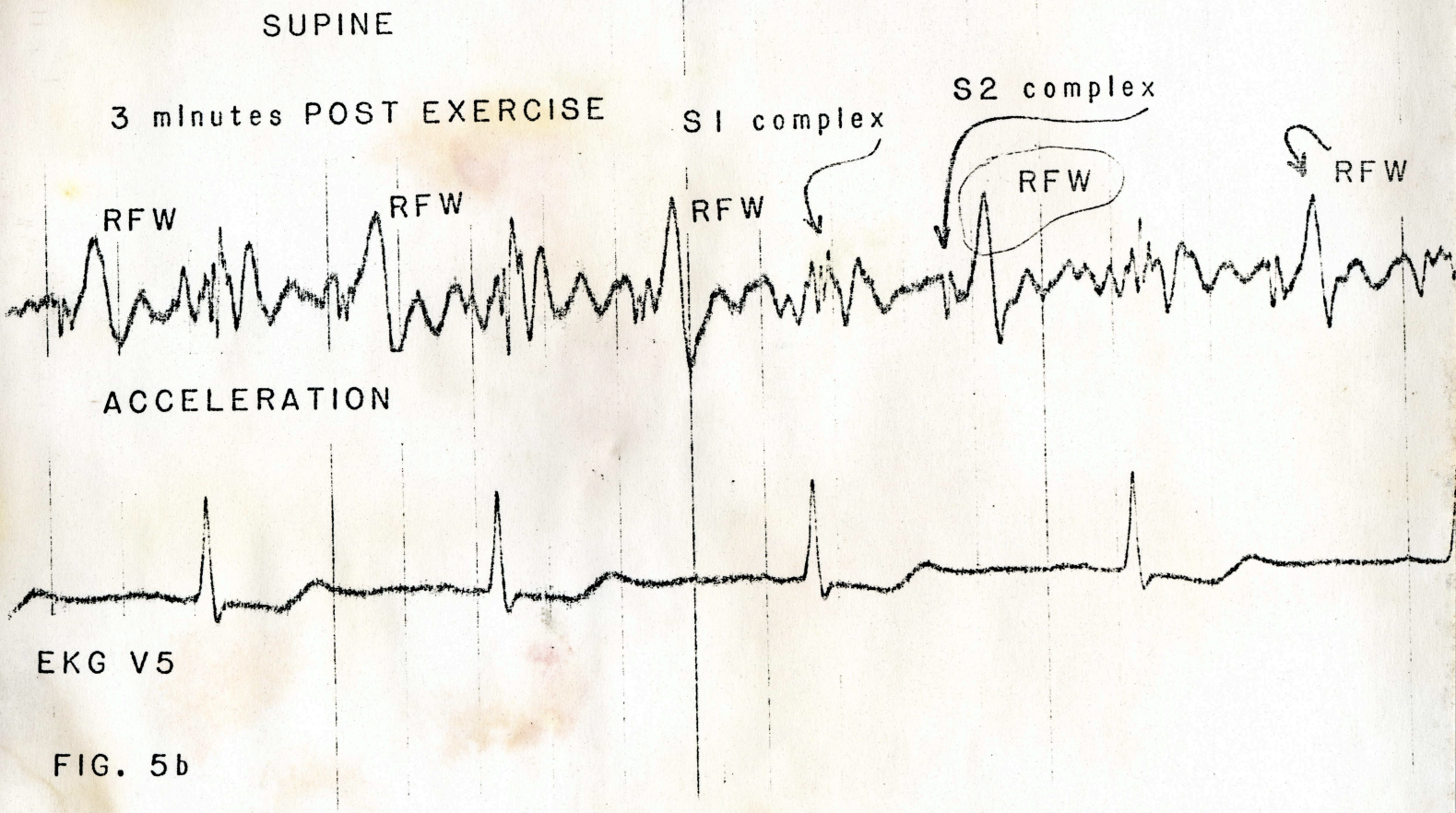

SUPINE
3 minutes POST EXERCISE
S1 complex
S2 complex
RFW
RFW
RFW
RFW
RFW
ACCELERATION
EKG V5

FIG. 5b

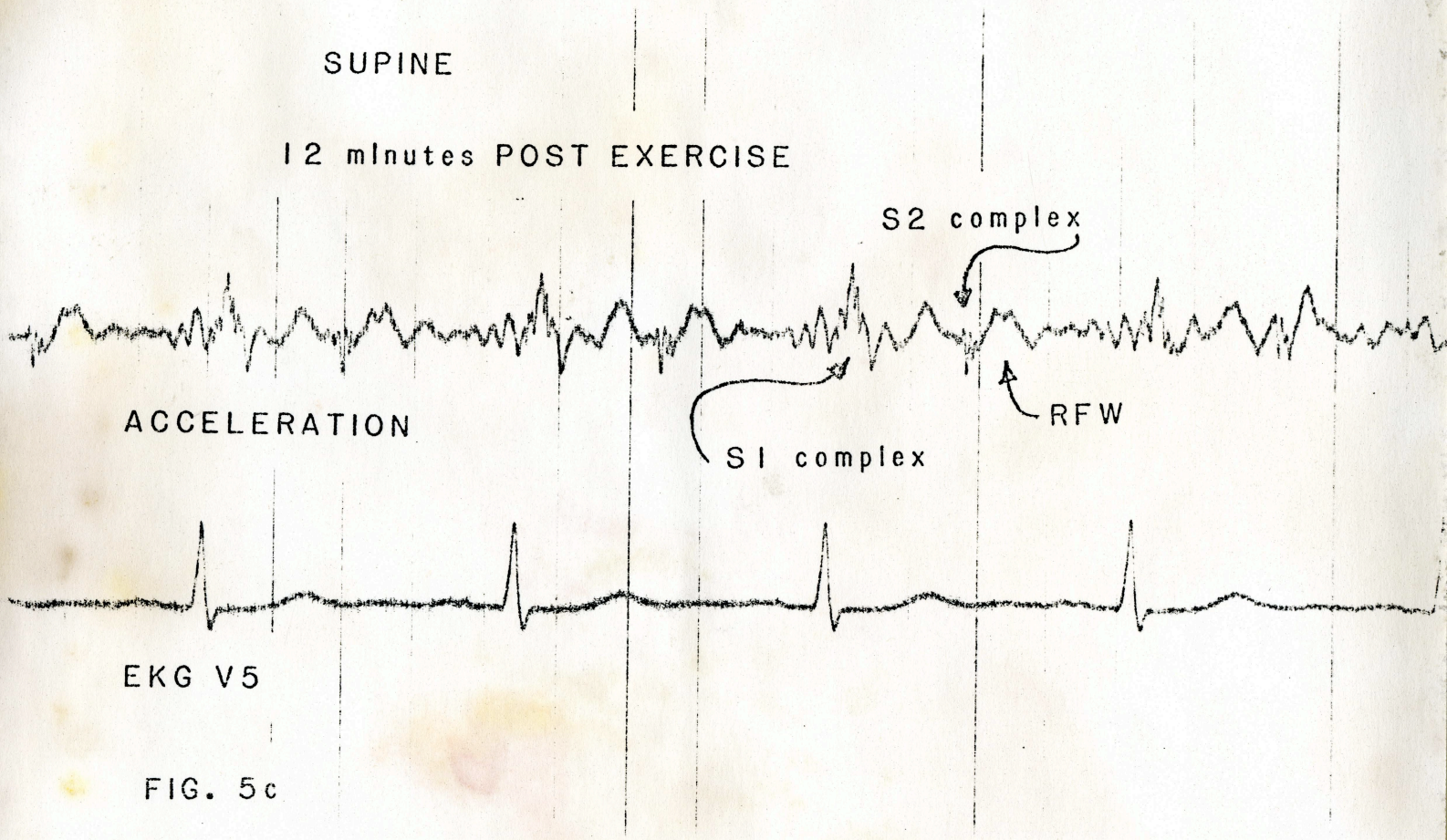

SUPINE
12 minutes POST EXERCISE
S2 complex
RFW
S1 complex
ACCELERATION
EKG V5

FIG. 5c

SUPINE PRE-EXERCISE

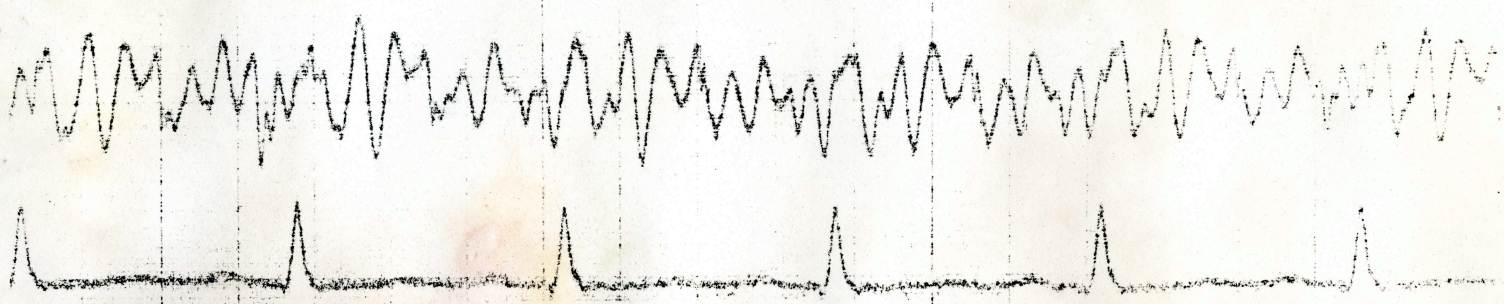


FIG. 6a

SUPINE 3minutes POST EXERCISE

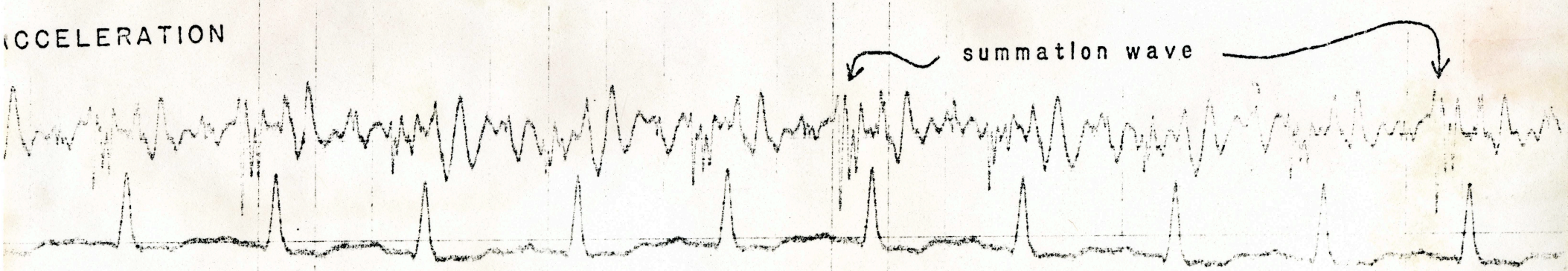


FIG. 6b

SUPINE PRE-EXERCISE

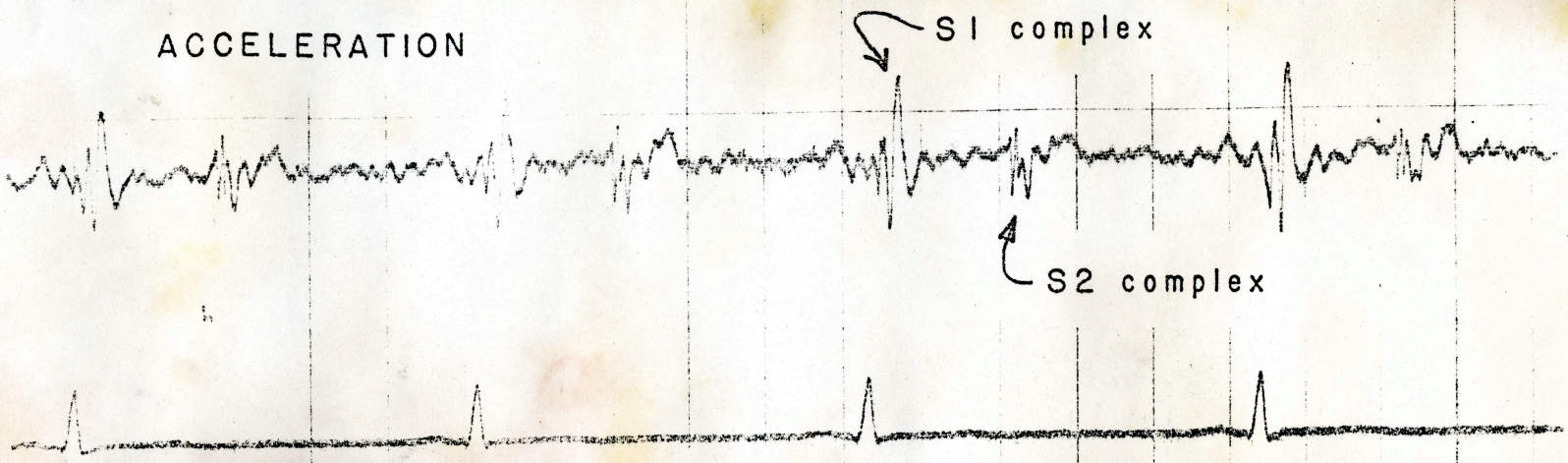


FIG. 7a

SUPINE 3 minutes POST EXERCISE

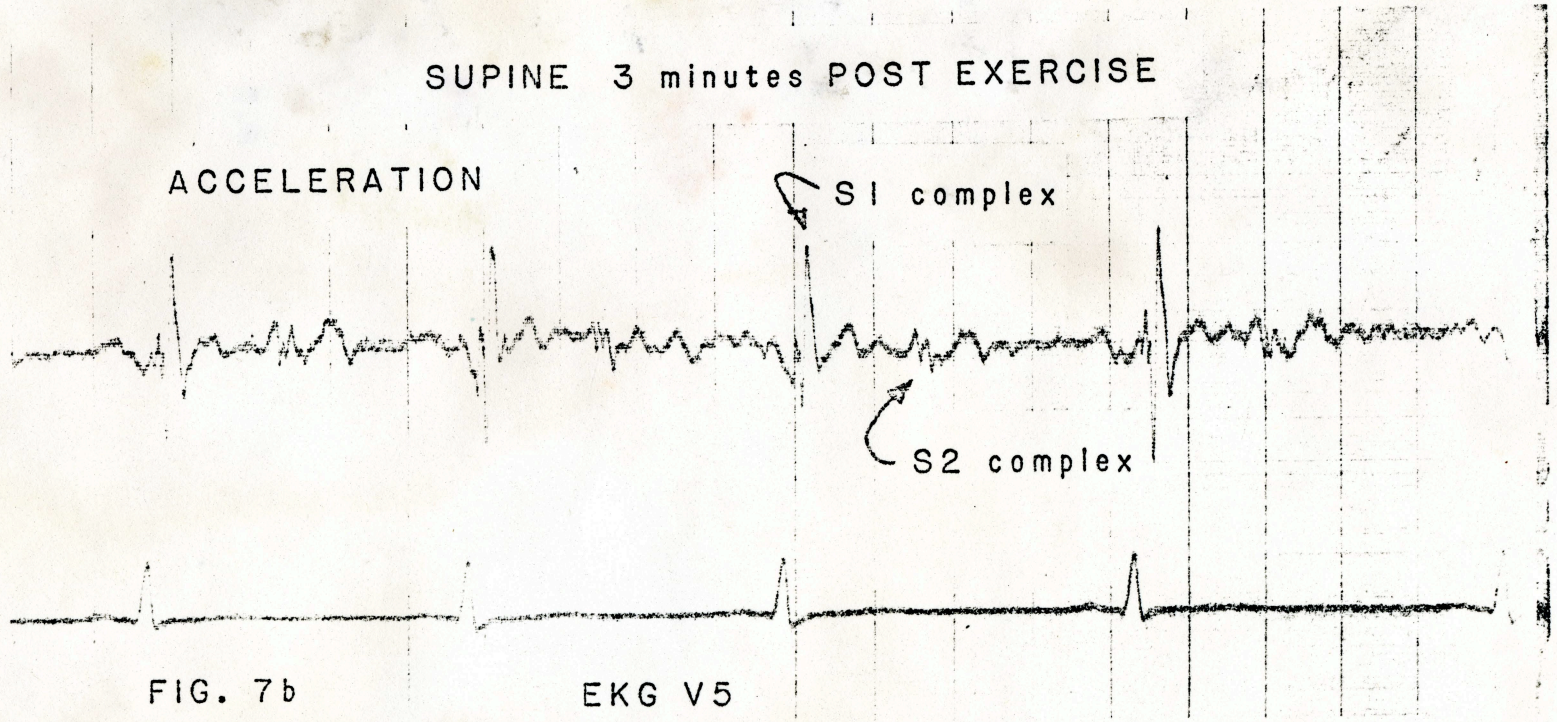


FIG. 7b