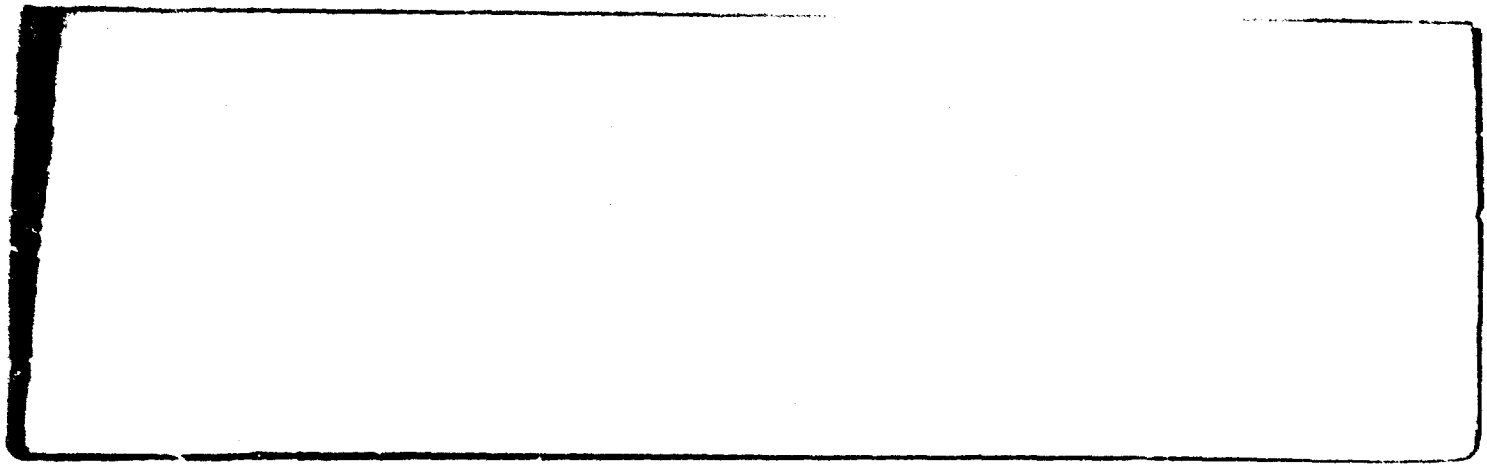

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Journal of Applied Physiology
Vol. 28, No. 3, March 1970. Printed in U.S.A.

A method for recording myocardial ECG in animals during intense vibration

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SASS, DONALD J. *A method for recording myocardial ECG in animals during intense vibration.* J. Appl. Physiol. 28(3): 361–364. 1970.— In studies of effects of whole-body vibration in cats we have been unable to record the electrocardiogram using conventional methods when the animal is vibrated with peak acceleration exceeding about ± 4 *g*. The present study shows, however, that the electrocardiogram can be recorded from myocardial electrodes during whole-body vibration with peak acceleration up to ± 15 *g*. Satisfactory electrodes consist of two no. 30 AWG enameled copper wires sutured into the myocardium, at one end of each wire with the two free ends brought out through the vascular system. Clinical quality electrocardiograms were not recorded using this method in anesthetized cats, but tracings were produced in which the base line and R waves are distinct throughout the period of vibration. This paper describes one method for implanting the electrodes and illustrates the results with reproductions of tracings from some of the experiments.

electrocardiogram; intense whole-body vibration; transvenous implanted myocardial electrodes; vibration artifacts in the electrocardiogram; vibration studies in water-immersed animals

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ELECTROCARDIOGRAMS are frequently difficult to record during experiments concerned with effects of acceleration in animals. Motion artifacts and shifting base lines in the tracings are most troublesome during rapid changes in acceleration, e.g., during impact on a sled decelerator or during the period when a centrifuge changes from one acceleration plateau to another. These instances are frequently brief, well within a cardiac cycle for many impacts, or within a few heart beats for the centrifuge example. In these cases transient interruptions in the tracing may be relatively unimportant. However, when the accelerative stress is whole-body vibration, both the direction and magnitude of the acceleration continually change, and, depending upon how intensely the animal is vibrated, a useful ECG may be impossible to record when conventional methods are used.

Investigations have been performed in this laboratory since 1952 to determine the effects of low-frequency, high-amplitude mechanical vibration in man and animals. The subjects of these experiments were vibrated on a table which oscillated up and down with sinusoidal motion. Frequency and peak acceleration were controllable over the range of 3–25 Hz and ±1 to ±15 *g*, respectively. Our experience with animals has shown that useful electrocardiograms cannot be obtained with conventional techniques when peak acceleration of the vibration table exceeds about ± 4 *g*. Vibration with this intensity is painful in man wearing a lap-belt restraint (2), but is below the stress known to produce substantial injury in well-restrained anesthetized animals. Experiments in anesthetized cats restrained by water immersion have demonstrated that peak accelerations reach approximately ±8 G_x or ±8 G_z before visible injury and mortality are produced (3, 5). Thus, while routine methods may produce satisfactory electrocardiograms during experiments with low-level vibration in man, routine methods cannot be relied upon in studies with intense vibration in animals.

We found it necessary to monitor the heart beat continuously during vibration in studies of the mechanisms of injury from vibration in cats and rhesus monkeys. Visible injury to the heart was rarely produced at any vibration intensity, although there was evidence that disturbances in cardiac function may have occurred during vibration. This evidence, not all of which has been published, is as follows. *1*) Pulmonic interstitial emphysema, mediastinal emphysema, air embolism, and congestive atelectasis were produced regularly in animals subjected to ±15 G_z vibration to a degree that would be expected to interfere with cardiac function (5). *2*) Asystole was a common finding in postvibration ECGs in animals that died during brief exposures with little or no visible injury to any part of the body. Air embolism was suspected to have caused death in these animals. *3*) In a few animals multiple small subendocardial hemorrhages were found in the left ventricle immediately below the annulus of the mitral valve and at the anchor points of the chordae tendineae. No other cardiac injury was visible in these few animals. The location of these hemorrhages indicates that the mitral valve had experienced excessive tensile forces. Possibly, valvular stress was produced by vibrational acceleration of the mass of blood in the outflow tract, impacting the mitral valve during systole. The aortic valve is open at this time and the mitral valve is subjected to the inertial effects of the large communicating mass of blood in the left ventricle and thoracic aorta.

On the basis of these experimental findings it appears that significant functional impairment of the heart can occur during vibration with little or no visible injury to the heart, and this impairment in function may be an important part of the mechanism of injury from vibration. A study of cardiovascular function during intense vibration will be necessary to examine this possibility. However, preliminary to further study we had to develop a method for recording the electrocardiogram in animals during vibration. Many techniques were tried, but only those recordings obtained from electrodes implanted in the myocardium with the connecting wires restrained by the vascular system were satisfactory.

This paper describes one method for implanting the electrodes in cats and illustrates the results with reproductions of tracings recorded during some of the experiments.

EXPERIMENTAL METHOD

Cats were used in these experiments. Two electrode wires were implanted in each of four animals so that one end of each wire was sutured into the myocardium of either the right or left ventricle. The free ends of the wires were passed together through the wall of the right ventricle near the apex and between the cusps of the tricuspid valve into the right atrium, then upward within the superior vena cava and from the right external jugular vein out of the body. Satisfactory electrodes were a pair of no. 30 American

wire gauge (AWG) enameled copper wires (0.23 mm), washed in a silicone solution before implantation.

The following method was used to implant the electrodes. The right ventricle was catheterized with a polyethylene catheter (1.7 mm od x 1.2 mm id) introduced via the right external jugular vein. A right parasternal thoracotomy was then performed and the pericardium opened. When the tip of the catheter was palpable through the right ventricle, a malleable brass obturator (1 mm) was passed down the catheter to pierce the wall of the right ventricle. Two no. 30 AWG enameled copper wires were threaded through two "eyes" in the exposed obturator. The catheter and obturator were withdrawn, pulling the wires within the vascular channel and out the jugular vein. The opposite ends were anchored in the myocardium by taking a stitch with each wire near the point where the two wires pass into the right ventricle. The pericardium was then approximated, a chest tube inserted for drainage and to remove the pneumothorax, and the thoracotomy incision closed. Later the chest tube was removed and the free ends of the wires were buried in the subcutaneous tissues of the neck until the day of the vibration experiment. Figure 1 illustrates the implanted electrode scheme.

A brief word concerning the vibration apparatus and instrumentation is appropriate. The cats in these experiments were anesthetized and immersed in water for restraint. The immersion apparatus consisted of a transparent acrylic cylinder filled with water and mounted vertically on a vibration table. The tank positioned the cat such that the direction of vibration was along the long axis of the body ($\pm G_z$). Details of the apparatus have been described elsewhere (4). The free ends of the electrodes near the animal's neck were soldered to the differential input terminals of a small battery-powered solid-state preamplifier mounted within the immersion tank near the animal. A third wire from the amplifier circuit ground was sewn into skin over the flank. The amplifier circuit was similar to the low-noise, interference-resistant circuit developed by Schuler et al. (6). Low-frequency response of the amplifier used in the present studies extended to approximately 0.05 Hz. The unit was encapsulated in a clear silicone resin and proved capable of withstanding up to ± 15 *g* vibration over the frequency range of 3–25 Hz while immersed in water without introducing recording artifacts. A self-contained 1.5-Hz square-wave generator served as a calibration source, and as a check for instrumentation artifacts during vibration. Noise due to cable flexing was negligible because of the very low output impedance of the amplifier and no special cables or shielding were required.

Anesthesia was maintained in each animal throughout both the surgical procedure and the vibration experiment. An initial dose of sodium pentobarbital was administered intraperitoneally (25 mg/kg body mass), and supplemental doses were given intravenously when indicated.

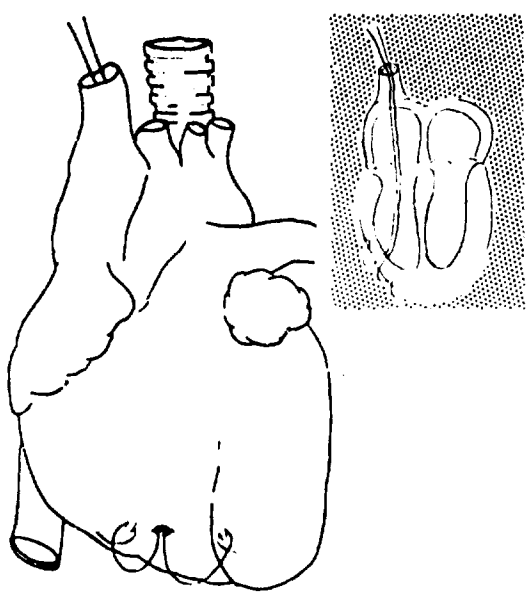

FIG. 1. Myocardial electrode scheme in animals. Fluid immersion restrains electrode wires during intense vibration.

RESULTS

Electrocardiograms recorded from myocardial electrodes in anesthetized water-immersed cats during vibration along the long body axis ($\pm G_z$) are reproduced in Figs. 2 and 3. The acceleration trace shown above the ECG in each figure was derived from an accelerometer mounted on the vibration table. High-frequency noise was removed from this trace by a low-pass filter having a cutoff frequency of approximately twice the vibration frequency. The ECG traces were not filtered. The recording reproduced in Fig. 2 was obtained during vibration at ± 10 G_z, 12 Hz, 7 days after the leads were implanted. The recording reproduced in Fig. 3 was obtained from another cat during vibration at ± 10 G_z, 17 Hz, 30 days after surgery. A large number of similar recordings were obtained from these and the other animals during repeated exposures at various peak accelerations between ± 1 G_z and ± 15 G_z. The exposures at ± 10 G_z and above were limited in duration to 30 sec to minimize injury, and in this manner the reproducibility of the method was established in repeated experiments with the four animals. Clinical quality ECG recordings with all components evident were not produced in these experiments, especially when peak acceleration exceeded about ± 8 G_z. However, all of the tracings produced in these experiments show distinct R waves throughout the vibration exposure with only minimal shift in base line. Interestingly, none of the recordings show an appreciable change in heart rate from the previbration value during whole-body vibration at any peak acceleration up to ± 15 G_z.

Small-amplitude oscillations around the base line are most noticeable in recordings obtained in experiments at ± 10 G_z and above. Expanded time scale recordings such as reproduced in Fig. 3 demonstrate synchronism with table acceleration, suggesting that vibrational motion of the electrodes produced the oscillatory component. P and T waves were obscured by this artifact although what appear to be injury currents could still be detected as illustrated in the recordings reproduced in Fig. 2. No attempts were made to remove this base-line oscillation by filter or subtraction methods.

DISCUSSION

The difficulties in recording an ECG during ± 15 *g* whole-body vibration become apparent when it is understood what ± 15 *g* represents in terms of body motion. An animal experiences peak accelerations of this magnitude when the animal is vibrated with harmonic motion at a 12 cycles/sec rate and with a peak-to-peak amplitude of 2 inches. Whole-body vibration with this intensity is severe and will produce substantial injury within minutes in well-restrained animals (3, 5).

The electrocardiogram recorded using conventional electrodes is usually obscured by artifacts during severe whole-body vibration. For example, recordings made with commercial silver-silver chloride skin electrodes are sufficiently free of artifacts to permit measurement of heart rate only up to ± 2 *g*. Small needle electrodes have been found similarly useful to only ± 4 *g*, and as a rule ± 4 *g* is about the upper limit of vibration stress to an animal during which an ECG, demonstrating at least the heart rate, can be recorded using conventional electrodes. Vibration at greater intensity increases the size of artifacts and the tracing is masked. The sources of these artifacts are not completely understood.

The largest source of vibration induced artifact is related to separation of the electrodes from the heart. Relative motion of the tissues between the heart and electrodes due to vibration rhythmically changes the electrical properties of the tissues. The propagation of ECG currents may be differentially altered as the tissues rapidly move back and forth that potentials related to this motion are introduced. Improved recordings were obtained when the electrodes were placed directly on the heart. Initially the electrodes were made from two lengths of Teflon-insulated wire, one end sutured into the myocardium and the free ends brought out through the chest wall. The recordings were satisfactory to ± 6 G_z. With greater peak accelerations, vibration of the portion of wire

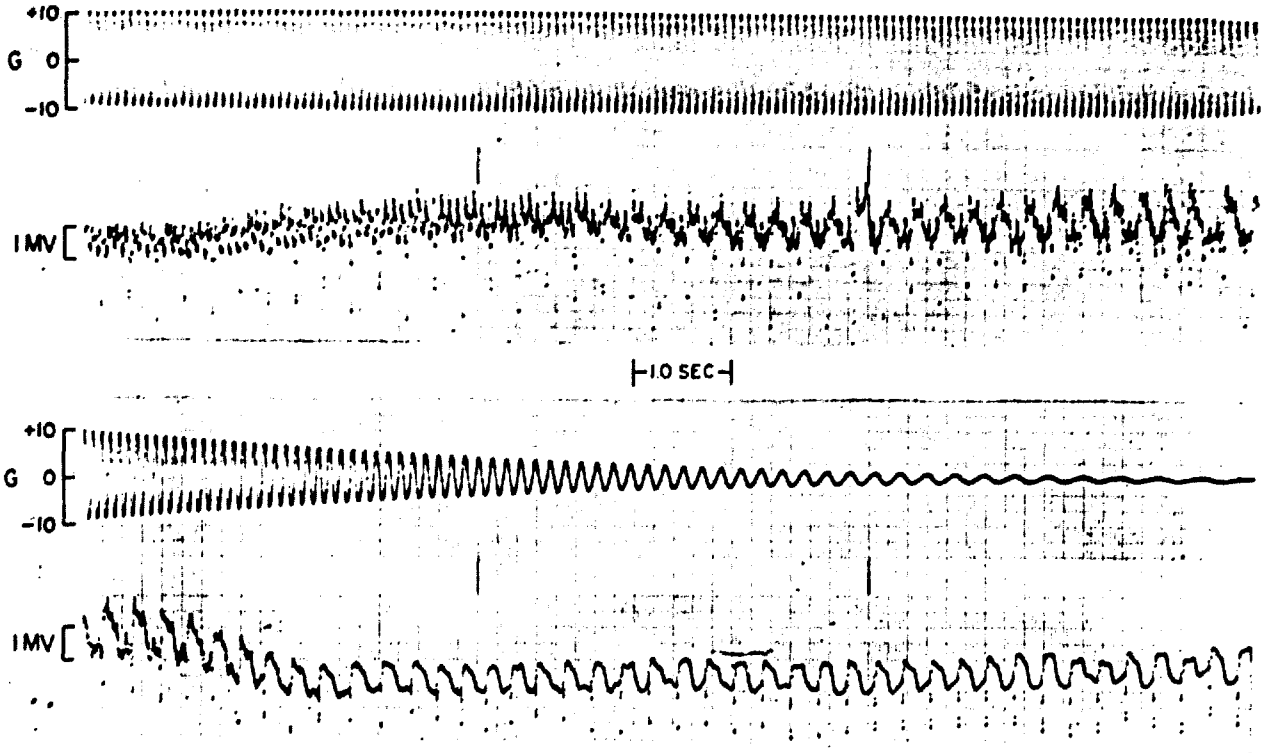

FIG. 2. Recording obtained from a water-immersed cat during vibration 7 days after surgery. Lower pair of traces continues from upper pair. Injury currents are shown developing during the last 5 or 6 sec of a 30-sec exposure at ± 10 G_z, 12 Hz. Sodium pentobarbital anesthesia, second cat.

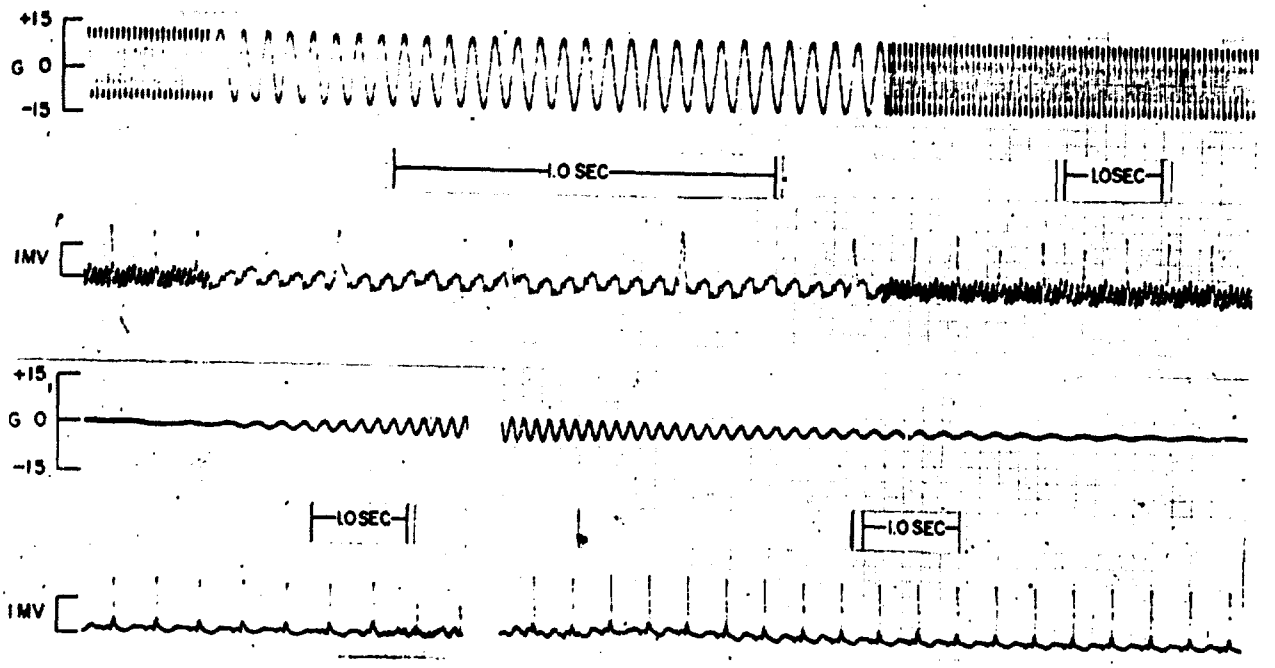

FIG. 3. Expanded time scale tracing was made during a 20-sec exposure at ± 10 G_z, 17 Hz, and demonstrates that small amplitude oscillations around base line are synchronous with table acceleration. Lower pair of recordings are from start and finish of this exposure. Sodium pentobarbital anesthesia, fourth cat, 30 days after surgery.

within the chest was detected in roentgenograms and presumably movement of the wire exerted a repetitive tug against the myocardium. Artifacts can be readily produced by light tugging on the wires at the time of surgery. The problem was then to find a better method of fixing the wires to the heart.

The best method was to implant the electrode wires within the heart and communicating vessels. Immersion of the wires within the vascular system reduces the net accelerative forces acting on the wires during vibration and thereby minimizes motion of the wires relative to the point of attachment in the heart muscle. This paper describes one method of implanting the wires and illustrates recordings obtained from cats during ± 10 G_z vibration. The recordings show results obtained with the technique. They are not intended to demonstrate effects of vibration on the cardiovascular system. This remains to be done.

The method has two drawbacks. First, there is a need for a thoracotomy when the wires are implanted as described. However, it may be possible to develop a percutaneous method of implanting wires attached to an endocardial screw. Carlsson and Milne (1) have implanted tantalum screws in the endocardium of dogs by a percutaneous route and a modification of their procedure may be applicable. Second, there is a possibility of cardiac injury from the electrodes and connecting wires during vibration, although cardiac injury was not found in the four animals studied here. Cardiac rate and rhythm varied little during the brief exposures to ± 15 G_z, and only minimal fibrosis was visible at the electrode site when the animals were autopsied. Thus, neither drawback is likely to prevent application of this technique in studies of effects of vibration in animals.

I am grateful to Drs. John C. Keesey, Seymour L. Friess, and Lay Fox for helpful advice in preparing this paper, and to Dr. Frank Letcher for assistance with the surgical procedure. The skilled technical assistance of Mr. William Lovejoy in performing the experiments and Mrs. Fannie Epstein in preparing the manuscript are gratefully acknowledged.

This study is designated Research Task No. MR 005.04-0037, from the Bureau of Medicine and Surgery, Navy Department, and was supported, in part, by NASA Contract R-10.

The opinions or assertions contained herein are the private ones of the author and are not to be construed as official or reflecting the views of the Navy Department or the naval service at large.

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Received for publication 7 April 1969.

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UNCLASSIFIED

Security Classification

DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author)

NAVAL MEDICAL RESEARCH INSTITUTE
NATIONAL NAVAL MEDICAL CENTER
BETHESDA, MARYLAND 20014

2a. REPORT SECURITY CLASSIFICATION

UNCLASSIFIED

2b. GROUP

3. REPORT TITLE

A METHOD FOR RECORDING MYOCARDIAL ECG IN ANIMALS DURING INTENSE VIBRATION

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)

Medical research interim report

5. AUTHOR(S) (First name, middle initial, last name)

Donald J. SASS

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
March 1970	4	6

8a. CONTRACT OR GRANT NO	9a. ORIGINATOR'S REPORT NUMBER(S)
b. PROJECT NO.	MR005.01.01-0105B, Report No. 16
c.	9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)
d.	

10. DISTRIBUTION STATEMENT

11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
Reprinted from the Journal of Applied Physiology 28: 361-364, 1970	Bureau of Medicine and Surgery (Navy) Washington, D. C.

13. ABSTRACT

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14 KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Electrocardiogram						
Intense whole-body vibration						
Transvenous implanted myocardial electrodes						
Vibration artifacts in the electrocardiogram						
Vibration studies in water-immersed animals						