Table 1. Tularemia in rodents after ingestion of infective carcasses. All fed animals became infected.

Species	Common name	No. used	No. fed	Day of death or sacrifice
Onychomys leucogaster	Northern grasshopper mouse	16	16	3–4
Citellus leucurus	Antelope ground squirrel	16	15	3–4
Eutamias minimus	Least chipmunk	6	5	3–7
Reithrodontomys megalotis	Western harvest mouse	8	5	3–6
Peromyscus maniculatus	Deer mouse	16	13	3–5
Peromyscus crinitus	Canyon mouse	15	10	3–4
Peromyscus truei	Pinyon mouse	16	15	3–6
**Dipodomys microps*	Chisel-toothed kangaroo rat	16	15	3–6
**Dipodomys ordii*	Ord kangaroo rat	16	11	3–7
**Neotoma lepida*	Desert wood rat	16	10	4–7
**Perognathus parvus*	Great Basin pocket mouse	16	4	5–6

* Required starvation periods of 48 hours before carcasses were introduced.

placed with each of the experimental animals, from which food and water had been withheld for 24 or 48 hours. At the end of 24 hours the carcasses or remains were removed, and food and water were restored to the test animals.

When the exposed animals died, or when they exhibited acute symptoms of infection, they were autopsied, or killed and autopsied, and liver and spleen homogenates were cultured on glucose cysteine blood agar. If *P. tularensis* was not isolated by culture, 0.2-ml aliquots of the liver and spleen homogenates which had been kept frozen at −28°C were inoculated intraperitoneally into healthy deer mice in a further attempt to isolate the organism. Those animals that survived exposure to infective carcasses were held for 14 days, after which they were killed and autopsied. The tissues were examined for *P. tularensis* as outlined above. In all cases definitive identification of the isolated organisms was made by specific slide agglutination tests.

Under the experimental conditions described, the first seven species listed in Table 1 showed little reluctance to feed on deer mouse carcasses. Most of the ground squirrels and chipmunks readily consumed the entire carcass, while the other rodents generally ate only the anterior part of each carcass. The pocket mice, wood rats, and kangaroo rats were reluctant to eat the dead flesh unless regular food supplies were withheld for 48 hours.

In all cases, every rodent that ingested infective flesh contracted tularemia; those that did not ingest infective flesh did not contract the disease.

Although the extent to which wild rodents supplement their natural diet with flesh has not yet been determined, these results indicate that carnivorism among desert rodents may occur and that it may be of importance not only in the maintenance of this disease in nature, but also in the propagation of tularemia epizootics. The first seven species listed in Table 1 must be considered to be potentially capable of contributing to the maintenance and dissemination of tularemia among desert rodent populations. Because of their reluctance to feed on dead flesh, the other four species must be considered of somewhat less importance in the transmisison of this disease.

E. Dean Vest
Nyven J. Marchette
Ecological Research,
University of Utah, Dugway

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5. Cultures of the Schu A strain of *Pasteurella tularensis* were obtained through the courtesy of Dr. H. T. Eigelbach, Fort Detrick, Frederick, Md.
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29 April 1957

Radio-frequency Resistive Impedance Pulsations over the Heart, Lungs, and Abdomen

The accurate timing of the major volumetric events in the atria, ventricles, lungs, and periphery of the human body may easily be accomplished without blood letting by surface study of the electrical resistive impedance changes around or over a given region of the human subject. Figure 1 illustrates the relative amplitude and direction of the pulsatile vertical impedance changes observed during held respiration over a 5 by 5 cm ventral surface area. These changes were studied in progressive areas in the left mid-clavicular line downward from the clavicle to the region below the umbilicus. Pertinent landmarks in the transition are identified together with the distance in centimeters below or above the sternal angle of our normal adult male subject Control electrocardiogram and deltoid-chest-deltoid tetrapolar electrical resistive impedance plethysmogram (*1–4*) serve to line up the corresponding tracings from each level. A decrease in electrical resistive impedance records upward in the trace and corresponds to an increase in volume pulsations. The paper speed is 50 mm/sec.

The study shows that with constant instrumental sensitivities the largest segmental impedance pulsations are found directly over the heart and apical regions 10 to 15 cm below the level of the sternal angle. The minimal impedance pulsations occur in the abdomen at the level of the umbilicus or lower.

An increasing volume pulse is present above the sternal angle and corresponds in its systolic upstroke with the control impedance systolic pulse of the upper torso body segment. These events begin about 0.11 to 0.12 second after the onset of the ventricular QRS of the electrocardiogram. These result from expansion of the great vessels, lungs, and chest segments with blood.

Just below the sternal angle level, we observe a transitional resistive impedance curve having a sigmoid-shaped downward deflection beginning 0.02 to 0.04 second after the onset of QRS in the electrocardiogram and 0.08 to 0.10 second before the control impedance pulse. This event is probably due to contraction of the atrial blood pool, which produces a decreased electrical conductance. A similar early change occurs at −10 and −15 cm below the sternal angle.

The initial ventricular emptying is best seen at −10 and −15 cm (reduced to 1/5 scale) in this case. Like the atrial tracing, it is also directed downward in the record. It is also sigmoid in shape and begins simultaneously with the positive pulse in the control tracing at 0.10 to 0.12 second after the onset of QRS deflection. The ventricular refilling at the −15-cm segment corresponds to the completion of the T wave of the electrocardiogram at 0.36 second after the onset of QRS deflection. A distinct notching or inflection occurs simultaneously with the onset of the U wave of the electrocardiogram in the record at 0.40 second. Since we did not take a simultaneous phonocardiogram, we cannot ascertain in this study whether it is signaled by dynamic events in the heart valves.

Figure 2 illustrates our observations, in the same subject, of resistive imped-

ance pulsations in a transverse line across the chest at the level of the heart. Controls and sensitivities are the same as those in Fig. 1. The traces in the upper line are identified in five 5-by-5-cm positions over the right and left chest and precordium. The dominant systolic positive pulsations over the right and left axillary lines are probably derived from the systolic arrival and distribution of blood to the lungs and underlying chest wall. The rising systolic slope begins simultaneously with the control upper chest pulse at 0.10 second after the onset of QRS deflections, and it extends beyond the end of the T wave of the electrocardiogram. Compared with the control pulsation, it appears to be an overly damped volume pulse. Both pulsations are produced by the movement of blood away from the heart before the diastolic event which follows the closure of the pulmonic valve.

The evidence by electrical resistive impedance for atrial contraction is observed in the records immediately to the right of the mid-sternum, 0 to 5 cm to the left of the mid-sternal line, and most marked in the 5- to 10-cm segment from the mid-sternal line over the heart region. As in Fig. 1, the early onset of a decreasing blood pool begins about 0.02 to 0.04 second after the onset of QRS deflection. On the basis of simultaneous pressure studies in the pulmonary artery, brachial artery, and right ventricle, we believe that this event precedes the ventricular contraction and that it is probably related to auricular contraction. Additional evidence for this has been obtained and suggested by the close correspondence of the roentgenphotofluorokymograms of the right and left auricular areas of another subject. The time interval between the impedance event over the atrial region and the subsequent positive pulsation in the upper chest is only 0.06 second. It appears, therefore, that some of the atrial contraction may have escaped detection except as a positive event since the P-R interval of the electrocardiogram in this case measures 0.12 to 0.14 second. In a case of auricular fibrillation, the atrial impedance deflection was absent, and the major precordial systolic impedance change corresponds to the systolic phase of increasing right intraventricular pressure. An isometric ventricular contraction effect in the impedance pulse is often distinct and proven in cases of atrial fibrillation with simultaneous intraventricular and direct pulmonary artery pressures.

In conclusion, electrical resistive impedance measurement of the chest and precordium reveals changes characteristic of atrial, ventricular, lung, and great vessel volume pulsations. The influence of stream line flow during the cardiac cycle may be a factor in the pulsatile change but cannot be dissociated from the major volume change. The dynamic cardiac and vascular events are easily recorded for long intervals without discomfort to the subject (*2*).

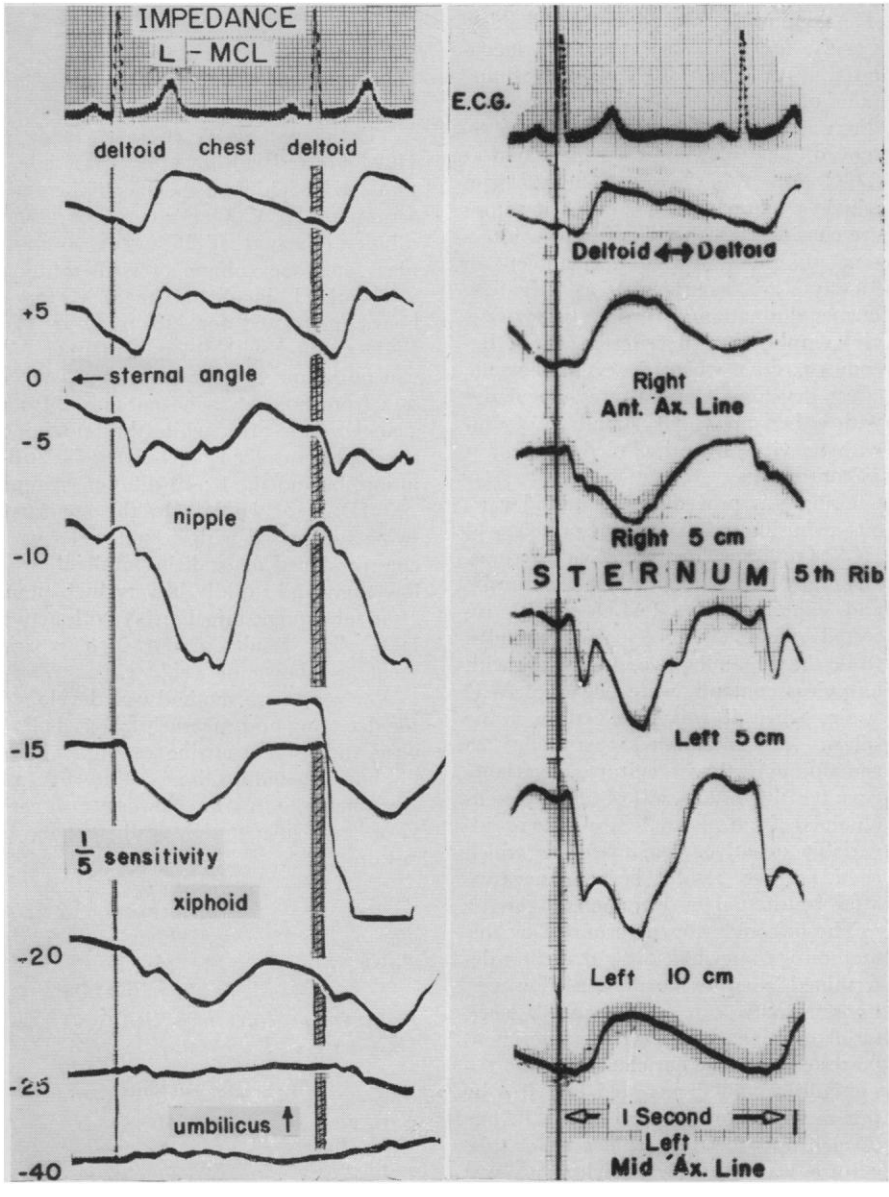

Fig. 1. (Left) Comparison of impedance pulsations between shoulders, and over 5-by-5-cm areas in left mid-clavicular line of chest and abdomen simultaneously with electrocardiogram (upper graph). The numerals indicate distance above or below level of sternal angle. The thin vertical line corresponds to the beginning of the QRS complex. Fig. 2. (Right) Comparison of impedance pulsations of 5-by-5-cm areas selected over the anterior chest from the right to left anterior axillary line at about the level of the fifth rib at the sternum. The shoulder-to-shoulder pulse and the electrocardiogram were recorded simultaneously with each study and then were lined up to correspond with the beginning of QRS complex in the graph.

JAN NYBOER
RODNEY E. WILLARD
CLETO DI GIOVANNI, JR.
Departments of Medicine and
Cardiovascular Physiology,
Harper Hospital, Detroit, Michigan

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