corresponding to an average diet of about 50 $\mu\mu$c Sr^{90}/g Ca, which is about four times the U.S. average for the same period. Thus, while the average in this series in which high values were sought is about 25 percent greater than the U.S. average, a few samples are quite high, which may reflect an appreciable quantity of caribou in the diet.

A few tribes that consume caribou almost exclusively are known in Alaska, but bone samples did not become available from this small group. Therefore, in order to get an adequate index of their exposure to strontium-90 and thus what the level would be in newly deposited bone, urine samples were obtained on six such individuals in February 1961 with the results shown in Table 5. Their tribe is located near Shungnak in northwest Alaska about 50 miles north of the Arctic Circle and 150 miles from the coast along the Kobuk River. Thus, in addition to caribou, they had access to fresh-water fish, but probably not marine life. It is noted that the strontium-90 concentration averaged 25 $\mu\mu$c (range 20 to 32) in the individuals of this predominately caribou-eating tribe.

Table 4. Strontium-90 content of bones from native Alaskan children and adults.

Age	$\mu\mu$c Sr^{90}/g Ca
Single bones, Nov. 1959–Dec. 1960	
4 mo	2.4 ± 0.3
7 yr	3.4 ± 0.3
16	2.4 ± 0.1
20	1.0 ± 0.2
20	0.8 ± 0.2
20	0.6 ± 0.1
24	0.4 ± 0.1
24	0.7 ± 0.2
25	0.2 ± 0.1
26	0.4 ± 0.2
26	< 0.3
26	< 0.1 ± 0.0
26	1.9 ± 0.2
29	0.4 ± 0.1
30	0.5 ± 0.1
30	< 0.2
32	0.2 ± 0.1
33	0.7 ± 0.1
35	1.4 ± 0.1
36	< 0.2
38	0.3 ± 0.1
38	0.3 ± 0.1
39	0.8 ± 0.1
40	< 0.4
42	< 0.4
44	0.4 ± 0.1
46	< 0.4
46	< 0.2
48	0.5 ± 0.1
53	0.4 ± 0.1
54	0.7 ± 0.2
54	0.3 ± 0.2
58	0.2 ± 0.1
60	0.8 ± 0.2
60	0.4 ± 0.1
60	0.4 ± 0.1
61	0.4 ± 0.1
62	0.5 ± 0.1
Composite samples, Jan.–Feb. 1961	
38	0.58 ± 0.06
38	0.38 ± 0.04

Table 5. Strontium-90 content in human urine specimens from Alaska, February 1961.

Sample	Dpm/liter	$\mu\mu$c Sr^{90}/g Ca
1	10.4	23.4
2	22.6	32.1
3	11.4	19.9
4	8.9	21.1
5	12.3	31.6
6	7.5	22.1
Av.	12.2	25.0

In earlier work comparing the diet and urine concentrations of strontium-90 in three diverse population groups, it was shown that the diet level is about twice that found in the urine (*8*). Therefore, these individuals had diet levels of about 50 $\mu\mu$c Sr^{90}/g Ca. The diet/bone discrimination against strontium-90 is four, and thus these individuals are forming bone at concentrations of 12 $\mu\mu$c Sr^{90}/g Ca. This is more than four times the U.S. average in 1961 and corresponds with the highest value obtained in bone among the 36 samples received and individually analyzed from native residents of Alaska.

The following conclusions are reached:

1) The foodstuffs consumed by the bulk of the population in Alaska probably have strontium-90 concentrations similar to those in the North Temperate Zone.

2) The concentration of strontium-90 in Alaskan caribou exceeds that of other foods by a factor of ten or more.

3) Urine concentration of strontium-90 in a caribou-eating tribe indicates that new bone is being laid down with about 12 $\mu\mu$c Sr^{90}/g Ca, which is more than four times the average U.S. concentration.

4) Three human bones of 35 that were analyzed also indicated new bone deposition of about this concentration.

References and Notes

1. Most of the sample procurement was accomplished by Dr. Christine Heller of the Arctic Health Research Institute of the Public Health Service at Anchorage, Alaska. This followed arrangements made in cooperation with Dr. A. B. Colyar and Dr. E. M. Scott in October 1959. Some caribou samples were obtained by Col. J. D. Fulton of the Ladd Air Force Base in Fairbanks. The wheat, cabbage, and potatoes were obtained by Dr. Arvo Kallio of the Department of Horticulture, University of Alaska. The California antlers were submitted by Dr. Harry Foreman of the Los Alamos Scientific Laboratory, Los Alamos, New Mexico. Professor J. L. Kulp critically read the manuscript and offered helpful suggestions. The Lamont analyses were conducted by Rieta Slakter Hirshman and Karin Bartelsen. This research was supported by the U.S. Atomic Energy Commission on contract AT(30-1)-1656. This report is Lamont Geological Observatory contribution No. 537.
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11 September 1961

Electroesophagogram of Individual Hookworm (Ancylostoma caninum)

Abstract. A device for recording regular rhythmic electrical phenomena that are synchronous with esophageal contractions in *Ancylostoma caninum* is described.

A method for studying the hookworm in vitro under conditions simulating to some extent those obtaining in nature (*1*) has been previously published. Essentially, the method is as follows: by means of a steel needle a live hookworm is threaded through a thin rubber membrane so that the head is on one side of the membrane and the tail on the other. The membrane with the hookworm is then mounted between two chambers. The "head chamber" is filled with a nutrient solution (usually dog's blood or plasma), and the "tail chamber" with a saline solution.

In this report, a description of a device for detecting electrical phenomena originating from the worm is presented, and a preliminary study of the nature of the recordings thus obtained is given.

After the live worm is placed in the apparatus, silver electrodes (diameter, 0.5 to 0.8 mm) are introduced into the head and tail chambers (Fig. 1, left) and connected, by means of an a-c coupled amplifier, to an oscilloscope or an ink recorder. The chambers are kept in a water bath at 37°C. The pulses observed on the oscilloscope are at first somewhat irregular (especially if the worms have been recently removed from the dog), but within 1 or 2 hours they become steady. If the worms are first kept at least 4 hours in a 50-percent mixture of dog's plasma

and Ringer's solution at 37°C, the pulses occur at once. The pulses are mostly runs of repeated, regular wave complexes, lasting as long as several minutes; after short rest intervals, the regular wave complexes begin again.

The general description of each complex which follows is based upon a total of 40 determinations. Each regular complex, termed "*A* wave" (Fig. 1, right), is composed of at least three major deflections. The first is a negative one, arbitrarily termed α (the head electrode being negative with respect to the tail electrode), with an amplitude of 2 to 3 mv and a duration of the order of 20 to 30 msec. The α deflection is followed by a sustained negative potential of 0.5 to 1.0 mv, which lasts from 60 to 250 msec (termed the β deflection). This β component ends sharply with a positive γ deflection, with duration of the order of 20 to 50 msec. An interval of 50 to 100 msec separates one complex from the next. The duration and amplitude of the various deflections and intervals, although variable from one hookworm to another, are quite constant for a single hookworm.

At times, particularly at the beginning of an experiment, another type of response occurs; it has been termed the "*B* wave" (Fig. 1, right). These *B* waves are characterized by oscillations that grow in amplitude and suddenly disappear.

The rhythmicity of the *A* waves suggests that they are related to the regular contractions of the worm's esophagus. This impression is confirmed by direct inspection of the worm under the microscope, with simultaneous observation of the "esophagogram" (EG) either visually or by listening to its auditory equivalent. The movements of the esophagus are clearly synchronous with the observed impulses on the oscilloscope screen. One complete cycle of the EG appears to correspond to the period between the opening and closing of the esophageal cavity. However, what phase of the motion (Fig. 2) corresponds to each deflection has not yet been determined with exactitude. The *B* waves are more frequent at the start of experiments and under any conditions which are presumably unfavorable to the worm, such as trauma and irritation. From visual observation of the worm under the microscope, it appears that these *B* waves are associated with uncoordinated inefficient movements of the esophageal musculature.

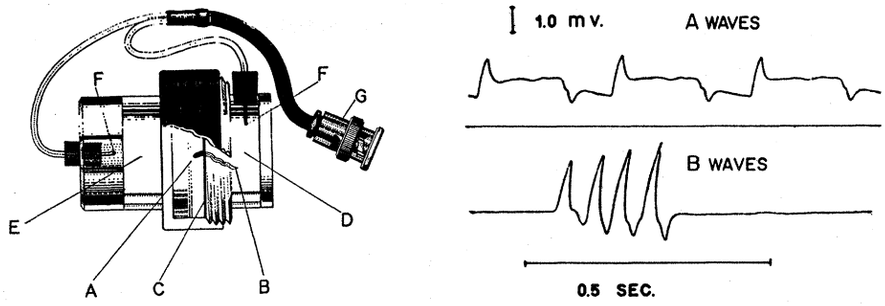

Fig. 1. (Left) Apparatus for the in vitro study of the hookworm. *A*, Cephalic end of the worm; *B*, caudal end of the worm; *C*, thin rubber membrane; *D*, lucite "tail chamber" filled with a saline solution; *E*, lucite "head chamber" filled with nutrient solution; *F*, silver electrode; *G*, connection with amplifier and oscilloscope. (Right) Electrical potentials produced by contraction of worm's esophagus. See text.

Further evidence that the pulses do originate from the esophageal region is furnished by exploration with a microelectrode. The live hookworm is embedded in 2-percent Ringer agar, care being taken to leave its head in a small blood-filled cavity, and the agar is connected with the ground electrode. A steel exploring microelectrode, with a tip of about 3 μ, is brought into contact with the cuticular surface of the worm. We were not able to penetrate the cuticle. When the exploring electrode is moved along the body of the worm, the potentials are maximal at the anterior end and diminish gradually as they approach the tail. If the worm is transected just behind the posterior end of the esophagus, the potentials disappear immediately on the tail side, while they persist for 20 to 30 minutes on the head and esophagus side. The evidence indicates that the recorded potentials originate in the anterior end of the worm and that they are synchronous with the movements of the esophagus. Whether these potentials are ultimately produced by the esophageal muscle itself or by a nervous structure such as the periesophageal nerve ring could not be determined. The shape of the curves obtained suggests a muscular, rather than a nervous, origin, but further research would be required to determine this point.

The esophagogram varies somewhat from worm to worm, but not essentially so; if proper conditions are maintained, the impulses last many hours. No difference in the EG is observed if dog's plasma instead of blood is used, up to

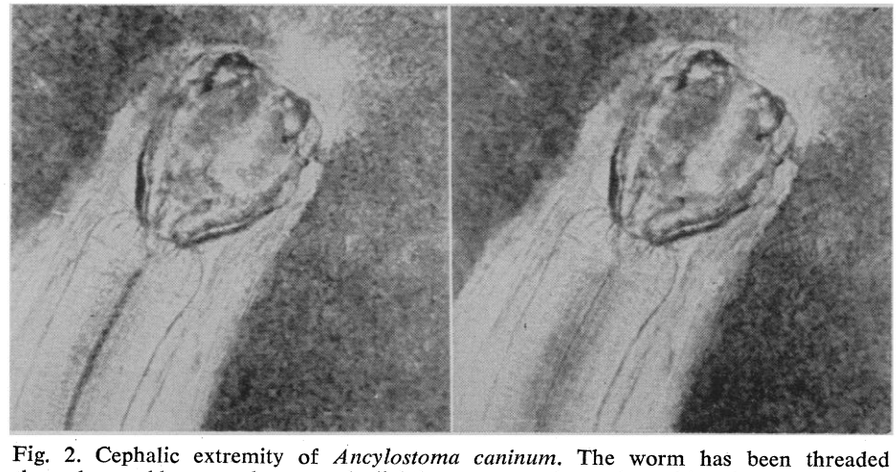

Fig. 2. Cephalic extremity of *Ancylostoma caninum*. The worm has been threaded through a rubber membrane and slightly compressed on the rubber by means of a coverslip, thus pushing aside blood, which is seen surrounding the worm (about × 150). (Left) The esophageal cavity is dilated, the anterior esophageal valve is open, and blood is rapidly flowing through the esophagus. (Right) The esophageal muscles have just contracted, the anterior esophageal valve has closed, and the blood has been pushed backward.

10 hours after the experiment has been started. This seems to indicate that the worm does not require anything in the erythrocytes to maintain normal esophageal contractions, at least for relatively short periods.

Potassium ions markedly modify the esophagogram. If plasma to which 50 meq of potassium per liter has been added is placed in the anterior chamber, the β deflection begins to rise within 15 to 20 minutes until it reaches a plateau, interrupted brusquely by the deflection. The EG returns to normal in 30 to 60 minutes if the head of the worm is placed again in a normal dog's plasma or blood.

With the system described above, it is possible to observe the worm for many hours and to make continuous graphic recordings. This same arrangement may also prove suitable for the study of drugs and of various conditions affecting the esophageal musculature and its metabolism. This method may presumably be applied to other organisms that are similar to hookworm (*2*).

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2. We thank Dr. Gunnar Svaetichin and Dr. Michael Verzeano for their help with the microelectrode experiments.

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Oxidation of Carbon-14 Labeled Galactose by Subjects with Congenital Galactosemia

Abstract. Galactose-1-C^{14} oxidation was studied in eight individuals with congenital galactosemia. Although two of these subjects fulfilled the usual diagnostic criteria for this disorder, they oxidized the intravenously administered sugar to $C^{14}O_2$ in a nearly normal fashion.

Congenital galactosemia is an inherited metabolic disease characterized by mental retardation, cataracts, hepatosplenomegaly, and severe malnutrition in infants fed diets containing galactose. This clinical syndrome has been associated with elevated galactose levels in the blood, increased erythrocyte galactose-1-phosphate, and galactosuria. Kalckar and his associates (*1*) have established that the metabolic defect is an absence of galactose-1-phosphate uridyltransferase, and the assay for this enzyme in hemolysates has become a widely used diagnostic procedure. Hemolysates from galactosemic individuals are unable to oxidize galactose-1-C^{14} to $C^{14}O_2$, an observation also proposed as a basis for a diagnostic test (*2*).

Several investigators have attempted to assess the ability of galactosemic individuals to metabolize galactose by determining the fraction of ingested galactose excreted in the urine (*3, 4*). Whereas normal individuals excrete none of the ingested galactose, galactosemics excrete 15 to 60 percent of the sugar in the urine over a 24-hour period, and it was assumed the remainder was either stored in the body or metabolized. In order to obtain a more accurate assessment of galactose metabolism by galactosemic subjects, we have assayed the oxidation of intravenously administered C^{14}-labeled galactose to $C^{14}O_2$ in expired air for a 5-hour period after injection. Our results follow.

Experiments were performed with eight galactosemic subjects ranging in age from 6 to 30 years (Table 1). All but two of the subjects had a typical history of the galactosemic syndrome which was noted shortly after birth and which subsided upon institution of a galactose-free diet. The other two, E.W. and P.R., were discovered in childhood because of the clinical findings of mental retardation and cataracts. The manifestations of the disease in infancy have been reported for subjects T.B. (*5*), J.D. (*3*), and L.J. (*6*). All of the subjects were mentally retarded and had cataracts when our study was made. All of them lacked detectable transferase in their red cells, and the corresponding hemolysates were unable to oxidize C^{14}-galactose to $C^{14}O_2$.

Table 1. The oxidation of C^{14}-labeled galactose by galactosemic subjects. After an overnight fast galactose-1-C^{14} (4.72 μc/mg) obtained from the National Bureau of Standards was given intravenously either in normal saline (1 μc/ml) or mixed with a 20-percent solution of unlabeled galactose. Expired air was collected, and $C^{14}O_2$ was analyzed as previously described (*9*). Abbreviations: M, male; F, female; W, white; N, Negro. The studies of galactose metabolism in normal subjects have been reported previously (*10*).

Subjects	Age	Sex	Race	Galactose-1-C^{14}		Administered C^{14} in expired air after 5 hr (%)
				Grams	μc	
Galactosemic						
J.O'D.	6	M	W	0.00042	2	1
B.A.	7	M	W	1.0	2	8
E.W.	9	M	W	0.00042	2	0
P.R.	9	F	W	0.00042	2	3
	11			1.0	1	7
L.J.	11	M	W	0.00042	2	1
L.Br.	11	M	N	0.00042	2	35
				1.0	3	35
J.D.	17	M	W	0.00053	2.5	3
				1.0	2.5	5
T.B.	30	M	N	0.00053	2.5	19
				1.0	2.5	26
				2.0	2.5	28
				10.0	2.5	19
Normal (No.)						
4	18–21	M	W	0.00106	5	30–35
1	18	M	W	10.0	5	29
2	18–21	M	W	20.0	5	25–27

Table 1 shows the percentage of the administered C^{14} in expired air after 5 hours. The amount metabolized to $C^{14}O_2$ varied from 0 to 8 percent in six of the patients when either 1 mg or 1 g of galactose was given. In two of the subjects, L.Br. and T.B., normal or near-normal conversion of these quantities of galactose to $C^{14}O_2$ took place during the period of study. It should be noted (Table 1) that normal subjects become saturated with respect to their galactose oxidizing capacity when about 20 g are given, whereas saturation occurred in subject T.B. when 10 g were given.

Subject L.Br., like the other 11-year-olds, was prepubertal, while T.B. was postpubertal. J.D. was also postpubertal but metabolized galactose as poorly as the younger children. Thus it appears that the ability of certain galactosemic subjects to metabolize galactose at a near normal level is not specifically related to onset of puberty or to chronological age by itself. It is interesting to note that both L.Br. and T.B. are Negroes.