Discussion. Hunt and co-workers[5] have studied the inheritance of susceptibility to dental caries in the white rat, and have obtained in a series of 12 generations, a marked increase in susceptibility by inbreeding susceptible strains. They could decrease the susceptibility of the resistant strains by similar means. McClure and Arnold[6] in a study on the influence of fluorides and iodoacetate on the incidence of caries in the white rat have pointed out a difference in caries susceptibility of offspring from different parent stock. Arnold[7] has also observed that a difference in caries susceptibility existed in hamsters, when offspring from different parents were compared. These observations indicate the importance of heredity as a factor in the production of experimental dental caries and also point out the necessity of recognizing this factor in designing properly controlled experiments, especially where animals are of heterogeneous stock.

Summary. A difference in susceptibility of different strains of cotton rats to dental caries has been observed and shown to be statistically significant. Preliminary results from second generation offspring indicate that their susceptibility is similar to that of the parent stock.

[5] Hunt, H. R., Hoppert, C. A., and Erwin, W. G., *J. Dental Res.*, 1944, **23**, 385.

[6] McClure, F. J., and Arnold, F. A., Jr., *J. Dental Res.*, 1941, **20**, 97.

[7] Arnold, F. A., Jr., *U. S. Pub. Health Rep.*, 1942, **57**, 1599.

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Effect of Cardiac Cycle Length upon Magnitude of Ventricular Gradient.

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The ventricular gradient of Wilson[1] may be defined as the net electrical effect of differences in time course of repolarization of different parts of the ventricular muscle as this effect is projected onto the frontal plane of the body. In the electrocardiogram it is measured by the mean manifest net area of the QRS-T group of deflections. In previous papers[2,3] it has been pointed out that the magnitude of the gradient varies inversely as the heart rate or directly as the average cardiac cycle length. The present report seeks to explain this relationship.

Three main procedures were used. In one, the string galvanometer recorded the action current derived from the apex and base of the excised turtle ventricle, in air. Apical negativity yielded an upward deflection. Records were obtained when the ventricle was driven at various rates, as shown by the cycle lengths in Table I. In a second set of experiments the apex was cooled, though, as the irregularity of the results shows, no attempt was made to keep the temperature constant. Table I also shows the Q-T interval for each cycle length, and the net area of QRS-T in units of about 30 microvolt-seconds. The method of measuring the areas has been published elsewhere.[4] Since the initial deflections were small, the larger part of the net QRS-T area is within the T wave, except when the cycles were short.

It may be observed that the net areas are

[1] Wilson, F. N., Macleod, A. G., and Barker, P. S., *Tr. A. Am. Physicians*, 1931, **46**, 29.

[2] Ashman, R., Ferguson, F. P., Gremillion, A. I., and Byer, E., *Am. J. Physiol.*, 1945, **143**, 453.

[3] Ashman, R., and Byer, E., *Am. Heart J.*, 1943, **25**, 36.

[4] Ashman, R., Byer, E., and Bayley, R. H., *Am. Heart J.*, 1943, **25**, 16.

TABLE I.
Relationship between Cycle Length and Net QRS-T Areas.

Cycle lengths (sec)	Apex not cooled		Apex cooled	
	Net QRS-T areas	Q-T intervals (sec)	Net QRS-T areas	Q-T intervals (sec)
20	—220	1.70	+268	1.92
10	—200	1.66	+380	2.12
7	—136	1.60	+280	2.10
5	— 76	1.52	+198	2.02
2	— 20	1.30	— 4	1.42
1.2	— 8	0.84	— 52	0.88

TABLE II.
Relationship between Cycle Length and Duration of Monophasic Curve.

Cycle lengths (sec)	Ventricle at room temp. (24°C) Duration of monophasic curve (sec)	Ventricle 11.5°C below room temp. Duration of monophasic curve (sec)	Ventricle 20°-18°C below room temp. Duration of monophasic curve (sec)
20	1.40	2.70	4.40
10	1.40	2.65	4.00
7.5	1.40	2.47	2.96
5	1.40	2.27	2.35
4.2	untested	untested	1.87
4	"	"	1.67
3	"	1.65	refractory
2	"	1.10	"
1.4	1.05	refractory	"
1.1	0.77	"	"

negative, that is, below the base line of the electrogram, when the apex was at the room temperature of 24°C. This finding indicates that in this heart the repolarization of the apex was more rapid than that of the base. The significant finding is the very small area when the cycles were shortest, and the progressive decrease in area as the minute heart rate increased. This was the consistent result in 5 experiments.

Table I also shows that the Q-T interval decreased progressively as the cycle length was shortened. In major part this was due to the well known effect of cycle length change upon Q-T duration; in part, in this case, it was a cumulative effect of activity.[5]

When the apex was cooled, the time required for repolarization in that region was longer than it was at the base. This is demonstrated by the increase which occurred in the Q-T interval. The cooling was not quite so great at the 20-second cycle as it was throughout the rest of the experiment. At the longer cycle lengths the net QRS-T areas were positive, but at the very short cycles, the net areas became negative, in spite of the fact, shown by a control curve at the end of the experiment, that the apex was still cool. It is noteworthy that in this experiment, when the cycles were very short, the base became repolarized more slowly than the apex. This means that the inherent tendency of the base to recover more slowly than the apex in this heart again manifests itself when the increased rate greatly shortened the recovery time at the cool apex. This latter phenomenon is often seen in the human heart when, as a result of a locally deficient blood supply, a part of the ventricular muscle becomes repolarized more slowly than the rest. Then, in association with a premature beat, a normal form of the T wave is often restored. The ischemic muscle evidently behaves like cool muscle in this respect.

The third procedure reveals the cause of these changes. The ventricle was immersed in Ringer's solution at room temperature

[5] Blair, H. A., Wedd, A. M., and Young, A. C., *Am. J. Physiol.*, 1941, **132**, 157.

(24°C) and again in a cooler solution. By means of a suction electrode[6] injury was produced, and the monophasic curves were recorded when the galvanometer was connected to this electrode and to another electrode several centimeters distant in the solution. Table II shows that when the cycles were long the monophasic curve of the cool muscle, measured from the top of its ascending limb to its end, had nearly double the duration of the curve derived from the uncooled ventricle. When the cycles were short, however, the monophasic curves were practically equal in duration at both temperatures. This experiment was typical of four. It explains the effect of rate increase in the uninjured heart, namely, that the decrease in the net QRS-T area is due to a change in the relative times required for repolarization, in the sense that the time becomes more nearly the same in all parts of the muscle.

This evidence indicates that in the human heart, beating slowly, the large gradient which is usually present (and which accounts for the normal T-wave directions) is due to the slower repolarization of some parts of the muscle than of other parts. When the rate is very rapid, the time course of repolarization is everywhere nearly the same, and the gradient is, therefore, reduced in magnitude.

6 Wiggers, H. C., *Am. J. Physiol.*, 1938, **123**, 215.

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Form Variation in Group C Salmonella Strains.*†

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The occurrence of strains of the Newport group (VI, VIII) which lacked antigen VI, the O component possessed in common with the Choleræ-suis (VI, VII) and Carrau-Onderstepoort (VI, XIV . . .) groups, occasioned a closer study of the O antigens of group C Salmonella cultures. Kauffmann[1] demonstrated that antigen VI is divisible into at least 2 fractions which he designated VI_1 and VI_2. The majority of cultures of *S. choleræ-suis* contained only VI_1 antigen, although a few contained only VI_2. The remaining strains of the VI, VII group which were studied contained both VI_1 and VI_2. On the contrary, cultures of the Newport group (VI, VIII) contained only VI_1. Hormaeche, Peluffo, and Ricaud de Pereyra[2] found that antigen VI was more complex than had been generally realized and that it was composed of at least 3 principal fractions and additional minor antigens. Further, they remarked that they had found its distribution rather irregular. Also, in routine typing it has been observed that cross agglutination between the VI, VII and VI, VIII groups was quite variable. The lack of antigen VI in certain cultures and the irregularities noted in its behavior suggested that the antigen, or a portion thereof, was subject to the form variation or O variation which Kauffmann[3,4] found in antigen I and in a fraction of antigen XII. Accordingly, cultures of *S. newport* and *S.*

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1 Kauffmann, F., *Z. f. Hyg.*, 1937, **120**, 177.

2 Hormaeche, E., Peluffo, C. A., and Ricaud de Pereyra, V., *J. Bact.*, 1944, **47**, 323.

3 Kauffmann, F., *Acta path. et microbiol. Scand.*, 1940, **17**, 135.

4 Kauffmann, F., *J. Bact.*, 1940, **41**, 127.