brief periods of time when the electrode tip was presumably within a cell body. These were associated with injury discharge and eventual death of the cell and have not persisted long enough for study. It was found that unitary spikes of this type could be recorded only with the smaller microelectrodes, preferably of 1 μ or less. Multiple units of different amplitude were obtained with larger electrodes.

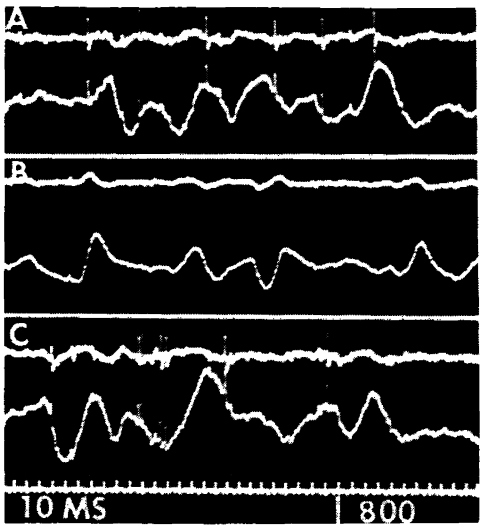

FIG. 2. Unitary spikes and slow waves recorded with a microelectrode connected to 2 amplifier oscilloscope systems for simultaneous recording with a short and a long time constant. The slow waves do not appear, therefore, in the upper tracing in each sample. Records taken at a depth of 1800 μ. *A*, a burst of slow waves and spikes in the animal without general anesthesia; *B*, taken from the same point 8 min following the intravenous administration of 50 mg Nembutal; *C*, taken 1 hr later, when the animal had recovered somewhat from the anesthesia.

Slight anoxia or Nembutal anesthesia would readily abolish the spike discharges, leaving the bursts of slow waves relatively unaffected (Fig. 2), the spikes returning with oxygen administration or by the administration of picrotoxin to counteract the effect of Nembutal. When the spikes disappeared with hypoxia or anesthesia, careful search was made through the cortex in several places in the attempt to find other units that might account for the slow waves, but only an odd sporadic unit was found. It appeared that unit discharge in the cortex had been readily suppressed with slight hypoxia or anesthesia without having much effect upon the rhythmic slow waves.

Higher voltage slow waves caused by applying strychnine or eserine and acetylcholine to the cortex were more consistently related to bursts of rapid spikes during the rising phase or negative peak of the slow wave, although even then they sometimes continued much longer at a frequency unrelated to the slow waves.

These results would seem to prove that the brain waves commonly used by the electroencephalographer as an index of cortical activity may, under certain circumstances, bear little relationship to the active discharge of individual cortical cells, at least of the type from which records can be obtained with microelectrodes. Certainly there is no support for the supposition that the slow waves result from envelopes of spike discharge.

There is also no support for the notion that the "spontaneous" rhythms of the brain are due to nerve impulses circulating in reverberating closed chains of self-re-exciting units, since the slow waves continue when unit discharge (at least as recorded with microelectrodes) is suppressed by hypoxia or anesthesia. In the depths of the cortex one may find extremely active discharge of neurons, or they may be remarkably silent, with very little reflection of these vastly different conditions upon the brain wave record obtained from the cortical surface.

Brain waves do seem to be phenomena of a different order. The most plausible hypothesis is that they represent synchronized oscillations in membrane potentials, possibly involving small interneurons and dendrites in the cortical matrix, oscillations which would have a definite effect upon neuronal excitability, but not dependent upon neuronal discharge. They would thus be classed with the so-called "synaptic" or soma potentials of the spinal cord, which are also obtained from anterior horn cells after the discharge of these cells has been suppressed by barbiturate anesthesia (*5*). Their oscillatory character is of particular interest, since it does not seem to depend upon repetitive discharge of the larger cortical cells of the type whose activity was recorded with the microelectrodes used in these studies. They may still be related to impulses circulating in the fine dendritic network of the cortex, whose unitary activity may not appear with the techniques here employed.

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Manuscript received June 2, 1952.

Influence of Amino Acids upon Incidence of Tumors in *Tu*^{50j} Stock of *D. melanogaster*

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In a recent report Mittler (*1*) has shown that nutrition of *Drosophila melanogaster* when restricted to certain yeasts alters the penetrance of *tu*^{50j}, a second chromosome recessive mutation located near 90 that produces melanotic growths in the abdomen. Other workers, Tannenbaum (*2*) and Herskowitz and Bur-

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dette (*3*), have found that nutrition influences tumor incidence. In general, poor nutrition or low calorie intake reduces the incidence of tumors. What, then, are the important nutritional factors that play a leading role in tumor production? In an attempt to solve this problem, the diet of Drosophila was rigorously controlled by employing a minimal diet that contains no vitamins or amino acids (Table 1), a diet which differs from the original one employed by Mittler (*1*) in that $NaNH_4HPO_4$ is used for the nitrogen source instead of $(NH_4)_2SO_4$. This medium is inoculated with *Hansenula anomala, H. saturnus,* or *Debaromyces globosus,* yeasts that can grow in the vitamin- and amino acid-free medium. Thus, the nourishment of the larvae was restricted to the yeast employed. A pair of flies was placed in small vials containing about 10 ml of medium to prevent overcrowding and to maintain a standard of comparison.

TABLE 1

MINIMAL MEDIUM

(VITAMIN- AND AMINO ACID-FREE)

Agar	15 (g)
$C_6H_{12}O_6$	10
K_2HPO_4	1
$NaNH_4HPO_4$	2
$CaCl_2$	0.25
$MnSO_4$	0.25
$MgSO_4$	0.25
$FeSO_4$	0.25
H_2O	1000 ml

Amino acids (1 g/1000 ml) were added to the minimal medium, and the incidence of tumors in adult flies was recorded. The results are presented in Table 2. The control experiments were those in which the highly inbred tu^{50j} were reared on minimal medium plus a yeast. The incidence of tumors is much higher when the flies are reared on the highly complex cornmeal-molasses mixture, which evidently contains a more complete diet, and this agrees with the reports in which tumor incidence is high if the nutrition is good. Excess amounts of tryptophane, lysine, and asparagine were found to increase the tumor incidence. Cystine, alanine, phenylalanine, and leucine decrease the penetrance of tu^{50j}. Tyrosine, glutamic acid, arginine, norleucine, histidine, methionine, and threonine have no apparent effect upon the tumor formation.

The role that tryptophane plays in tumor formation is not known; however, 1.4% of tryptophane added to the diet of white rats increased the occurrence of diethylstilbestrol-induced mammary cancer in rats, whereas 4.3% decreased tumor formation (*4*). One would expect that phenylalanine and tyrosine, which can be converted to melanin, would play a role in melanotic growths, but this is not the case in tu^{50j}. Excess amounts of some amino acids have been found to inhibit growth (*5, 6*), and this may explain the lowering of tumor incidence by cystine, alanine, phenylalanine, and leucine.

Phenylalanine (*6*) and arginine (*7*) were found to increase tumor incidence in the fat body of *D. melanogaster*; however, this is a different tumor than tu^{50j}. There is no apparent relationship (or if it does exist it is not known) between those amino acids that increase or decrease tumor incidence. In the case of tumor production in tu^{50j} stock, low caloric intake does reduce the tumor incidence, but not if large amounts of tryptophane, lysine, or asparagine are present, for more tumors are produced than when the flies are reared on a cornmeal-molasses medium enriched with dry yeast. Other nutritional substances

TABLE 2

PENETRANCE OF tu^{50j} WHEN REARED ON VARIOUS MEDIA

		Yeast Inoculants					
		Hansenula anomala		*Hansenula saturnus*		*Debaromyces globosus*	
		Percentage tu	No. flies	Percentage tu	No. flies	Percentage tu	No. flies
Cornmeal-molasses medium		9.2	1380	6.7	1053	6.3	1113
Minimal medium (Mm)		5.2	1259	3.8	1392	3.3	1302
Mm ± 1 g/1000 ml of	L cystine	1.5	1264	2.4	1251	1.1	1091
	DL alanine	2.7	1411	1.3	1233	3.1	1129
	L tryptophane	8.3	1447	7.4	1268	15.7	1338
	L tyrosine	3.7	1351	3.3	1182	1.5	1132
	Glutamic acid	4.3	1301	4.6	1239	4.3	1209
	L arginine	6.9	1163	5.8	1018	5.2	1289
	L lysine	14.6	1436	15.2	1117	11.1	1262
	DL phenylalanine	1.6	1184	2.1	1143	1.8	1222
	DL norleucine	3.9	1281	4.6	1194	4.1	1172
	L asparagine	12.1	1569	12.6	1268	7.7	1431
	Histidine	4.7	1237	5.2	1211	4.6	1214
	DL methionine	4.2	1119	4.6	1261	4.5	1264
	DL leucine	2.1	1189	1.6	1314	1.4	1193
	DL threonine	5.6	1232	4.9	1163	4.8	1228

besides amino acids are being investigated, for they may play an important role in tumor formation.

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Manuscript received June 9, 1952.

The Einthoven Triangle: An Observation Regarding the Validity of the Originally Proposed Triangular Representation of the Human Body[1]

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The Einthoven triangle, either by direct statement or implication, is usually treated as an equilateral triangular frame of reference inscribed within a homogeneous volume conductor in the form of (*a*) a plane circular slab or (*b*) a sphere (*1*). In some analyses the frame of reference is considered to be located within a homogeneous volume conductor of infinite dimensions (*1*). The potential difference between any two apices of the triangle may be regarded as the projection of an electrical vector, the so-called manifest potential, on a line joining the two apices. It may be shown that when the source of potentials in the volume conductor is an electric doublet situated in the plane of the frame of reference at its center, the manifest potential and the axis of the doublet are codirectional. This is of some importance, since the more nearly the human body approximates the conditions of idealization, the more nearly will manifest potentials, plotted from electrocardiographic potentials or recorded by means of the vectorcardiograph, represent *true* electrical vectors of the heart.

In contradistinction to the electrical models described above, the idealization originally proposed by Einthoven (*2*) consists of a thin slab of homogeneous volume conductor limited by an equilateral triangular boundary. The heart is represented by a centrally located doublet. Whether or not such a model is superior to the more commonly employed forms, it is of interest to ascertain by rigorous analytic methods whether the manifest potential calculated from the apex potentials of the Einthoven model also indicates accurately the direction of the axis of the doublet. We have been able to demonstrate both theoretically and experimentally that such an identity of orientation does exist.

[1] This work was supported in part by a grant from the Memphis Heart Association and a Cardiovascular Teaching Grant, National Heart Institute, U. S. Public Health Service.

The theoretical analysis was accomplished by converting the well-known case of potential distribution due to a centric doublet within a circle, by means of a Schwarz-Christoffel transformation, into the obscure situation of a centric doublet within an equilateral triangle. The circular case (Fig. 1) is that of a circle

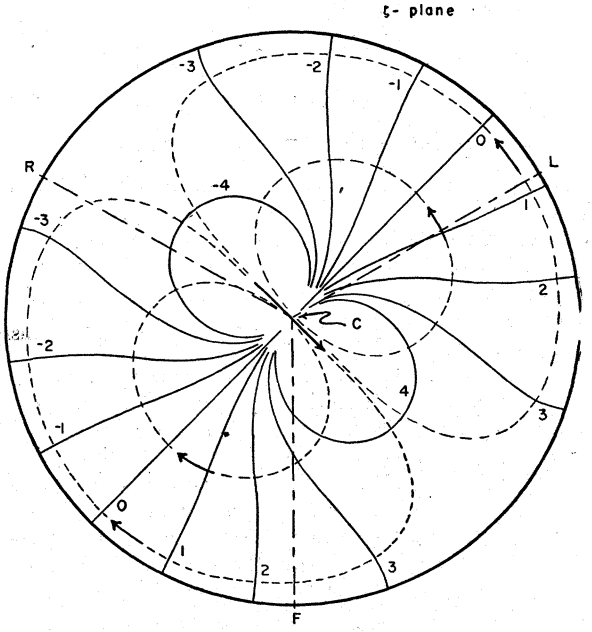

FIG. 1. Centric doublet in a circular, electrically homogeneous slab. Solid curves are isopotential lines; dashed curves are streamlines. Plotted on the basis of $V + iS = Mei^{\alpha}(1/\zeta + \zeta/R^2)$ where V is the potential function, S is the streamline function, M is the "strength" of the doublet, and α is the direction of its axis, ζ is a complex number representing any point in the plane, and R is the radius of the circle. In this figure $\alpha = -45°$. (The equation may be expressed in polar coordinates (r, θ) as a pair of conjugate functions, $V = M(1/r + r/R^2)$ cos $(\theta - \alpha)$ and $S = M(r/R^2 - 1/r)$ sin $(\theta - \alpha)$. These functions satisfy the Laplace equation $\nabla^2 V = \nabla^2 S = 0$. The boundary condition, $\delta V/\delta n = 0$, where n is the unit normal at the boundary, is also satisfied. These properties are not altered by the Schwarz-Christoffel transformation described in the text [*3*]).

of unit radius in the complex ζ-plane with three circumferential points equidistant from each other, $L(\sqrt{3}/2, i/2)$, $R(\sqrt{3}/2, i/2)$, and $F(0, -i)$. By substitution in an appropriate formula (*3*), a mapping function is determined which transforms the circle into an equilateral triangle in the complex w-plane, the coordinates of L, R, and F remaining unchanged. The mapping function is defined by the relation

$$dw = K(\zeta^3 - i)^{-\frac{2}{3}} d\zeta \qquad (1)$$

where K is a complex constant arbitrarily chosen so that the points L, R, and F will not be rotated by the transformation, or the distance between them altered, and $i = \sqrt{-1}$. Eq (1) can be solved for w by a binomial expansion of the radical expression followed by term-by-term integration. However, the desired information can be obtained without the necessity of resorting to such lengthy manipulations: The radii CL, CR, and CF can be expressed as