pressed contractile strength, increased refractory period and threshold, and partial conduction block—largely disappeared within about 30 min after washout (7). Therefore, quantitative testing for adrenergic blockade was usually carried out about 30 min after washout of DCI. Despite its persistence, the blockade appeared to be of the reversible competitive type, susceptible to "breakthrough" by very high concentrations ($\sim 10^{-5}$ g/ml) of epinephrine and norepinephrine. That the blockade was relatively specific was indicated by the fact that DCI did not significantly antagonize the positive inotropic effects of added Ca^{++} or strophanthin. On the other hand, the increase in strength with suprathreshold stimulation was antagonized by DCI as effectively as was that due to added epinephrine or norepinephrine (Fig. 1, *B* and *D*). This finding strongly supports the hypothesis that the increase with suprathreshold stimulation is mediated through release of some adrenergic substance (probably norepinephrine).

Additional strong evidence for the hypothesis was obtained with atria from guinea pigs which had been pretreated with reserpine (approximately 1 to 5 mg/g day intraperitoneally for 1 to 2 days) in order to deplete cardiac catechol amines. Such atria still responded well to added epinephrine or norepinephrine, but with suprathreshold stimulation they now gave either no significant increase in strength or a slight decrease (occasionally followed by a slight delayed increase) (Fig. 1, *E* and *F*). It should be noted that Burn has similarly used reserpine to show that the stimulating effect of nicotine on isolated rabbit atria is actually due to adrenergic mediator released by this agent (8). We have confirmed Burn's results with nicotine, using normal and reserpinized guinea-pig atria, and in addition have found that DCI can block the positive inotropic action of nicotine.

In preliminary experiments on cat papillary muscle, DCI and reserpine have yielded results completely analogous to those reported above for left atria of guinea pigs. Thus, we directly support Whelan's hypothesis that the increase in contractile force of papillary muscle with suprathreshold stimulation is due to the release of "one of the epinephrine compounds." However, we must disagree strongly with Whelan's suggestion that "epinephrines" and acetylcholine, released at each spontaneous beat or electrically driven beat at threshold voltages, are responsible for such phenomena as positive treppe and poststimulation potentiation. Such phenomena are still strikingly demonstrable in both guinea-pig atria and cat papillary muscles when potentiation due to suprathreshold stimulation has been eliminated by DCI or reserpine (9). Also, negative treppe in isolated rat atria is not altered by atropine blockade of the depression due to suprathreshold stimulation.

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4. The first part of the work discussed in this report was carried out at Washington University in 1953 by the senior author and Mrs. de Gubareff, with support from the American Heart Association. The second part, involving DCI and reserpine, was carried out at the State University of New York during the past year by the senior author and Mr. Grossman, with support from the New York Heart Association. We are indebted to Dr. I. H. Slater of Eli Lilly and Co. for the DCI and to Dr. A. J. Plummer of Ciba Pharmaceutical Products, Inc., for the reserpine used in this study.
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7. Lower concentrations of DCI (10^{-6} to 10^{-5} g/ml) produced insignificant side effects of the types mentioned and usually strengthened contractions somewhat but did not produce a sufficient degree of blockade for our purposes.
8. J. H. Burn and M. J. Rand, *Brit. Med. J.* 1958I, 137 (1958).
9. W. Sleator and T. de Gubareff [*The Physiologist* 1, 4, 73 (1958)] have recently used reserpine to show that treppe in guinea-pig atria is not due to release of adrenergic mediator.

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Earth Oblateness in Terms of Satellite Orbital Periods

Abstract. A theoretical equation relating the earth's oblateness to the anomalistic and nodical periods and orbit parameters of an earth satellite is presented. In the absence of exact data on nodical periods, Vanguard prediction data are utilized to obtain a check calculation for the oblateness and to establish the validity of the method.

The utility of artificial earth satellites for independent determination of the earth's oblateness by means of observations of the secular perturbations of the orbit [precession of the node and of perigee (1)] is well recognized. In the course of studies of such orbit perturbations, I have derived an equation for the difference between the anomalistic period T_A (perigee to perigee) and nodical period T_N (node to node) which should provide an additional independent means for determining the oblateness. Specifically,

$$T_A - T_N = \frac{JR^2 r_o^2 T_o}{a^4(1-e^2)^{5/2}}\left(2-\frac{5}{2}\sin^2 i\right) = \frac{2\pi JR^2 r_o^2}{(GM)^{1/2}a^{5/2}(1-e^2)^{5/2}} \times \left(2-\frac{5}{2}\sin^2 i\right) \quad (1)$$

where J is the coupling constant in the oblate earth's potential (2), R is the equatorial radius of the earth, r_o is the distance from the center of the earth to the satellite at the node, T_o is the satellite period for an assumed spherical earth, G is the constant of gravitation, M is the mass of the earth, a is the semimajor axis of the orbit, e is the orbital eccentricity, and i is the angle of inclination of the orbit to the plane of the equator.

Because of the precession of the line of apsides (3) r_o will range in magnitude from a perigee distance of $(1-e)a$ to an apogee distance of $(1+e)a$; and we come to the interesting conclusion that the difference between the anomalistic and nodical periods will exhibit a cyclic variation whose period is equal to that of the precessional motion of the line of apsides. The extremes of $(T_A - T_N)$ are then

$$(T_A - T_N)_{\substack{\text{min.}\\ \text{max.}}} = \frac{JR^2 T_o (1 \mp e)^2}{a^2(1-e^2)^{5/2}} \times \left(2-\frac{5}{2}\sin^2 i\right) = \frac{2\pi JR^2(1\mp e)^2}{(GM)^{1/2}a^{1/2}(1-e^2)^{5/2}} \times \left(2-\frac{5}{2}\sin^2 i\right) \quad (2)$$

which can differ by as much as 20 seconds for orbits of low inclination. Note that for $\sin^2 i = 4/5$ the difference in periods vanishes, which is in accord with the fact that at this inclination, 63.5°, the line of apsides neither advances nor regresses (3). For i less than 63.5°, the anomalistic period will be greater than the nodical period; and, conversely, at larger inclinations the nodical period will be the greater. In any case, the difference in periods increases with orbit eccentricity, and only for near-circular orbits ($e=0$) will there be no cyclic variations in $(T_A - T_N)$. Unlike the motions of the node and perigee, which fall off rapidly with distance, $(T_A - T_N)$ falls off very slowly with increasing orbit size; and hence the effect should be detectable even for orbits at great distance.

Equation 1 has been developed to first-order in the oblateness parameter J on the presumption of no drag or other perturbing factors. For satellites with perigees above the region of sensible atmospheric drag the orbit parameters should be measurable to great accuracy, which

in turn will be reflected in precise determination of J. Even for orbits in which atmospheric drag will slightly perturb the periods, the alteration in T_A should be very closely the same as that in T_N, so that the difference should be relatively unaffected by drag.

With the exception of satellite 1958β2 (Vanguard I), the existing satellite orbits are too seriously affected by drag for the above equations to be precisely applicable; and even for Vanguard I precise data on nodical periods have not yet been published. In the absence of such information one can utilize the predicted times of equator crossings, as issued by the Vanguard Computing Center and the Naval Research Laboratory, to derive approximate nodical periods. In this manner values of $(T_A - T_N)$ were calculated and plotted as a function of equator pass number in Fig. 1. The predicted cyclic variation, with minima and maxima corresponding to perigee and apogee occurrences near the node, is indeed evident. The lack of complete symmetry in the curve is a consequence of the fact that values of the nodical period were interpolated to the nearest 0.1 second, while the predicted times of equator crossings were given only to the nearest second.

On the basis of the Vanguard prediction data and Eq. 1, a value for the oblateness parameter has been calculated as $J = 0.001631 \pm 0.000031$. In turn this corresponds to an earth oblateness (*2*) of $1/297.6 \pm 2.7$. This is to be compared with the international value of 1/297.0 and O'Keefe's preliminary value (*4*) of $1/298.3 \pm 0.1$, which was derived from the secular motions of the node and perigee of satellite 1958β2.

Obviously, in spite of the near agreement between the value herein obtained and the other quoted values for the oblateness, little significance can be attached to this figure because of the associated large statistical probable error. However, even from these approximate calculations, it is clear that a significant check on the validity of the theory has been obtained. Since no method for independent determination of the earth's oblateness should be left unexplored, it is urged that every effort be made by tracking stations and computation centers to determine satellite anomalistic and nodical periods accurately.

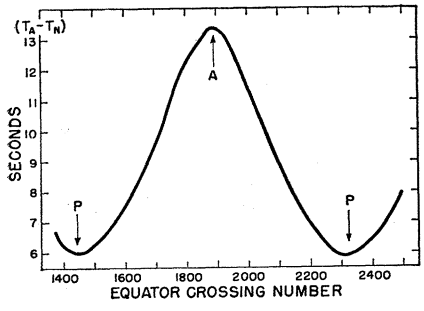

Fig. 1. Difference between anomalistic and nodical periods as a function of equator pass number for satellite 1958β2. Approximate perigee (P) and apogee (A) occurrences at the equator are indicated by arrows (*5*).

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References and Notes

1. Those points on the celestial sphere where the satellite crosses the equator are called the nodes. The point on the orbit nearest the earth is called the perigee, while the major axis, extended indefinitely, is called the line of apsides.
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5. Derived from prediction data issued by the Vanguard Computing Center and the Naval Research Laboratory.

19 November 1958

Inhibition of Growth of Excised Tomato Roots by 2-Diethylaminoethanol

Abstract. Approximately 40 μ*M* 2-diethylaminoethanol (DEAE) caused 50-percent inhibition of growth of the main axis. Inhibition was relieved by 2-dimethylaminoethanol and choline but not by ethanolamine. A marked morphogenetic effect of DEAE is attributed to differences in sensitivity of main and lateral meristems and to an effect upon the postulated hormonal system controlling apical dominance.

The work reported here forms part of a project designed to study the replacement of vitamin B_6 by ethanolamine (2-aminoethanol) in the nutrition of excised tomato roots (*1*).

Ethylation of the amino group of ethanolamine gives compounds which, because of their structural similarity to the metabolites 2-mono-, 2-di-, and 2-trimethylaminoethanol (choline), are possible antimetabolites which could be useful in the study of the metabolism of ethanolamine. No reports of the effects of such ethylated derivatives on plant growth are known to me, although some indication of the value of these compounds as inhibitors is given from work with animals. Thus the incorporation of the ethyl carbon of ethionine into choline and creatine of rat tissue (*2*) suggested that growth inhibition by ethionine could be due, at least in part, to inhibitory effects of ethyl analogs of choline or substances containing choline. Subsequently (*3*) it was shown that triethylcholine inhibited the growth of rats. The inhibition was relieved by choline and, to a lesser extent, by methionine.

2-Diethylaminoethanol is not a proven antimetabolite. However it forms part of the structure of a number of drugs, and, consequently, it has been studied by animal physiologists who have reported various pharmacological effects (*4*). It is not known whether any such effects are due to interference with choline metabolism. However, in a study of the oxidation of choline-like substances by rat-liver preparations (*5*) it was found that, although choline and a number of structural analogs were oxidized, DEAE was not oxidized. Furthermore, DEAE gave a 25-percent depression of choline oxidation by the homogenate. All these results suggested that DEAE might be valuable as an inhibitor in vivo.

A sample of DEAE was given to me by the Jefferson Chemical Company (New York). This report describes the inhibitory effects of DEAE on growth of excised tomato roots grown in sterile culture and some nutritional experiments on the reversal of the inhibition. The clone of excised tomato roots used as a source of inocula is designated R5 (*6*). The general experimental techniques, and some cultural requirements of the clone, are described elsewhere (*6, 7*). All additions to the basal medium used here were autoclaved in the medium. The measurements recorded are of roots grown for 6 days from 10-mm tips and are measurements of the final length of the main axis per root and of the total length of the ten basal laterals per root. The number of laterals per root in all experiments was found to be proportional to the length of the main axis and is, therefore, omitted from the results.

The effects of a range of concentrations of DEAE on the growth of roots is shown in Fig. 1. There was a marked difference between the growth response to DEAE of the main axis and of the lateral roots. The inhibition of lateral growth at low concentrations was relieved at higher concentrations which inhibited growth of the main axis. The explanation of this differential effect probably lies in the observation (*8*) that apical dominance (inhibition of lateral roots by the main apex) is manifested in excised tomato roots. In the presence of a growth inhibitor, the growth of lateral roots will be controlled by the inhibitions due to both the exogenous inhibitor and the factors causing apical dominance. Presumably, at those concentrations of DEAE which relieved the inhibition of lateral growth caused by low concentrations of DEAE, the simultaneous inhibition of main axis growth resulted in a removal of the factors causing apical dominance. This, in turn, led to an in-