are compatible with the view that some of the central effects of reserpine are mediated through the release of serotonin. It is conceivable that the beneficial effects of reserpine in mental disturbances result from the liberation of serotonin. The possibility that reserpine also affects the level of serotonin in brain is now under investigation.

ALFRED PLETSCHER*
PARKHURST A. SHORE
BERNARD B. BRODIE
Laboratory of Chemical Pharmacology, National Heart Institute, National Institutes of Health, U.S. Public Health Service, Bethesda, Maryland

References and Notes

1. J. H. Gaddum, Ciba Foundation Symposium, London, (1953); D. W. Woolley and E. Shaw, *Proc. Natl. Acad. Sci.* (*U.S.*) 40, 228 (1954).
2. P. A. Shore, S. L. Silver, B. B. Brodie, *Experientia*, in press.
3. ———, *Science* 122, 284 (1955).
4. J. H. Gaddum, *J. Physiol.* 121, 15P (1953).
5. W. A. Stoll, *Schweiz. Arch. Neurol. Psychiat.* 68, 279 (1947).
6. S. Udenfriend and E. O. Titus, *Federation Proc.* 13, 411 (1954).
7. S. Udenfriend, H. Weissbach, C. T. Clark, *J. Biol. Chem.*, in press.
8. R. L. Bowman, P. A. Caulfield, S. Udenfriend, *Science* 122, 32 (1955).

* Guest worker from Hoffmann-La Roche, Basel, Switzerland.

25 May 1955

Burger Triangle as a Method for Correcting Inaccuracies of Einthoven Triangle

The triangle of Burger and van Milaan (*1*) was constructed on the basis of the lead vector concept. Burger and van Milaan demonstrated that, in contrast with the triangle of Einthoven, their triangle is accurate for calculating cardiac vectorial directions in the frontal plane of a human phantom, regardless of thorax form, tissue nonhomogeneity, and eccentricity of the assumed resultant heart vector. The shape of the Burger triangle is determined by these factors; the three sides are usually unequal (though the influence of the dispersion of the electromotive forces of the heart is not considered in their concept). To use such a triangle it is necessary, before beginning the usual procedure, to divide the deflections written in the classic limb leads by the length of the corresponding sides. Subsequent studies (*2–8*) suggest that the Burger triangle may prove to be more valuable in clinical electrocardiography than the Einthoven triangle.

The present communication demonstrates the relationship between the shape of the Burger triangle and the inaccuracy of the Einthoven triangle in the calculation of the vectorial directions in the frontal plane. A Burger triangle from a given subject (human or animal, living or dead, or electrolytic model) could have one of three shapes: equilateral, isosceles, or scalene. If it is equilateral, which is exceptional, the Einthoven triangle is obviously accurate. If it is not equilateral, the Einthoven triangle is inaccurate. But isosceles or scalene triangles can have various accentuations and departures from the equilateral triangle.

Figure 1 shows four hypothetical Burger triangles from four subjects; *a* and *b* are isosceles, whereas *c* and *d* are scalene. But *b* and *d* depart more from the equilateral than do *a* and *c*. For convenience, a Burger triangle may be transformed to a triaxial reference system (just as the Einthoven triangle has been transformed to the triaxial reference system of Bayley) with parallel transposition of the three sides of the Burger triangle toward its geometric center until they coincide. In the same figure four triaxial reference systems—transformed from Burger triangles *a, b, c,* and *d,* respectively—are shown.

We may designate *l, m,* and *n* as the lengths of the lead vectors *RL, RF,* and *LF* of a Burger triangle; and p_1, p_2, and p_3 as projections of the heart vector upon *l, m,* and *n*. On the basis of the concept that the deflection of an electrocardiographic lead equals the scalar product of heart vector and the lead vector, one gets

$$L_I = lp_1,\ L_{II} = mp_2;\ L_{III} = np_3$$

In order to demonstrate the relationship between the Burger triangular shapes in Fig. 1 and the inaccuracy of the Einthoven triangle, it is necessary to assume the heart vector to be of equal length in the same arbitrary direction, V (+45°), in each case. From the terminus of V in each triaxial reference system, perpendicular lines to three sides were drawn; p_1, p_2, and p_3 were measured. From the equations, the deflections in L_I, L_{II}, and L_{III} were calculated. The values were used to plot the vectorial direction for each subject in the triaxial reference system of Bayley (Fig. 2). The directions *a, b, c,* and *d* correspond to subjects with Burger triangles *a, b, c,* and *d,* respectively. It may be observed that they deviate from V (the arbitrary "true" direction); that $\angle boV$ is larger than $\angle aoV$, and that $\angle doV$ is larger than $\angle coV$.

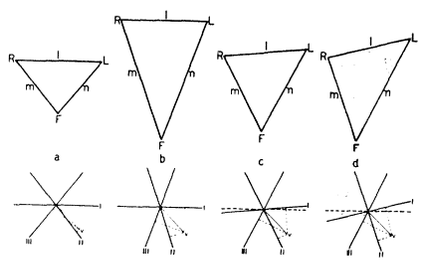

Fig. 1. Four hypothetical Burger triangles and corresponding triaxial reference systems. V is the arbitrarily true heart vector, which is assumed to be the same in each case.

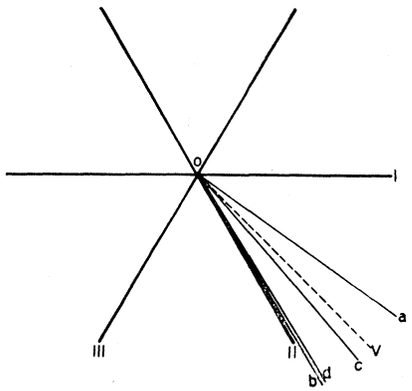

Fig. 2. The triaxial reference system of Bayley showing the directions of V and the calculated vectors.

The Einthoven triangle is inaccurate for subjects possessing Burger triangles either of isosceles or scalene shape. The more the triangle departs from the equilateral, the more the vectorial direction, calculated in the Einthoven triangle, deviates from the true one. Use of the Burger triangle permits correction of these potential errors.

ZANG Z. ZAO
Ribeirão Preto Medical School, University of São Paulo, Brazil

References

1. H. C. Burger and J. B. van Milaan, *Brit. Heart J.* 8, 157 (1946); 9, 154 (1947).
2. F. N. Wilson, J. M. Bryant, F. D. Johnston, *Am. Heart J.* 37, 493 (1949).
3. A. G. T. Becking, H. C. Burger, J. B. van Milaan, *Brit. Heart J.* 12, 339 (1950).
4. R. McFee, R. Stow, F. D. Johnston, *Circulation* 6, 21 (1952).
5. D. A. Brody and W. E. Romans, *Am. Heart J.* 45, 263 (1953).
6. E. Frank and C. F. Kay, *Circulation* 9, 724 (1954).
7. Z. Z. Zao, paper presented at 6th annual meeting, Brazil Association for the Advancement of Science, 1954.
8. ———, *Z. Kreislaufforschg.*, in press.

14 April 1955

Photochemical Activity of Chloroplasts Isolated from Sugar Beet Infected with Virus Yellows

Sugar beet virus yellows is a serious disease in Europe, where even the mild forms can reduce sugar yields by more than 20 percent (*1*). The disease now appears to be widespread in the western United States. Watson and Watson (*2*)

showed that the disease decreased the net assimilation rate of the plant and suggested, as one interpretation, that photosynthesis was slowed. Our experiments show that the disease interferes with the photochemical activity of the chloroplasts. This could decrease the amount of photosynthate available, which could well result in a decreased sugar yield. The effects of pathological conditions on the metabolic properties of chloroplasts have apparently not been investigated.

Kausche and Ruska (*3*), and Black, Morgan, and Wyckoff (*4*) published electron micrographs that indicated the presence of tobacco mosaic virus within the chloroplasts of infected tobacco plants. More recently Leyon (*5*) published electron micrographs of preparations from leaves infected with beet yellows virus that showed filamentous particles associated with the chloroplasts. Leyon suggested that the filaments represent the virus and that at least some of the virus was formed within the chloroplasts, although Nixon and Watson (*6*) argued that these filaments represent only a small part of the anomalous material in infected plants.

These experiments (*7*) were carried out with chloroplast fragments from leaves (*8*) of control and virus-yellows inoculated sugar beet plants (var. U.S. 75). Chloroplast fragments were prepared and stored as previously described (*9*). Chloroplast activity was measured

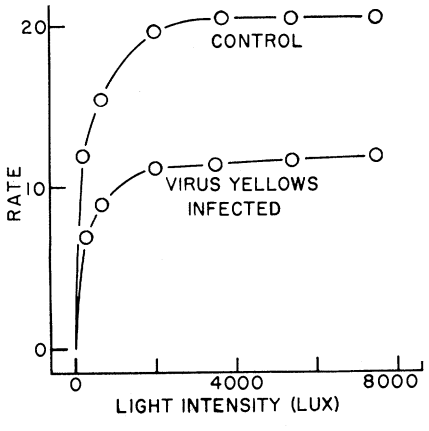

Fig. 1. Curves showing the effect of light intensity on the photochemical activity of chloroplast fragments isolated from control and virus-yellows-infected sugar beet leaves. The rates are expressed as millimoles of ferricyanide reduced per hour, per milligram of chlorophyll; the incident light intensities are expressed in lux. The reaction system was 0.0005*M* in potassium ferricyanide, 0.01*M* in potassium chloride, 0.10*M* in potassium phosphate buffer of *p*H 6.80, and 0.17*M* in sucrose. The reaction system (3.0 ml) contained chloroplast fragments equivalent to 100 mg/lit ($1.1 \times 10^{-4}M$) chlorophyll. Illumination was provided by reflector-type incandescent bulbs; the reaction was run in flowing tank nitrogen at a temperature of 15°C.

by a potentiometric technique (*10*) as a function of light intensity in order to determine whether the virus affected the rate-limiting photochemical reaction, the rate-limiting dark reaction, or both (*11*).

The results of a typical experiment with plants raised under normal fertility are shown in Fig. 1. The slope of the curves at zero light intensity is proportional to the rate-limiting photochemical reaction; the asymptote approached by the curves at infinite light intensity is a measure of the rate-limiting dark reaction. The curves show that the rates of both processes are decreased in the chloroplast fragments from the infected plants. For more quantitative determinations, the data may be plotted in the form (light intensity)/(velocity) versus (light intensity). In this form straight lines are obtained in which the rate constant k_L for the limiting photochemical reaction is proportional to 1/intercept, and the rate constant k_D for the limiting dark reaction is proportional to 1/slope as discussed elsewhere (*11*). The values of the rate parameters as calculated in this way by the method of least squares are as follows: for control plants, $k_L = 0.12$, $k_D = 22.8$; for infected plants, $k_L = 0.07$, $k_D = 12.5$. Thus, both of the rate-limiting chloroplast reactions are decreased by approximately 50 percent (on a chlorophyll basis) in the infected plants.

These preliminary experiments indicate that the beet yellows virus could decrease photosynthesis and sugar production in the plant by a direct action on the chloroplasts rather than by some indirect effect on the photosynthetic mechanism or by interference with the translocation of carbohydrate in the phloem as has been shown in the case of the curly top virus of sugar beet. The results further indicate that the degree of yellowing of the infected leaves is not necessarily related to the severity of chloroplast injury, for the decrease in chloroplast activity noted is calculated on a chlorophyll basis.

JOHN D. SPIKES
Department of Experimental Biology, University of Utah, Salt Lake City
MYRON STOUT
Field Crops Research Branch, Agricultural Research Service, U.S. Department of Agriculture, Salt Lake City

References and Notes

1. F. C. Bawden, *Plant Viruses and Virus Diseases* (Chronica Botanica, Waltham, Mass., 1950), p. 7.
2. J. D. Watson and M. A. Watson, *Ann. Appl. Biol.* 40, 1 (1953).
3. G. A. Kausche and H. Ruska, *Naturwissenschaften* 28, 303 (1940).
4. L. M. Black, C. Morgan, R. W. G. Wyckoff, *Proc. Soc. Exptl. Biol. Med.* 73, 119 (1950).
5. H. Leyon, *Arkiv Kemi* 3, 105 (1951); *Exptl. Cell Research* 4, 362 (1953).
6. H. L. Nixon and M. A. Watson, *Nature* 168, 523 (1951).
7. This work was supported by grants from the U.S. Atomic Energy Commission and the University of Utah Research Fund.
8. The leaf samples were made available through the courtesy of Charles Price, Agricultural Research Service, Field Crops Research Branch, U.S. Department of Agriculture, Riverside, Calif.
9. J. D. Spikes, *Arch. Biochem. and Biophys.* 35, 101 (1952).
10. J. D. Spikes *et al.*, *Plant Physiol.* 29, 161 (1954).
11. R. Lumry, J. D. Spikes, and H. Eyring, *Ann. Rev. Plant Physiol.* 5, 271 (1954).

28 April 1955

Hemagglutination after Immunization with Schistosome Antigens

Boyden (*1*) and recently Stavitsky (*2*) reported the adsorption of protein antigens on sheep erythrocytes treated with tannic acid and their subsequent hemagglutination by specific antiserums. This technique was adapted to the antigen-antibody system in schistosomiasis. Agglutination of living cercariae of *Schistosoma mansoni* has been previously reported in the serology of schistosomiasis by Liu and Bang (*3*), Standen (*4*), and Stirewalt and Evans (*5*). In our investigations agglutination was observed in some but not all samples of normal, full-strength, inactivated serum of man, horse, cow, sheep, steer, goat, dog, hamster, rabbit, and pig. In the serums of 13 vertebrate species immunized with frozen cercariae of *S. mansoni, in vitro* agglutination of living cercariae was observed during the course of immunization (*6*). The agglutinin titer for living cercariae varied from 1:8 to 1:128, and a more sensitive and specific technique for the detection and titration of these antibodies was desirable.

The method of conducting the hemagglutination test (*7*) followed Boyden (*1*). Sheep cells in Alsever's solution were washed in buffered saline (*p*H 7.2) and incubated with a 1:20,000 dilution of tannic acid for 10 min at 37°C. The packed tanned cells were then exposed to 5 vol of a 1:5000 dilution by weight of lyophylized cercariae of *S. mansoni* (0.001 mg of lyophylized cercariae per 5 ml of saline buffered at *p*H 6.4) for 15 min at room temperature. Coated cells were adjusted to a 2-percent suspension in 1:250 normal rabbit serum. Serial dilutions were made in 1:100 normal rabbit serum. One drop of antigen-treated cells was added to each tube with an Ives dropping pipette. The test was read after 2 hr and after the cells had remained at room temperature (23°C) overnight.

Hemagglutination titers were obtained for the serums of 16 rabbits immunized by six intravenous injections administered twice weekly for 3 wk with various stages of the life-cycle of *S. mansoni* and *Schis-*