tion of photolytically formed hydrogen atoms with the flavin in the ground state, but, under the present conditions, the hydrogen atoms are scavenged by the methanol, forming H_2.

Since the Q of H_2 is, in general, much less than the $Q(e^-_{aq})$ value (see Table 2), it can be assumed that e^-_{aq} and the flavin triplet are mainly engaged in the formation of semiquinone and of the leuco (1,5-dihydro) form of the flavins. The electron-ejection process of flavins by illumination at 253.7 nm very likely takes place from the singlet manifold, as has already been demonstrated for phenol and related compounds (*15*), aromatic amines (*19*), and monophenylphosphate (*21*).

N. Getoff
S. Solar
Institut für Theoretische Chemie und Strahlenchemie, Universität Wien, A-1090 Wien, Austria
D. B. McCormick
Section of Biochemistry, Molecular, and Cell Biology and *Division of Nutritional Sciences, Cornell University, Ithaca, New York 14853*

References and Notes

1. B. Holmström and G. Oster, *J. Am. Chem. Soc.* **83**, 1867 (1961).
2. W. M. Moore, J. T. Spence, F. A. Ramond, S. D. Colson, *ibid.* **85**, 3367 (1963).
3. E. C. Smith and D. E. Metzler, *ibid.*, p. 3285; C. S. Yang and D. B. McCormick, *ibid.* **87**, 5763 (1965); W. L. Cairns and D. E. Metzler, *ibid.* **93**, 2772 (1971).
4. W. M. Moore and C. Baylor, *ibid.* **91**, 7170 (1969).
5. M. Gladys and W. R. Knappe, *Chem. Ber.* **107**, 3658 (1974).
6. P. S. Song and D. E. Metzler, *Photochem. Photobiol.* **6**, 691 (1967); G. E. Treadwell, W. L. Cairns, D. E. Metzler, *J. Chromatogr.* **35**, 376 (1968).
7. B. Holmström, *Photochem. Photobiol.* **3**, 97 (1964); K. G. Radda, *Biochim. Biophys. Acta* **112**, 448 (1966).
8. M. Green and G. Tollin, *Photochem. Photobiol.* **7**, 129 (1968); N. Lassel and J. Feitelson, *ibid.* **21**, 249 (1975).
9. D. B. McCormick, in *Excited States of Organic Chemistry and Biochemistry*, B. Pullman and N. Goldblum, Eds. (Reidel, Dordrecht, 1977), pp. 233–235.
10. S. P. Vaish and G. Tollin, *Bioenergetics* **1**, 181 (1970); *ibid.* **2**, 61 (1971); J. M. Gillard and G. Tollin, *Biochem. Biophys. Res. Commun.* **58**, 328 (1974).
11. D. B. McCormick, M. C. Falk, F. Rizzuto, G. Tollin, *Photochem. Photobiol.* **22**, 175 (1975).
12. D. B. McCormick, *ibid.* **26**, 169 (1977).
13. W. M. Moore and R. C. Ireton, *ibid.* **25**, 347 (1977).
14. L. I. Grossweiner, G. W. Swenson, E. F. Zwicker, *Science* **141**, 805 (1963); L. I. Grossweiner and H. I. Joschek, *Adv. Chem. Ser.* **50**, 279 (1965); J. Jortner, M. Ottolenghi, G. Stein, *J. Am. Chem. Soc.* **85**, 2712 (1963).
15. J. Zechner, G. Köhler, G. Grabner, N. Getoff, *Chem. Phys. Lett.* **37**, 297 (1976); G. Grabner, G. Köhler, J. Zechner, N. Getoff, *Photochem. Photobiol.* **26**, 449 (1977).
16. G. Köhler, J. Zechner, G. Grabner, N. Getoff, in *Abstracts of the Seventh International Congress on Photobiology* (Italian Organization Committee, Rome, 1976), p. 11.
17. H. B. Steen, *J. Chem. Phys.* **61**, 3997 (1974).
18. T. R. Hopkins and R. Lumry, *Photochem. Photobiol.* **15**, 555 (1972).
19. G. Köhler, C. Rosicky, N. Getoff, in *Excited States of Organic Chemistry and Biochemistry*, B. Pullman and N. Goldblum, Eds. (Reidel, Dordrecht, 1977), pp. 303–311.
20. N. Getoff and S. Solar, *Monatsh. Chem.* **105**, 241 (1974).
21. G. Köhler and N. Getoff, *J. Chem. Soc. Faraday Trans.* **74**, 1029 (1978).
22. L. I. Grossweiner, A. G. Kaluskar, J. F. Baugher, *Int. J. Radiat. Biol.* **29**, 1 (1976).
23. W. Föry, R. E. MacKenzie, D. B. McCormick, *J. Heterocycl. Chem.* **5**, 625 (1968).
24. M. C. Falk, P. G. Johnson, D. B. McCormick, *Biochemistry* **15**, 639 (1976).
25. N. Getoff and G. O. Schenck, *Photochem. Photobiol.* **8**, 167 (1968).
26. R. N. Smith, P. A. Leighton, W. G. Leighton, *J. Am. Chem. Soc.* **61**, 2299 (1939); M. Neumann-Spallart and N. Getoff, *Monatsh. Chem.* **106**, 1359 (1975).
27. R. L. Willson, C. L. Greenstock, G. E. Adams, R. Wageman, L. M. Dorfman, *Int. J. Radiat. Phys. Chem.* **3**, 211 (1971).
28. R. W. Fessenden and N. C. Verma, *Discuss. Faraday Soc.* **63**, 104 (1977).
29. J. P. Keene, *Radiat. Res.* **22**, 1 (1964).
30. Two of us (N.G. and S.S.) received financial support from the Fonds zur Förderung der Wissenschaftlichen Forschung and the Ludwig Boltzmann Gesellschaft in Austria. D.B.M. was supported by research grant AM-04585 from the National Institutes of Health.

19 October 1977; revised 6 March 1978

Experiments with Controlled Langmuir Circulations

Abstract. *Laboratory experiments in which wind was blown gently over a regular crossed-wave pattern have clearly shown the existence of regular, steady, and reproducible Langmuir circulations. The circulations were present only when both the wind and the wave patterns existed at the same time. With circulations having an across-wind wavelength of 44 centimeters, the descending vertical motions carried turbulent surface water downward 39 centimeters to the bottom of the wind and wave tank. The observations are in general agreement with the mathematical model of Craik and Leibovich, although further experiments will be required to definitively test the analytical model.*

The phenomena known as Langmuir circulations (LC's) consist of longitudinal roll vortices in the surface mixed layers of lakes and oceans. Their axes lie more or less parallel to the wind direction, there may be more than one scale present at any time, and the spacings of the convergence lines of the largest scale may be comparable to the depth of the mixed layer (*1*). These circulations were first systematically studied by Langmuir (*2*), who pondered the cause of lines of seaweed in the open ocean with spacings of the order of 100 m and similar but smaller-scale (1 to 10 m) lines of floating debris in Lake George, New York. From a variety of ingenious experiments over a period of several years, he concluded that these circulations were "the essential mechanism by which the epilimnion is produced" (*2*, p. 123). He believed that wind stirring was the primary cause of mixing and that organized rolls (as opposed to more random turbulence) were the dominant mechanism for the vertical transport of heat and momentum. The importance of LC's for planktonic activity has long been recognized (*3*), for these circulations concentrate floating materials in lines and may cause slightly buoyant particles to undergo large vertical excursions into and out of the photic zone.

An understanding of the basic mechanism leading to the generation of LC's has been clouded by the complexity of the natural environment and the consequent lack of definitive observations. A number of plausible theories have been advanced but most of these have been shown to be inadequate (*4*). An idealized mathematical model by Craik and Leibovich (*5*), however, has recently attracted wide attention and has been followed by a series of papers (*6–9*) that extend the application of the basic Craik-Leibovich (CL) model to more realistic conditions. The significance of these later papers, however, rests on some of the same basic premises as the original CL model. Accordingly, the goal of this research has been to experimentally test the original idealized model. That model involves a nonlinear interaction of the shear flow produced by the wind stress with two crossing trains of surface waves. In brief, the spatially variable Stokes drift associated with the pattern of waves is believed to twist the vorticity of the shear flow into the vorticity of the LC's.

Figure 1 illustrates the essential geometry of a wind and wave tank in which a regular pattern of crossed waves is generated by a double wedge that oscillates vertically at a resonant frequency σ. A light wind is drawn over the waves by an exhaust fan at the far end of the tank. With the wind alone or with the waves alone no LC's occur. With both wind and waves, however, the well-organized circulations depicted in Fig. 2 are observed.

A series of experiments was carried out with the following conditions: wind speed U = 173 cm/sec measured at a height z = 7 cm above the water surface, σ = 9.94 sec^{-1}, and wave geometry as indicated in Figs. 1 and 2. The circulation was determined from cine photography of surface floats and $KMnO_4$ crystals sprinkled over the bottom. Figure 2 shows how the floats (weighted polyethylene rods 2.54 cm long and 1.27 cm in diameter) converged into two lines in a matter of 20 seconds. At the same time streaks from the dye crystals showed a three-band pattern of convergence in the return flow at the bottom.

Analysis of the float trajectories from repeated experiments with the same conditions is represented by the data in Fig. 3. The essential results were that (i) the same transient response occurred immediately after turning on the waves or after turning on the wind, the other component being already in action; (ii) the exponential response time for the development of the cells near the surface was $T = 12$ seconds; (iii) the steady-state maximum across-wind component of velocity at the surface was $V^* = 0.65$ cm/sec; and (iv) the dye at the bottom remained undisturbed for 90 ± 20 seconds after the wind or the waves were started. After 90 seconds the dye was disrupted by turbulence that was carried to the bottom directly beneath the two surface convergence lines. The dye pattern (Fig. 2) then developed quickly and wisps of dye could be seen ascending from each of the convergent zones. The time of 90 seconds corresponds to a vertically averaged (and time averaged) downwelling speed of 0.43 cm/sec. Interior dye tracers confirmed the regular (but turbulent) cellular structure. Since the wind had a rather short fetch over the water, the depth of the turbulent layer with the wind alone was only a few centimeters, but with both wind and waves the entire fluid layer became turbulent through the convective action of the cells. The floats remained perfectly aligned for the entire useful length of the tank, which was about 3 m beyond panel 4 (Fig. 1), the principal area of observation.

Certain interesting estimates can be made from the available data. As a rough approximation, a spatially and temporally constant eddy viscosity ν_e is assumed, and the cells are taken to be square and periodic so that the wave number of the cells, $l = 2\pi/L$, where L is the across-wind wavelength of the LC's, is the same in the horizontal and vertical directions. Then assuming in addition (i) a source of vorticity independent of the strength of the cells, (ii) that when the cells are fully developed the source of vorticity is balanced by viscous (eddy) dissipation, and (iii) that internal dissipation dominates side wall and bottom effects, it follows from linear theory that the exponential time constant for the rate of generation of V^* should be given by $T = (2\nu_e l^2)^{-1}$. Using $T = 12$ seconds, it is found that $\nu_e = 2$ cm^2/sec, a reasonable value. The corresponding rate of dissipation of kinetic energy of these simple cells is given by $\rho \nu_e V^{*2} l^2 L/2$ (ρ is the fluid density), which equals 0.38 erg/cm^2-sec for the data given above. The rate of energy input by wind stress is given by $\tau_0 u_0$, where $\tau_0 = 0.065$ dyne/cm^2 was the estimated wind stress (*10*) and $u_0 = 9$ cm/sec was the measured water surface speed. This energy input was therefore 0.59 erg/cm^2-sec. Thus the rate of energy dissipation in the cells was of the same magnitude at the rate of energy input by the wind stress, but it should be noted that some of the energy for the cells may be drawn from the waves and may be a significant sink of energy for the waves. If so, LC's may represent a mechanism whereby wave energy is converted to that of organized convection and turbu-

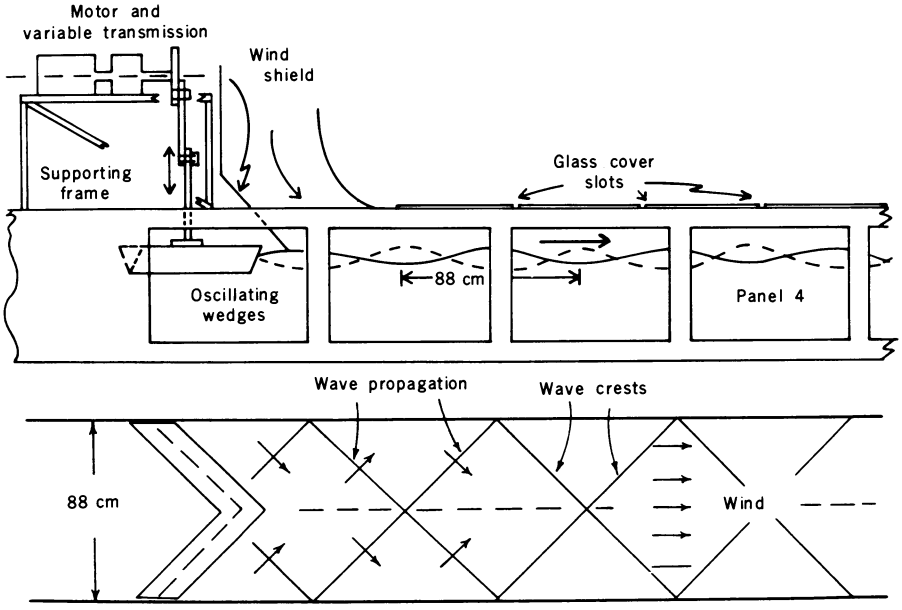
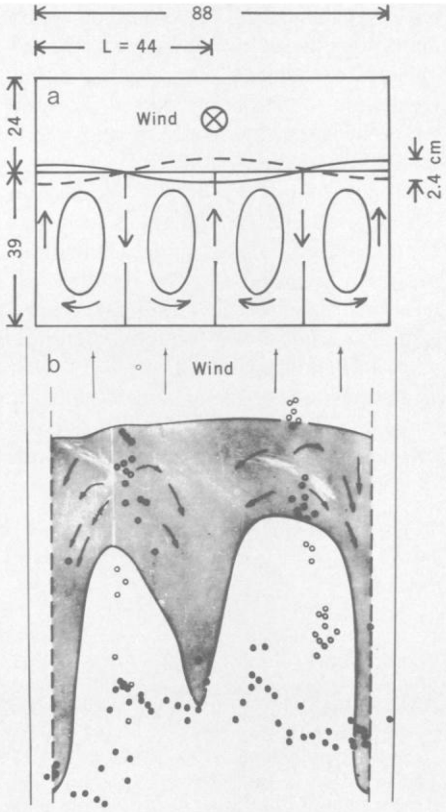


Fig. 1. Essential features of the wind and wave tank and the regular pattern of waves produced by the double-wedge wave generator.



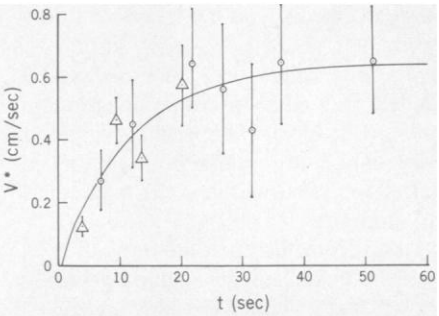


Fig. 2 (left). Langmuir circulations generated by wind over a regular pattern of waves. (a) Vertical section indicating the scale and sense of rotation of the cells relative to the wave pattern. (b) Plan view of the displacement of surface floats from their initial positions (●) to their positions 20 seconds later (○). The shaded area indicates the corresponding bands of convergence of dye at the bottom. Arrows indicate streaks from individual dye crystals. The side walls of the tank at the water surface are indicated by solid lines. The dashed lines indicate the walls at the bottom, displaced inward by photographic parallax. Fig. 3 (right). Rate of development of the Langmuir circulations after turning on the wind (△) or the waves (○), the other component (waves or wind) being already in action. The points were computed by fitting a sine function with amplitude V^* to the across-wind displacements of the floats over a 5-second interval. The error bars represent 1 standard deviation in the variation of the observations from the assumed sine function. The abscissa is running time (t).

lence for deepening of a mixed layer.

With the estimates of τ_0 and ν_e, it is now possible to evaluate certain parameters in the CL model. The Langmuir number (*6*) is given by La = $(\nu_e k/Au_*)(\nu_e/\sigma)^{1/2}$, where A is the wave amplitude, k is the wave number of the surface waves, and $u_* = (\tau_0/\rho)^{1/2}$ is the friction velocity. For this experiment La = 0.59, compared to the value La = 0.01 used in the computations of (*6*) and (*7*). A smaller value of La implies faster growth of the cells. In the CL model the time scale for the growth of the cells is given by $T_d = (\nu_e\sigma)^{1/2}/\sigma u_* Ak$, and it is suggested (*6, 7*) that a time of 10 T_d is required for full development of the cells. For the experimental data it is found that T_d would be 29 seconds. The experimental response time for V^* was T = 12 seconds, a somewhat faster response than predicted by the theory, although there is some uncertainty in the value of ν_e and other assumptions.

The observed circulations are in general agreement with the CL model in that both wind and waves were necessary for the formation of the LC's and that the upwelling occurred beneath the traces of the wave crest intersections, as shown in Fig. 2. Trial reversals of the wind direction clearly reversed the circulation of the cells, a result that is also in agreement with the CL model, since the sign of the vorticity due to the wind stress was reversed. It is conceivable, however, that some wind-wave-turbulence interaction other than that of the CL model may also produce longitudinal rolls, and further experiments over a wide range of parameters will be required to definitively test the mathematical model.

Apart from the comparison with theory, the experiments have definitely shown that a light wind blowing over a pattern of waves of rather small amplitude can generate well-organized circulations. The characteristic time for the growth of surface cells in this case was about 18 wave periods. In a more complex pattern of waves it should be expected that circulations of many scales will be generated, perhaps with a dominant scale determined by the pattern of the dominant waves. Larger, secondary scales may then form by nonlinear interactions of the primary LC's and, as emphasized in (*1*), the larger scales may grow to dominate the entire pattern of flow. An alternative progression of events may occur through the instability recognized by Craik (*8*) and amplified by Leibovich (*9*).

Since the mechanism leading to the generation of LC's has not previously been well understood or well demonstrated, there has been a tendency for casual observers of the problem to equate LC's with thermal convective rolls, cloud bands, rolls in the laminar sublayer of turbulent boundary layers, or other mechanisms known to produce two-dimensional structures. It is now clear, however, that the interaction of the waves and the wind produces a distinctly different mechanism for the formation of longitudinal rolls, one that cannot be ignored by any serious student of the mixed layers of lakes and oceans.

ALAN J. FALLER

Institute for Physical Science and Technology, University of Maryland, College Park 20742

References and Notes

1. A. J. Faller and E. A. Caponi, *J. Geophys. Res.*, in press.
2. I. Langmuir, *Science* **87**, 119 (1938).
3. A. H. Woodcock, *J. Mar. Res.* **5**, 196 (1944); *ibid.* **9**, 77 (1950); A. H. Sutcliffe, E. R. Baylor, D. W. Menzel, *Deep-Sea Res.* **10**, 233 (1963); D. L. Johnson and P. L. Richardson, *J. Exp. Mar. Biol. Ecol.* **28**, 255 (1977).
4. See A. J. Faller, *Annu. Rev. Ecol. Syst.* **2**, 201 (1971); R. T. Pollard, *Deep-Sea Res.* (Sir George Deacon Aniversary Supplement) (1976).
5. A. D. D. Craik and S. Leibovich, *J. Fluid Mech.* **73**, 401 (1976).
6. S. Leibovich, *ibid.* **79**, 715 (1977).
7. _____ and K. Radhakrishnan, *ibid.* **80**, 481 (1977).
8. A. D. D. Craik, *ibid.* **81**, 200 (1977).
9. S. Leibovich, *ibid.* **82**, 561 (1977).
10. A. J. Faller, Technical Note BN-864, Institute for Physical Science and Technology, University of Maryland, College Park (1978).
11. Sincere appreciation is extended to R. Carlwright for his participation in this study, to S. Daugaard for his careful measurements of wind speed, and to the National Science Foundation for its support under grant ATM 76-82061.

9 March 1978; revised 8 May 1978

Left Ventricular Receptors Inhibit Brain Serotonin Neurons During Coronary Artery Occlusion

Abstract. *Acute coronary artery ligation in pargyline-treated rats decreased serotonin and increased 5-hydroxyindoleacetic acid in the medulla and posterior hypothalamus. Lidocaine applied topically to the left ventricle completely prevented these alterations. No changes in serotonin were observed in the other brain regions examined. These data suggest a reflex inhibition of bulbar and hypothalamic serotonergic nerves by left ventricular receptors following acute coronary artery occlusion in the rat.*

Experimental coronary artery occlusion may result in cardio-cardiac reflex alterations in autonomic nerve traffic (*1–4*). Afferent signals may arise in either mechano- or chemoreceptors (*4–7*) in the left ventricular myocardium and travel through vagal fibers (*1, 5*) or sympathetic afferents, or both (*3, 4*), to the central nervous system. Neural integration has been reported at both spinal (*3*) and supraspinal levels (*1, 7*). The resultant alterations in efferent autonomic nerve traffic are important both as determinants of hemodynamic state (*1–3, 8*) and as contributors to the enhanced vulnerability to lethal arrhythmias (*9*) accompanying acute myocardial infarction. The identification of neurotransmitters that participate in these reflexes is of therapeutic importance.

Nerve fibers, containing the neurotransmitter serotonin, have been demonstrated in brain regions (*10, 11*) believed to be important in cardiovascular control (*12*). Recent studies have supported a role for central serotonergic neurons in blood pressure regulation (*13*) and in the regulation of cardiac autonomic tone (*14*). The present studies were performed to determine whether central serotonergic neurons participate in the reflex alterations in neural activity associated with acute myocardial ischemia.

Three groups of six rats were exam-

Table 1. Serotonin concentration in rat brain regions. Results are expressed as mean ± standard error.

Region	Control (μg/g)	Ligated (μg/g)	Lidocaine-ligated (μg/g)
Thoracic cord	0.96 ± 0.03	1.02 ± 0.02	1.00 ± 0.04
Medulla	2.77 ± 0.13	2.44 ± 0.07*	2.90 ± 0.03
Pons-midbrain	2.69 ± 0.02	2.57 ± 0.13	2.69 ± 0.03
Posterior hypothalamus	2.92 ± 0.04	2.64 ± 0.06†	3.16 ± 0.10
Anterior hypothalamus	2.88 ± 0.11	3.06 ± 0.06	2.99 ± 0.11
Thalamus	2.14 ± 0.10	2.24 ± 0.05	2.21 ± 0.06
Cerebellum	0.49 ± 0.01	0.50 ± 0.01	0.50 ± 0.01

*Different from control at $P < .05$ and from lidocaine-ligated at $P < .001$. †Different from control at $P < .005$ and from lidocaine-ligated at $P < .001$ (Student's *t*-test was used).