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THE BRAIN AS A COMPUTER
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Outlines the principles of "cybernetics" and relates them to what we know of behavior, both from the point of view of experimental psychology and neurophysiology. 413 pp., 71 illus., 1962—$9.00

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Thomas F. Jones, head of the electrical engineering school at Purdue University, has been named president of South Carolina University. He succeeds emeritus president Robert L. Sumwalt, who retired.

Howard E. Mitchell, of the University of Pennsylvania’s department of psychiatry, has been appointed director of a nationwide project to study the number and distribution, roles, and functions of psychiatric aides in state and county mental hospitals. The project is being conducted by the National Association for Mental Health under contract with the National Institute of Mental Health.

James Wei, of Socony Mobil Oil Company’s central research division, will serve as visiting associate professor of chemical engineering at Princeton University during the 1962-63 academic year.

Marion F. Brink, research biologist with the U.S. Naval Radiological Defense Laboratory, San Francisco, has joined the staff of the National Dairy Council, Chicago, as associate director of the department of nutrition research.

Ronald L. Martin, of Technical Research Group, Inc., Long Island, N.Y., has been appointed associate director of the particle accelerator division at Argonne National Laboratory (Ill.).

Gerrit Bevelander, former professor of histology at New York University, has been appointed professor and chairman of the department of histology at the University of Texas Dental Branch, Houston.

Marvin Mann, head of the New York Shipbuilding Corporation’s test program for the N.S. Savannah, has been appointed assistant to the director of regulation for the U.S. Atomic Energy Commission.

John R. Smith, associate professor of medicine at Washington University School of Medicine, is serving a 1-year term as visiting professor of physiology at the Universidad del Valle, Cali, Colombia.

Robert A. Jones, of Rutgers University, has been appointed associate professor and chairman of the department of psychology at Seton Hall University, South Orange, N.J.

Riojun Kinosita has retired as chairman of the department of experimental pathology at the City of Hope Medical Center, Duarte, Calif., to devote full time to research. Alfred G. Kaudson, Jr., former chairman of the department of pediatrics, will head the newly formed department of biology, which consolidates the departments of experimental pathology, genetics, and the section of virology of the department of pediatrics.

Frank Moya, assistant professor at Columbia University’s College of Physicians and Surgeons, has been named professor and chairman of the department of anesthesiology at the University of Miami School of Medicine. He succeeds acting chairman Andrew R. Piergeorge.

Jerome M. Glassman, former head of the department of pharmacodynamics at Wyeth Laboratories, Inc., has been appointed director of the pharmacological laboratories at U.S. Vitamin and Pharmaceutical Corporation, Yonkers, N.Y.

Max Flink, recently appointed research professor of psychiatry at Washington University, St. Louis, Mo., has been named the first director of the Missouri Institute of Psychiatry, St. Louis.

Herbert Friedman, recipient of the first $5000 Navy distinguished scientific achievement award presented in June of this year, has been granted an additional $5000 after further review of the accomplishments on which the original prize was based. Friedman, who is superintendent of the Naval Research Laboratory’s atmosphere and astrophysics division, is credited with numerous discoveries and achievements in upper air research.

Robert E. Ascheman, assistant instructor and research assistant in the department of agronomy at Ohio State University, has been appointed senior plant physiologist at Eli Lilly and Company’s Greenfield (Ind.) Laboratories.

John D. Tallant, physicist at the U.S. Department of Agriculture’s Southern Regional Research Laboratory, New Orleans, has received a Fulbright lectureship to serve as professor of textile physics at the University of Barcelona (Spain) during the current academic year.

Newlin F. Paxson, professor and chairman of the department of obstetrics and gynecology at Hahnemann Medical College, Philadelphia, has retired as emeritus professor. He plans to continue as an active member of the medical staff.

Recent Deaths

Jan van der Bilt, 86; lector-emeritus and astronomer at the Utrecht Observatory, Netherlands; 21 Sept.

Godfrey L. Cabot, 101; chemist and founder of the Cabot Corporation; 2 Nov.

Robert T. Crane, 82; a founder and former executive director of the Social Science Research Council, New York; 23 Oct.

Emery M. Emmert, 62; professor of horticulture at the University of Kentucky; 6 Oct.

Carl M. Epstein, 50; director of adult psychotherapy service at the Menninger Foundation, Topeka, Kan.; 13 Oct.

Charles G. Evensen, 35; professor of geology at Arizona State University; 21 Oct.

Arild E. Hansen, 63; director of research at the Bruce Lyon Memorial Research Laboratory, Children’s Hospital of the East Bay, Oakland, Calif.; 16 Oct.

Harriet Harvey, 42; associate professor of zoology at the University of Oklahoma; 18 Sept.

Earl H. Herrick, 59; professor of endocrinology at Kansas State University; 30 Oct.

John W. Kerr, 90; retired assistant surgeon general of the U.S. Public Health Service, division of scientific research; 27 Oct.

Eger V. Murphree, 63; president of Esso Research & Engineering Company, a vice president of Standard Oil of New Jersey, and general chairman of the 1960 AAAS annual meeting; 29 Oct.

Daphne P. Stamatis, 46; physician and cancer researcher for the National Institutes of Health and former head of the Cancer Institute at the National Medical College, Athens, Greece; 31 Oct.

Lloyd S. Tenny, 85; bacteriologist and former chief of the U.S. Department of Agriculture’s Bureau of Agricultural Economics; 2 Nov.

Richard E. Trees, 42; physicist at the National Bureau of Standards; 27 Oct.
bility to pulmonary infections is due to abnormal respiratory mucus secretion, which is hereditary.

Crow has reiterated his concept of "genetic load". Several of his other statements need clarification for the reader: (p. 59) "The quantity $W_i - W_i^*$ is called the average excess of the allele $A_i$." The average excess is defined by Fisher (1930, 1941) for two alleles as $W_i - W_i^*$ in the present notation. For multiple alleles, the excess concept proves inconvenient and is replaced by the deviation from general mean, namely, $W_i - W$. (p. 61) "An equilibrium may be unstable as well as stable." He means unstable or stable. (p. 65) "Persons who are very tall or very short have lower viability than persons of intermediate height." No supporting reference has been given, but Crow called my attention to an article entitled "Build and blood pressure study", (1959, Society of Actuaries). (p. 219) "... what is lethal now was not lethal a few generations ago." Apparently he said exactly the opposite of what he meant.

In discussing radiation effects on mice, Gowen presented data to show that radiation, up to a certain amount, has decreased the fertility of females but increased their longevity. Is this a beneficial or detrimental effect? Gowen's answer is simply: "This result will be considered favorable by some but not by others" (p. 193).

In discussing blood group and disease problems, Buckwalter says: "The findings did not indicate differences in frequencies of blood type between the patients and their controls of statistical significance in some instances. However, when the results are pooled, they indicate associations of statistical significance..." This seems a favorite target of attack for Wiener. Is the pooling justified? How is it to be done? For a symposium on methodology, these questions are more important than the associations claimed to be found.

In reading the reports and checking the references, I find the most annoying feature is the collectivized references. The only possible argument for this arrangement is that it avoids the duplication of references. This may lead to a very small saving of space, but it requires a tremendous amount of editorial labor which could be used more profitably. My objection to pooled bibliography, however, is more fundamental. The literature cited is an integral part of the paper and should not be broken and intermingled with other unrelated references. One may argue that some other symposium volumes also have pooled bibliography, but that is no justification. Many others do not.

I enjoyed the meetings but I enjoyed reading the reports even more.

C. C. Li
Graduate School of Public Health
University of Pittsburgh

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perivascular collagen fibers. Zucker, referring to the studies of Hugues, reported that when purified collagen fibers were added to citrated platelet-rich plasma, platelet aggregates rapidly appeared along the surfaces of the fibers. In this case platelet serotonin and factor 3 were not released, but "fusion" of the platelet aggregates occurred. Collagen fibers induce only weak agglutination of washed platelets. It is not known whether a cation is necessary for these reactions. Polybrene, a synthetic polysaccharide macromolecule, closely resembles collagen fibers in its action on platelets.

Endotoxins. McKay and Des Prez each described the platelet agglutinating activity of bacterial endotoxin. These two investigators used different endotoxin preparations. The induction of platelet agglutination by endotoxins is well illustrated by the generalized Shwartzman reaction, in which the intravenous injection of endotoxin into a previously sensitized recipient produces white thrombi primarily in the lungs, liver, and spleen but not in the kidney. If a second injection of endotoxin is given, thrombi are then formed in the kidney. McKay reported that injection of endotoxin was followed by a fall in platelet, leukocyte, and fibrinogen levels and a decrease in whole-blood clotting time. Apparently endotoxin in some way initiates in vivo thrombin production, with subsequent agglutination of platelets and thrombus formation. Special comment was made concerning the work of Lee and others, who have shown that if the sensitized recipient's reticuloendothelial system is first blocked by injection of denatured albumin, Thorotrast, or cortisone, the first injection of endotoxin produces thrombi in the kidney as well as in the lung, liver, and spleen. This was interpreted by various participants at the conference to mean that the reticuloendothelial system may play an important role in the prevention of intra-vascular thrombosis.

How does endotoxin initiate thrombus formation? But there was no agreement on this question, but the possibility that the induction of platelet agglutination by endotoxin is mediated through thrombin formation was discussed. Des Prez showed that the addition of endotoxin alone to platelet-rich plasma produces platelet agglutination with subsequent "fusion," and release of platelet serotonin and factor 3. The same changes in platelet-rich plasma can be initiated by the ad-
dition of endotoxin-antibody complexes. A better understanding of the mechanism of action of endotoxin may be attained through future tests with plasmas deficient in antibodies to endotoxin. It appears that platelets, or some other source of thromboplastin, are necessary for the coagulant action of endotoxin. However, a direct action of endotoxin on platelets was not demonstrated. The importance of understanding these problems is apparent, since syndromes similar to the generalized Shwartzman reaction can be induced by vitamin E deficiency in pregnancy, by certain diets, or by injection of such diverse agents as tissue thromboplastin, placenta, trypsin, some snake venoms, and various types of organic and inorganic particulate matter.

Prevention of Thrombosis

The prevention of thrombosis has long been the dream of clinician and research worker alike. There is ample proof that coumarin derivatives lower the plasma levels of prothrombin and certain other procoagulants. Heparin not only inhibits the generation of thrombin but blocks the action of thrombin on fibrinogen. Recently, numerous questions concerning the efficacy of coumarin and heparin therapy in the prevention of thrombosis have prompted investigators to study the effects of these agents on platelet agglutination. Both Berman and Borchgrevink reported that neither coumarin agents nor heparin, in the usual therapeutic dose, prevent the formation of hemostatic thrombi. On the other hand, Mustard reported that heparin, but not coumarin derivatives, prevents the formation of thrombi in extracorporeal shunts.

Mason and Brinkhouse reported that coumarin derivatives, when given in doses within the usual clinical dose range, do not abolish the in vitro generation of platelet agglutinating activity of plasma. Such hypoprothrombinemic plasma can still generate sufficient thrombin to induce rapid platelet agglutination. On the other hand, heparin over a wide range of concentrations not only inhibited the generation of thrombin in plasma but also blocked the agglutination of platelets by preformed thrombin. Zucker reported that heparin has little effect on the agglutination of platelets induced by collagen fibers. Perhaps a more thorough understanding of the sequence of reactions

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leading to platelet agglutination and thrombus formation will permit the development of new anticoagulants. It was suggested that anticoagulants might be found with sufficient specificity of action to inhibit pathologic platelet agglutination but leave physiologic agglutination mechanisms intact.

There was one final subject of discussion: What causes platelets to adhere to one another when agglutination occurs? There are indications that several of the agents known to induce platelet agglutination may mediate their action through thrombin. The action of other agents, such as ADP, thrombocyte agglutinating factor, and collagen fibers, cannot at present be explained in this manner. Indeed, it has been suggested that thrombin merely causes the release of ADP from platelets and that only then does platelet agglutination occur. Waugh proposed several possible mechanisms or models for platelet agglutination. In one model a platelet agglutinating agent such as thrombin would act to produce hiatuses in the platelet membrane by removing a single molecular species. This would render the membrane unstable, and molecules from the interior of the platelet would be attracted to these hiatuses. These molecules, since they differ from the molecular species originally present, would be unable to stabilize the membrane and would simply continue to congregate at the membrane surface. Eventually this process would lead to the formation of pseudopods, one of the earliest morphologic changes observed in the agglutination process. In another model a platelet agglutinating agent would act upon the platelet membrane either to remove or to rearrange structurally certain molecules. In this manner new electrical charges would be uncovered which could participate in binding platelets together. It is conceivable that these two processes operate simultaneously. Other molecules, released perhaps from the interior of the platelet, along with cations from the plasma, could form the bridges which would link adjacent membranes together. At present all of this is only educated speculation, but herein lies the challenge.

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Available in temperature ranges from 500°F, maximum to 1000°F. Maximum. Saf-T-Bilt Ovens provide precision heat control accuracy and maximum safety from explosion. Built for use with liquids, gases and volatile solids with explosion relief panel and forced exhaust system to dissipate volatile substances from work chamber. Special vapor tight conduit fittings and explosion proof housings for controls and contactors are provided to eliminate open arc. Heater units are remote from work chamber and insulated by use of diffuser plate.

Write today for bulletin 203-1 for complete information on Saf-T-Bilt Ovens.

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**SCIENTIFIC APPARATUS**


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