form a natural tribute to the honor of Benjamin Franklin.

The material in the book in the main is objective and is chosen from the highlights of history. The settled accomplishments of the human race form easy stepping-stones for the lay reader to reach a fascinating picture of electricity as a part of the world. A museum of electricity outlined from this book would be a grand institution. Not only are abstruse and difficult mathematical derivations omitted, but there are also a minimum of mathematical relationships. Likewise worthy attempts to correlate electrical forces with gravitational forces are not considered.

The first part of the book is designated as "Sparks" or the "Beginnings of Electricity." The illustrations are numerous and in keeping with the writing. Particularly the illustration of "Queen Elizabeth watching Gilbert's Experiments" is appropriate to an anecdotal account. Perhaps the explanation of the electrophorus would have been improved if attention had been given to the fact that Volta devised it as a voltage multiplier which enabled him to establish the relative values of the different metals in the Volta series. Also the reproduction of the scene where Volta was showing the Voltaic Pile to Napoleon Bonaparte would fit. An artist's conception of Madame Galvani discovering the twitching of frog legs when an electrical discharge took place in her kitchen would dramatically mark the advent of the first galvanometer. In the reference to the "attracted disk electrometer" of William Snow Harris and later by Lord Kelvin one may make the valuable implication that the advance in the knowledge of electricity is contingent upon instruments of measurement.

The second part of the book is under the caption, "Lightning," in which simple experiments by Franklin and others lay solid foundation for the science of electricity. Franklin's inspiration for this science was incited at the age of forty when he saw some experiments by Dr. Spence. Within a year he had well stated the theory of positive and negative electricity. Within a few years he experimented in every practicable manner. His procedure was simple and direct, and his interpretation has stood well. He showed that lightning was an electric discharge, and he gave the world an electrostatic motor, the lightning rod, the electric chime and the basis for the "inverse square law" from the "ice pail experiment" as interpreted by Joseph Priestley. Besides the author presents the versatile Franklin as making contributions to book-printing, flying machines, optics, eye-glasses, chemistry, geology, submarine boats, whirlwinds, stoves, street cleaning, musical instruments, aerial warfare, etc. In reading of Franklin's discussion of science before royalty and the greatest characters of the time, it would seem that as a salesman for science he is a model.

The author quotes Franklin's philosophic attitude, "nor is it of much importance to us to know the manner in which nature executes her laws. It is enough if we know the laws themselves. It is of real use to know that china (ware) left in the air unsupported will fall and break; but how it falls and why it breaks are matters of speculation. It is a pleasure indeed to know them, but we can preserve our china without it. Thus in the present case, to know this power of points (to discharge electricity) may possibly be of use to mankind, though we should never be able to explain it."

The reviewer would like to have seen reproductions of the early precursors of the Vande Graaff electrostatic generator such as were made by Sir William Thomson, Righi or Gray.

The third part of the book treats of modern electrical science up to and including cosmic rays. Maxwell's work is stated necessarily briefly, but the lay reader would hardly suspect him as the greatest intellect of all, whose work guided perhaps more research than any other man. Perhaps if Maxwell's electromagnetic model had been illustrated and explained, the reader might glimpse the great man's plight and vision. Nevertheless, this last part is an excellent short survey and should encourage many to learn more.

A commanding pageant, "Electricity," could be enacted in a three-hour period by selecting characters, apparatus and wording from Dr. Miller's book. Perhaps it would be bold for an ordinary reviewer to suggest that Dr. Miller could direct the staging of a film encompassing the outline and spirit of this book. By such works as these, other men may become inspired by science as Franklin was at forty.

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SPECIAL ARTICLES

AN ANALYSIS OF SKIN PIGMENT CHANGES AFTER EXPOSURE TO SUNLIGHT

WITH the use of the Hardy recording spectrophotometer and the methods set forth in an earlier paper,[1] we have been able to determine objectively the pigmentary changes in the skin after irradiation by sunlight. Here we demonstrate the events following a single one-hour exposure to the mid-day sun (in August, 1938).

[1] E. A. Edwards and S. Q. Duntley, *Am. Jour. Anat.*, 65: 1, 1939.

A normal male adult served as the subject, and the sacral region was used for the analysis. This area had not previously been exposed to direct sunlight for two years, and it was kept from further exposure after the start of the experiment. Readings were made with diminishing frequency for a total of nine and a half months. The changes following exposure are illustrated by the selected spectrophotometric curves here reproduced (Fig. 1).

The curve made before the exposure (upper curve, Fig. 1) is typical of the sacral region. The blood here is quite venous, as shown by the bluntness of the curve between 500 and 600 millimicrons. Carotene is evidenced by the extremely shallow depression or absorption band at 482 millimicrons, and melanin is moderate in amount as shown by the general upward slope from 400 (violet end) to 700 millimicrons (red end).

Hyperemia was the first effect of the radiation. It was marked in two hours, and maximum in eleven hours. The curve at this time (Fig. 1, lower curve) shows a great lowering in reflectance with pronounced oxyhemoglobin absorption bands, characteristic of an active arterial blood flow (hyperemia). The regression of the hyperemia was rapid in the next few days, and the local blood volume diminished, although it remained distinctly higher than normal. Moreover, there was a shift from oxyhemoglobin to reduced hemoglobin, indicating blood stagnation. This shift could be detected at four days, and is striking in the nineteen-day curve shown in Fig. 1. This curve indicates the gradually decreasing blood volume by the increased reflectance in the region of the twin bands of oxyhemoglobin, while the reduced arterialization is evidenced by the marked blunting of these bands. The venous trend is even more strikingly shown by the fact that despite a diminished blood volume, the nineteen-day curve passes below the eleven-hour curve in the red region of the spectrum (600 to 700 millimicrons). Reference to published curves of reduced and oxyhemoglobin[1] shows that this crossing indicates an increased venous blood content.

During the succeeding months the blood volume slowly receded but did not reach the pre-irradiation level. The shift to a more venous blood continued for four and a half months, after which the blood became fairly constant in its proportions of oxy- to reduced hemoglobin. Thus, while the hyperemia was an acute reaction, the blood stagnation persisted and was still detectable at nine and a half months. This illustrates that solar radiation exerts a long-lasting effect on the blood vessels, a fact previously mentioned by Finsen.[2]

Since melanin gives no sharply demarcated bands in the visible spectrum, but rather imparts a general

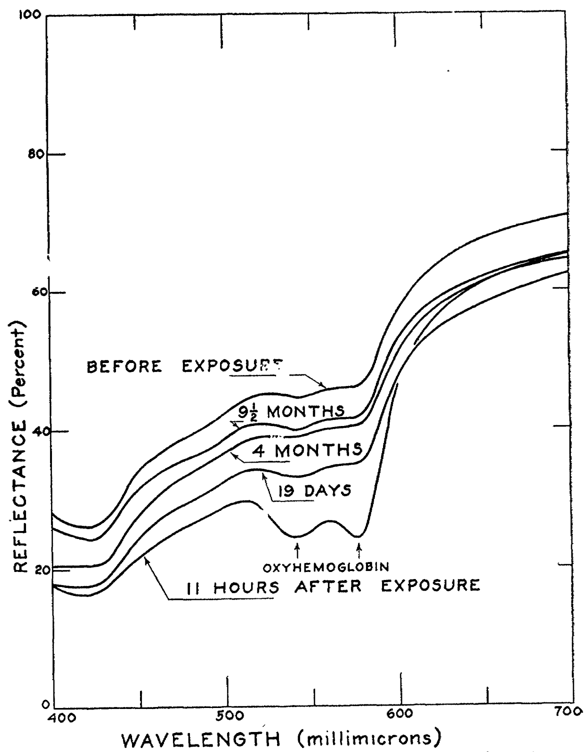

FIG. 1. Spectrophotometric curves of the skin after a single exposure to sunlight. Hyperemia (oxyhemoglobin) is maximal at 11 hours, melanin at 19 days, and melanoid at four months. Blood stagnation, as registered by reduced hemoglobin, persists from the time of the early disappearance of hyperemia for over nine months.

slope to the curve, an increase in this material will be indicated by a depression of the violet end of the spectrum (400 millimicrons). The concomitant effect of the changes in the other pigments must, however, be kept in mind in drawing deductions as to the amount of melanin present. An increase in melanin was apparent in two days, increasing to a maximum on the nineteenth day. The rate of formation of melanin was slowed after this time, and at one month its total quantity also began to diminish. The nine and a half month curve shows the melanin to have returned to approximately the same level as before exposure.

We have previously pointed out that the degeneration of melanin produces an allied pigment which we have named melanoid.[1] The presence of this pigment is shown by a flattening of the spectrophotometric curve near 400 millimicrons. This flattening can be seen in the curve at nineteen days and is maximum at four months, but one fails to find it at nine and a half months. Thus it is seen that the initial appearance of melanoid depends on the formation of considerable melanin. Melanoid increases for some time after the production of new melanin diminishes. Finally, with the disappearance of most of its mother substance, the melanoid ultimately fails to be recognizable.

[2] N. R. Finsen, *Meddelelser fra Finsens Medicinske Lysinstitut,* 1: 6, 1899.

While carotene showed some fluctuation from time to time, there were no changes in this pigment which could be definitely related to the irradiation.

From the purely physical data represented by spectrophotometric curves, the psychophysical specification of the color stimulus received by the eye of a normal observer was computed[3] for I. C. I. Illuminant C using data standardized by the International Commission on Illumination in 1931.

	Relative brightness	Dominant wave-length	Excitation purity
	Per cent.	mμ	Per cent.
Before exposure	48.6	580	20.2
11 hours after exposure	31.8	590	20.5
48 hours after exposure	31.8	592	20.9
4 days after exposure	36.9	587	20.7
19 days after exposure	37.4	585	20.7
4 months after exposure	42.6	580	20.5
9½ months after exposure	43.6	583	20.1

In this color language "relative brightness" is a measure of the quantity of light reflected by the skin, while "dominant wave-length" and "purity" describe the quality of this light. Thus, hyperemia caused the skin to be much darker and noticeably more red, but it produced only a very slightly more pure or saturated color. At the end of four days the skin was less red but still much darker than normal. This is ascribable not so much to melanin as to the venous stagnation previously referred to. After four months the color of the skin had returned to its original quality, but its brightness was reduced by melanin. The gradual recession of this substance is shown by the increased brightness of the nine and a half months' data. Even at this time the skin was darker than before exposure.

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PRESENCE OF A LEUKOCYTOSIS-PROMOTING FACTOR IN INFLAMMATORY EXUDATES[1]

THE recent studies of the writer have indicated the presence of a crystalline nitrogeneous substance liberated in areas of acute inflammation, capable *per se* of increasing capillary permeability and of inducing prompt leukocytic migration at the site of injury.[2, 3, 4] This substance has been termed leukotaxine. Its liberation and recovery from injured tissue offer a reasonable explanation for two of the basic initial sequences in the development of the acute inflammatory reaction. Leukotaxine has been shown to have no manifest physiological property in common with histamine.[2, 4, 5]

Leukotaxine, injected either subcutaneously or intravenously, fails to induce an increase in the leukocytic level of the circulation in both the dog and the rabbit. Repeated injections of 30 to 50 milligrams of the material over an interval of several days leave the number of circulating leukocytes essentially unaltered. The independence of the chemotactic factor (leukotaxine) from that concerned with leukocytosis is not wholly surprising when it is recalled that certain inflammatory processes characterized by marked leukocytic infiltration can even be accompanied by distinct leukopenia.

Having apparently eliminated leukotaxine as the factor responsible for the state of leukocytosis accompanying inflammation, studies were undertaken in an endeavor to determine whether the active principle might not be liberated in the exudate as a result of tissue injury. Ponder and MacLeod[6] recently expressed the opinion that in the blood stream of rabbits with peritonitis the shift to the left of polymorphonuclear counts is probably referable to the absorption of breakdown products of the cells appearing first in the exudate.

Inflammatory exudates were obtained by a variety of methods. In the majority of instances the exudative material resulted from an intrapleural injection of 1.5 cc of turpentine in dogs. Several of the exudates studied, however, were derived either after the intrapleural injection of 0.1 cc 10 per cent. croton oil in olive oil, the combined irritating action of several substances (*e.g.*, magnesium carbonate, staphylococcus *aureus* toxin and a mixture of aleuronat and colibactragen), or finally by physical injury. The latter consisted in recovering exudative material from a severe burn induced under nembutal anesthesia by scalding the limb of a dog in water heated to about 90° C. The exudate was obtained by removing the edematous subcutaneous tissue, and gently expressing the oozing and abundant exudative fluid content.

From 15 to about 25 cc of whole or cell-free exudate was injected by intracardiac puncture into the circulating blood stream of a normal dog. The results of eighteen such experiments indicate that there is a conspicuous rise in the leukocytic counts several hours after the introduction of an exudate into the blood. The increase in the number of circulating leukocytes averages 70 per cent.

[3] A. C. Hardy, "Handbook of Colorimetry," Technology Press, Cambridge, 1936.

[1] Aided by grants from the Milton Fund of Harvard University, from the International Cancer Foundation and from the Academy of Arts and Sciences.

[2] V. Menkin, *Jour. Exp. Med.*, 64: 485, 1936.

[3] *Ibid.*, *Jour. Exp. Med.*, 67: 129, 1938.

[4] *Ibid.*, *Physiol. Rev.*, 18: 366, 1938; *Proc. Soc. Exp. Biol. and Med.*, 40: 103, 1939.

[5] V. Menkin and M. A. Kadish, *Am. Jour. Physiol.*, 124: 524, 1938.

[6] E. Ponder and J. MacLeod, *Jour. Exp. Med.*, 67: 839, 1938.