pretectal region on each side of the posterior commissure. This was done in cats with the aid of the stereotaxic instrument. In these cats both pupils remained for weeks widely dilated and entirely unresponsive to light. That the sphincter nucleus of the third nerve was not damaged was shown by the fact that pupillary constriction still accompanied winking, the well-known lid closure reflex.

Four of these cats with complete loss of the pupillary light reflex are being allowed to live for a longer period of time in order to make sure that the loss is permanent. Three others were sacrificed after periods ranging from 3 to 7 weeks. Microscopical sections of the brains of these 3 cats showed that the lesions had been accurately placed on both sides of the posterior commissure in the pretectal region and that except for a small area near the midline in the most rostral part of the tectum the superior colliculi were intact. The superior quadrigeminal brachia, the lateral geniculate bodies and optic tracts had not been damaged.

These experiments confirm the conclusions drawn from the stimulation experiments that the pupillary light reflex is mediated through the pretectal region and not through the superior colliculus. The slight amount of damage to the rostral part of the superior colliculus we regard as inconsequential. But in order to remove any possible objection on this ground other experiments are in progress in which the effort is being made to abolish the pupillary reflex with still smaller lesions which do not in any way involve the superior colliculi.

## 7051 C

## Changes in the T Wave of the Electrocardiogram in Acute Rheumatic Fever.*

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Transitory flattening or inversion of the T wave of the electrocardiogram has been found during the active stage of rheumatic fever by Rothschild, Sacks and Libman,[1] by Shapiro,[2] and by Master.[3]

* Aided by the Frederick K. Babson Fund of the Michael Reese Hospital.

[1] Rothschild, M. A., Sacks, B., and Libman, E., *Am. Heart J.*, 1927, **2**, 356.

[2] Shapiro, M. J., *Am. Heart J.*, 1930, **5**, 504.

[3] Master, A. M., *Am. J. Med. Sci.*, 1931, **181**, 211.

However, Schwartz and Weiss[4] have claimed that the T wave of children with acute rheumatic fever does not differ from the T wave of the normal child. In view of this difference of opinion, it was decided to reinvestigate this subject.

Electrocardiograms of children suffering from rheumatic fever were selected for this study from the 8300 records in the electrocardiographic files of the Heart Station of Michael Reese Hospital. From these were selected those in which 2 or more electrocardiograms had been taken. The clinical charts of these patients were studied, and 12 cases, with electrocardiograms in both the active and inactive periods of the disease, were selected. The rheumatic fever was assumed to be active when a majority of the following signs and symptoms was found: rapid heart rate, pyrexia, "sore throat", joint pains, acute pericarditis and an elevated white blood count.

The electrocardiographic changes found in these cases confirm the observations of Cohn and Swift,[5] Rothschild, Sacks and Libman,[1] Shapiro,[2] and Master,[3] *i. e.*, the P wave was often altered in size, shape and direction; the P-R interval was often lengthened; the QRS complex was often notched, lengthened, or changed in ampli-

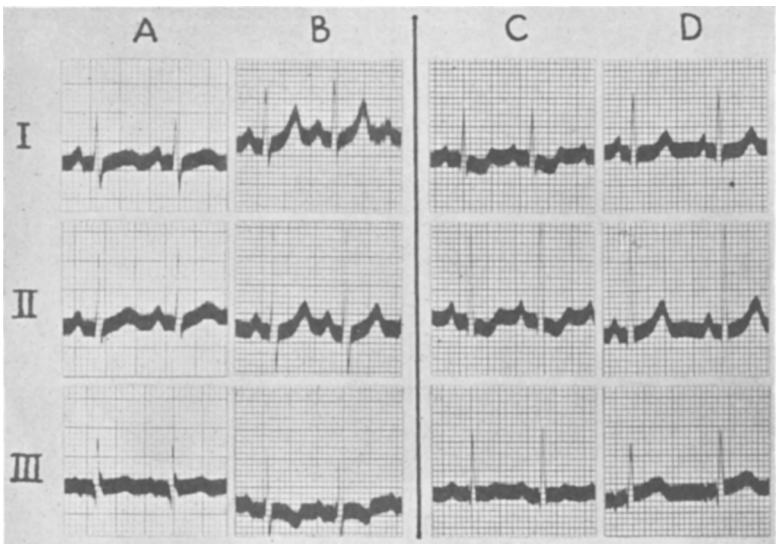


FIG. 1.

Segments of the electrocardiogram, standard 3 leads, taken during the active and inactive stages of acute rheumatic fever in 2 patients. A, active stage of one patient; B, inactive stage of same patient; C, active stage of a second patient; D, inactive stage of second patient.

[4] Schwartz, S. P., and Weiss, M. M., *Am. J. Dis. Child.*, 1929, **38**, 699.

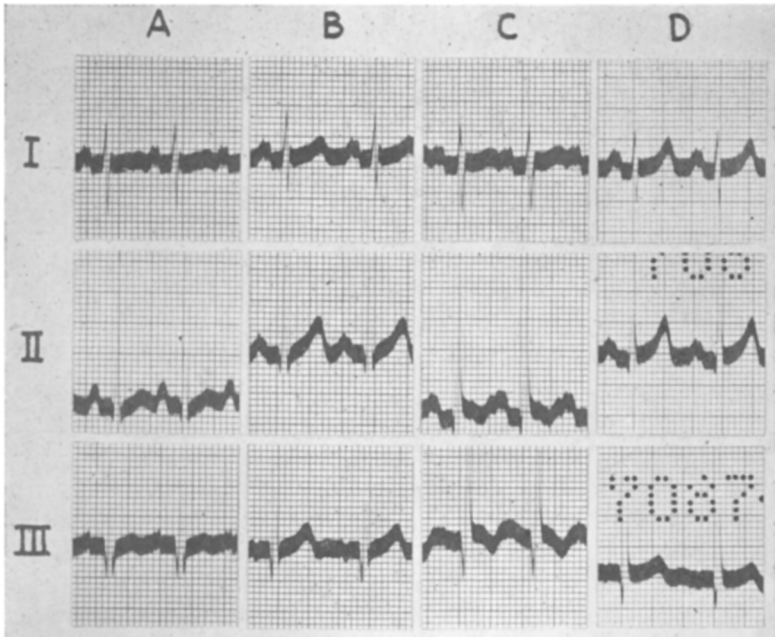


FIG. 2.
Segments of the electrocardiogram, standard 3 leads, taken during the active and inactive stage of acute rheumatic fever. A and C, during 2 active periods; B and D, during 2 inactive periods. Segments arranged in chronological order.

tude or direction; the S-T segment was often anisoelectric or sloping; and the T wave was flattened, diphasic, isoelectric or inverted. With recovery from the active stage of the disease there was a tendency, after a varying period of time, for the electrocardiogram to return towards normal. Typical changes are shown in Figs. 1 and 2.

The T wave during the active phase not only had become small but usually had lost its normal peaked top (Fig. 1, Record A). When the T wave was inverted, it tended to assume the appearance of the coronary T wave (Fig. 2, Record C, Lead III). With recovery from the active stage of rheumatic fever, the T wave tended to become taller and sharply peaked (Figs. 1 and 2. Records B and D). Some cases were followed through successive active episodes and intervening inactive periods, and the same changes were found with each new cycle of the disease (Fig. 2). T wave changes similar to those described in acute rheumatic fever, but more transitory, have been described in pneumonia (Master, Romanoff and Jaffe[6]; Arnett and Harris[7]; Abt and Vinnecour[8]), in typhoid fever (Chagas[9]), and in scarlet fever (Shookhoff and Taran[10]).

[5] Cohn, A. E., and Swift, H. F., *J. Exp. Med.*, 1924, **39**, 1.

Digitalis may produce changes in the S-T segment and T wave similar to those found in acute rheumatic fever (Cohn, Fraser and Jamieson[11]), but digitalis was used in only a few of the cases here reported. The changes are therefore most likely due to the rheumatic infection itself.

*Summary.* A study was made of the electrocardiograms of 12 cases of rheumatic fever in children, taken during both the active and inactive stages of the disease. During the active stages, the T wave was softly rounded and small, sometimes isoelectric, diphasic or inverted, and during recovery the T wave became upright, tall, and sharply peaked.

I am grateful to Dr. Louis N. Katz for his advice and suggestions.

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[6] Master, A. M., Romanoff, A., and Jaffe, H., *Am. Heart J.*, 1931, **6**, 696.

[7] Arnett, J. H., and Harris, S. E., *Med. Clinics of N. America*, 1931, **15**, 503.

[8] Abt, A. F., and Vinnecour, M. I., in press.

[9] Chagas, E., *C. R. de Soc. Biol.*, 1930, **106**, 505.

[10] Shookhoff, C., and Taran, L. M., *Am. J. Dis. Child.*, 1931, **42**, 554.

[11] Cohn, A. E., Fraser, F. R., and Jamieson, R. A., *J. Exp. Med.*, 1915, **21**, 593.