

## Antimyocardial Antibodies in Rheumatic Fever

Hypothesis of autoimmunizing pathogenesis of rheumatism was submitted for the first time by ROBINSON<sup>1</sup> on the ground of pathological alterations obtained by immunizing rabbits with homologous seral protein mixed with streptococcus antigens. In 1945 CAVELTI<sup>2</sup> found antibodies against myocardial tissue in the serum of rheumatics; in 1947<sup>3</sup> he demonstrated genesis of auto-antibodies in rats immunized by homologous antigen with streptococcus antigens. REJHOLEC, VANČURA, and WAGNER<sup>4</sup>, found auto-antibodies against myocardium in a certain percentage of sera of patients in acute stage of nephritis, and REJHOLEC and WAGNER<sup>5</sup>, discovered them regularly in the sera of rheumatics in the active stage of the disease. In patients in the active stage of glomerulonephritis, antirenal auto-antibodies were ascertained also by LANGE and collaborators in 1949<sup>6</sup>.

The present study aims at testing the occurrence of auto-antibodies in patients with diseases in which an immunotoxical (auto-immunizing) mechanism in the pathogenesis may be presumed.

**Material and method.** Preparation of sera: The sera of patients were regularly examined on the day of drawing. Early treatment is of importance for the results. The titres of auto-antibodies weaken in the course of few days.

Antigens: Human kidneys, myocardium and liver taken out at autopsy of persons killed in accidents, only a few hours after death, were, after crushing, extracted with 1.1% NaCl in relation of 1:5. The crushed tissue was eliminated by centrifuging and the supernatant diluted 1:80. Diluted extract was used for sensibilisation of collodion particles.

Preparation of collodion: Collodion (Explosia E22) particles for agglutinating reaction were treated by the Cavelti method. Ready particles were conserved in the dark and never used for longer than three weeks.

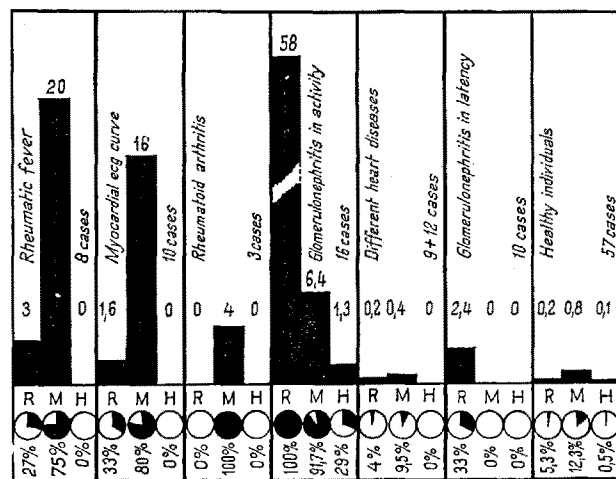
Sensibilization of collodion: Extracts of human organs diluted 1:80 are added to the collodion particles diluted with 1.1% NaCl approximately to the density No. 2 of the MacFarland scale. Incubation time 1 h at chamber temperature.

The test: The treated serum is diluted in geometric progression from 1:4 to 1:256 and 0.5 ml of sensibilized collodion is added into each test-tube containing 0.5 ml of the diluted serum. After shaking, it was incubated for 1 h at chamber temperature. Short centrifuging: speed 1500 rev./min, duration 3 min.

Results are read after mild shaking with the aid of agglutinoscope. Positive results are characterized by larger or smaller granules, easily discernible from homogeneous turbidity of the negative reaction and of the controls. Controls were always made with insensitized collodion and with sera known as negative and positive.

**Results.** 8 cases of rheumatic fever, 10 cases of acute tonsillitis showing the so-called myocardial ecg curve, 3 cases of rheumatoid arthritis and 16 cases of active diffuse glomerulonephritis were examined for the presence of organo-antibodies, and the results were compared with findings in several control groups. The control groups consisted of 9 cases of myocardial infarction,

Average titres and occurrence of antirenal (R), antimyocardial (M), and antihepatic (H) auto-antibodies in rheumatic fever and other diseases in %.



12 cases of inactive valvular diseases, 10 cases of latent chronic glomerulonephritis, and 57 healthy individuals.

Antimyocardial antibodies were found in 75% of the rheumatic fever cases without salicylate treatment with an average titre of 1:20. In 80% of the acute tonsillitis cases showing the myocardial ecg curve, the average titre was 1:16, and in 100% of the rheumatic arthritis cases 1:4. Antirenal antibodies were ascertained only in 33% of all these groups with an average titre of 1:1.5. In 100% of cases of active diffuse glomerulonephritis, the titres were markedly high (average 1:58, maximum 1:128), whilst in the period of latency of the same disease the antirenal antibodies were found in 33% of cases only and showed very low titres (average 1:2.4, maximum 1:8). Antimyocardial antibodies were found in 91.7% of glomerulonephritis cases in active stage, but their average titre was low (1:6.4). No antimyocardial antibodies were found in the stage of latency.

These results are in sharp contrast with those found in the control groups, where the occurrence of antimyocardial antibodies never exceeded 12.3% with the maximum titre of 1:8. Antirenal antibodies were found there in 5.3% of cases, the maximum titre being 1:8. Antihepatic antibodies did not reach any notable value in any of the control groups.

**Discussion.** The occurrence of antimyocardial antibodies in rheumatic fever and several other diseases is therefore strikingly high, in glomerulonephritis rather frequent. This fact seems to support the view that the presence of these antibodies has a similar connection with the immunotoxical pathogenesis of these diseases as has already been demonstrated in glomerulonephritis by the presence of antirenal antibodies. Since the beginning of the salicylate treatment, the antibodies recede or disappear.

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### Zusammenfassung

Kollodiumpartikel wurden mit Extrakten aus menschlichen Organen, wie Leber, Nieren und Myocard, sensibilisiert und damit Sera von Personen getestet, die an Krankheiten mit autoimmunisierenden Vorgängen in der Pathogenese litten. Die Ergebnisse dieser Versuche wurden mit denjenigen von Kontrollsera verglichen.

<sup>1</sup> J. J. ROBINSON, Arch. Pediatr. 61, 6 (1944).

<sup>2</sup> P. A. CAVELTI, Proc. soc. exp. biol. 60, 379 (1945).

<sup>3</sup> P. A. CAVELTI, Arch. Pathol. 44, 1, 27 (1947).

<sup>4</sup> V. REJHOLEC, A. VANČURA, V. WAGNER, and Z. MANDLÍKOVÁ, Sbor. lék. (Praha) 54, 195 (1952).

<sup>5</sup> V. REJHOLEC and V. WAGNER, Sbor. lék. (Praha) 54, 217 (1952).

<sup>6</sup> K. LANGE, A. GOLD, D. WIENER, and V. SIMON, J. Clin. Invest. 28, 1 (1949).