

$p\text{CO}_2$ during Metrazol induced attacks showed that there is a marked and long-lasting increase of $p\text{CO}_2$ (Fig. 2) which must be due to a considerable increase of the production of carbon dioxide during the seizure, during which, furthermore, the cortical blood flow is so much greater than normally.

The results so far obtained show that constant conditions for measurements of cortical $p\text{CO}_2$ may be achieved experimentally and that, in principle, the $p\text{CO}_2$ electrode described, when placed upon the intact cerebral cortex, reacts according to expectations. We have explored the possibility of measuring cortical $p\text{CO}_2$ after removal of the pia, or some of the gray matter. This lead to unstable conditions during which the $p\text{CO}_2$ of the injured surface was observed to increase. Therefore we have chosen surface measurements and we have interpreted the values obtained as representing a mean cortical $p\text{CO}_2$. At present, however, there is no information available as to, e.g. diffusion conditions of carbon dioxide in the brain. Exact knowledge of the capillary anatomy of the cat's cerebral cortex is also lacking. Such information is necessary before a theoretical evaluation of the data obtained can be carried out. A further analysis of the measuring conditions is under way.

D. H. INGVAR, B. SIESJÖ, and C. H. HERTZ

Institute of Physiology and Institute of Physics, University of Lund (Sweden), April 10, 1959.

Zusammenfassung

Der mittlere $p\text{CO}_2$ in der Gehirnrinde von Katzen wurde mittels einer neuen Elektrode kontinuierlich registriert. Es wird ein kurzer Bericht über die Messbedingungen, nebst Beispielen von $p\text{CO}_2$ -Veränderungen bei verschiedenen Funktionszuständen, gegeben.

Mast Cells in Bronchial Connective Tissue of Man

Importance of such Cells in Allergic Tissue Injury

This report concerns researches conducted on broncho-biopsies taken on the orifice of the middle lobe bronchus fixed with formalin Merck 10% and stained with toluidine blue at pH 6.5. Counting of mast cells was made in 20 fields using ocular $\times 10$ and objective $\times 100$.

Normal subjects (5 cases): Mast cells were numerous (45 ± 10): fusiform, circular, oval. Part of them ('normal' cells) showed cytoplasm thronged with violet metachromatic granules and intergranular cytoplasm extremely scarce or not demonstrable (Fig. 1A); the others ('abnormal' cells) had quite different aspect, i.e. light degranulation disruption and scattering of the granules. Proportion of 'normal' cells (NC) to 'abnormal' ones (AC) was always NC:AC > 1 . Sections treated with hyaluronidase (37°C for 18 h) revealed the opposite proportion owing to prevalence of degranulated cells with distinctly visible violet granules surrounded by amorphous metachromatic substance, which appeared dissolved in the cytoplasm thus assuming a purple-red staining (Fig. 1B). Most of the mast cells clearly revealed their nucleus because of the intense degranulation provoked by the enzyme.

Inflammatory injuries (5 cases): The number of mast cells was not far from the average observed in normal

cases. A more marked tendency to degranulation and scattering of the granules in the ground substance of the connective tissue (Fig. 1CD) was ascertained.

Treatment with dexamethasone (5 cases affected with aspecific bronchitis): Corticosteroid (Deltafluorene Leptit) was administered orally 3–4.5 mg/day after preliminary bronchobiopsical examination. Histological control after a treatment of at least 10 days showed a numerical reduction of mast cells (under 15). Identifiable cells presented a reduced diameter degranulation and small violet granules surrounded by amorphous intergranular substance metachromatically very light-red stained. There were also some disrupted cells with granules scattered in the connective tissue and mast cells presenting orthochromatic granules (Fig. 1EF).

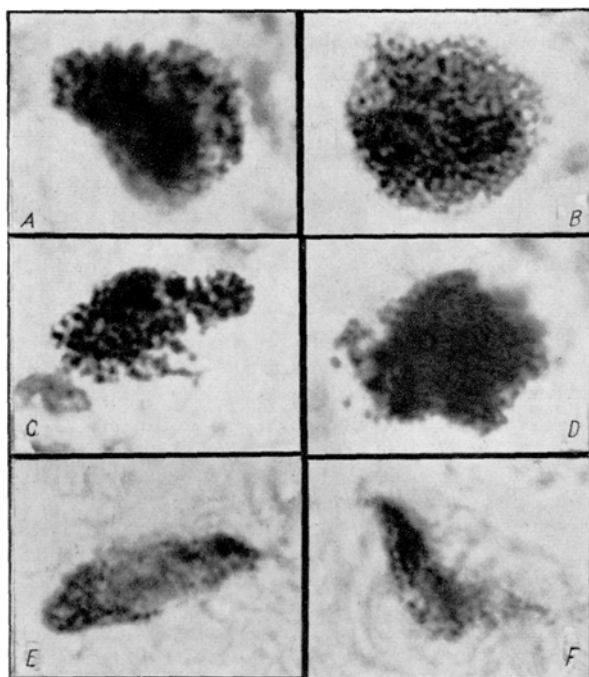


Fig. 1.—(A) Normal mast-cells. (B) Mast-cells after treatment with hyaluronidase. (C) Partial degranulation (bronchitis). (D) Partial disruption (bronchitis). (E–F) Damage following treatment with dexamethasone (Magnification $\times 2000$).

Serial sections of same biopsies incubated for 18 h at 37°C in hyaluronidase showed both a further reduction of granules' diameter and the amorphous intergranular substance dissolved in the cytoplasm thus appearing purple-red stained.

Allergic injuries (12 cases): Biopsies were taken from patients affected with bronchial asthma free of any secondary pathological complications. Counting of mast cells in remission phase of asthma gave a high number (35 ± 5), whereas the result in biopsies taken in full asthma attack was under 10: in several cases a careful examination of many histological sections was necessary before finding a single mast cell. Morphological and metachromatic changes were noticed in all examined cases both in remission phase and in full asthma attack: proportion of 'normal' cells (NC) to 'abnormal' ones (AC) in the former case was always NC:AC < 1 with a still more marked diversion from rule in the latter. The rare mast cells observed in biopsies taken in full asthma attack all exhibited a very intense degranulation and disruption with granules scattered in the ground substance of the connective tissue.

Granules were very reduced in size and violet-stained encircled with a halo of amorphous metachromatic intergranular substance giving the cellular cytoplasm a purple-red hue. The cells had a noticeably smaller diameter and their nucleus was hardly distinguishable inspite of intense degranulation (Fig. 2).

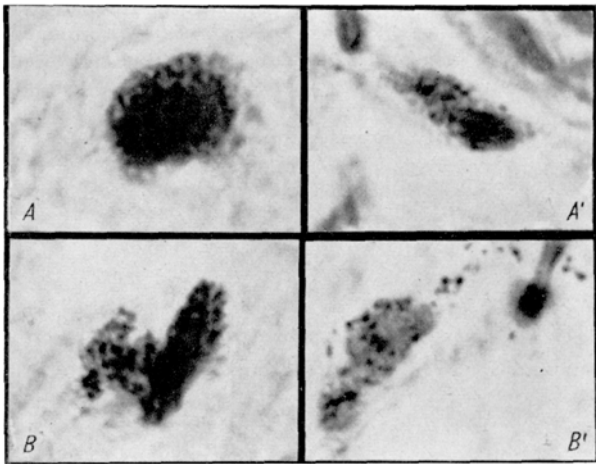


Fig. 2.—(A–B) Mast-cells in remission phase of asthma and (A'–B') in asthma attack (same patients). Magnification × 2000.

Conclusion: Mast cells are numerous in bronchial connective tissue of normal man. Their morphological changes correspond to various functional stages of the cells and the most evident manifestation of these changes is degranulation. In bronchial inflammation this phenomenon appeared increased, though not to so a high degree as in bronchial asthma, in which cellular damages were so evidently correlated with the acuteness of asthma attack that they can be taken as a histopathological token characteristic of this illness. The damages observed were similar to those provoked experimentally with dexamethasone. Although it was not possible to analyse the substances contained in the granules and liberated with degranulation, on the basis of the experimental findings of various authors¹ on animals, the assumption is justified that these substances are histamine, heparin, and hyaluronic acid. This is of undeniable interest considering that these active substances are of fundamental importance in the pathogenesis of hyperergic-patergic tissue reactivity.

G. SALVATO

Department of Medicine, Town Hospital Merano (Italy), April 8, 1959.

Riassunto

I mastociti, assai numerosi nel tessuto connettivo bronchiale umano normale, presentano in questo stesso distretto organico importanti modificazioni numeriche, morfologiche e istochimiche dopo trattamento con desametasone e nel corso di lesioni bronchiali di tipo disergico (asma bronchiale). Le alterazioni costantemente osservate negli asmatici fanno ritenere che esistano strette relazioni tra comportamento dei mastociti tessutali e manifestazioni disreattive dell'albero bronchiale.

¹ F. I. RILEY, Pharmacol. Rev. 7, 267 (1955). – G. B. WEST, Int. Arch. Allergy 13, 336 (1958). – M. HILL, Exper. 13, 395 (1957).

Interarterielle koronare Anastomosen

Es ist bekannt, dass bei stenosierender Koronarsklerose, bei Kammer-Hypertrophie verschiedener Ursache und bei chronischer Anämie in der Mehrzahl der Fälle postmortal interarterielle koronare Anastomosen festgestellt werden können^{1–8}. Über die Häufigkeit derartiger Anastomosen in normalen Herzen sind die Ansichten jedoch geteilt.

Die Häufigkeit interarterieller koronarer Anastomosen bei normalen und bei pathologisch veränderten Herzen

Diagnose	Anzahl	Anastomosen	
		nachweisbar	nicht nachweisbar
Normale Herzen	15	1	14
Stenosierende Koronarsklerose mit/ohne Infarkt oder «Narben»	27	21	6
Infarkt oder «Narben» ohne stenosierende Koronarsklerose	18	14	4
Kammer-Hypertrophie bei Klappenfehler oder arterieller Hypertension	13	6	7

Während einige Untersucher auch bei normalen Herzen in der Mehrzahl der Fälle Anastomosen nachzuweisen imstande waren^{3,7,9–11}, haben andere Autoren Anastomosen in weniger als 10% der Fälle finden können^{1,4,5,12}. Die Bedeutung dieser Anastomosen für die Prognose der Herzkrankheit bei stenosierender Koronarsklerose und der Wert chirurgischer Massnahmen zur Verbesserung der Durchblutung des Myokardes werden von den beiden Gruppen entsprechend verschieden beurteilt. Es erschien deshalb notwendig, das Vorkommen interarterieller Anastomosen in normalen Herzen noch einmal zu untersuchen. Bei 73 Herzen wurde postmortal die eine der beiden Koronar-Arterien mit physiologischer Kochsalzlösung durchströmt. Die Lösung enthielt Wachskügelchen mit einem Durchmesser von 35–45 μ und 75–90 μ . Die Durchströmung erfolgte unter einem Druck von max. 100 mm Hg. Das Bestehen von Anastomosen wurde dann als bewiesen angesehen, wenn Wachskügelchen in der aus der andern Koronar-Arterie gewonnenen Flüssigkeit vorhanden waren. Das Ergebnis der Untersuchung ist in der Tabelle zusammengestellt. Diese lässt erkennen, dass

¹ M. J. SCHLESSINGER, Amer. Heart J. 15, 528 (1938).
² H. L. BLUMGART, M. J. SCHLESSINGER und D. DAVIS, Amer. Heart J. 19, 1 (1940).
³ M. PRINZMETAL, B. SIMKIN, H. C. BERGMAN und H. E. KRUGER, Amer. Heart J. 33, 420 (1947).
⁴ P. M. ZOLL, S. WESSLER und M. J. SCHLESSINGER, Circulation (N.Y.) 4, 797 (1951).
⁵ J. B. MAILL und A. BLEDSOE, Arch. Path. (Chicago) 56, 577 (1953).
⁶ R. W. ECKSTEIN, Circulation Res. 3, 306 (1955).
⁷ G. BAROLDI, O. MANTERO und G. SCOMAZZONI, Circulation (N.Y.) 4, 223 (1951).
⁸ O. MANTERO, G. BAROLDI und G. SCOMAZZONI, Cardiologia (Basel) 11, 48 (1958).
⁹ M. PRINZMETAL, S. KAYLAND, C. MARGOLES und L. J. TRAGERMAN, J. Mt. Sinai Hosp. 8, 933 (1941).
¹⁰ M. M. VASTESAEGER, P. P. STRAETEN, J. FRIART, G. GANDELE, A. GHYS und R. M. BERNARD, Acta cardiol. (Brux.) 12, 365 (1957).
¹¹ W. LAURIE und J. D. WOODS, Lancet 1958/II, 812.
¹² A. RAVIN und F. F. GREEVER, Arch. int. Med. 78, 125 (1946).