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).

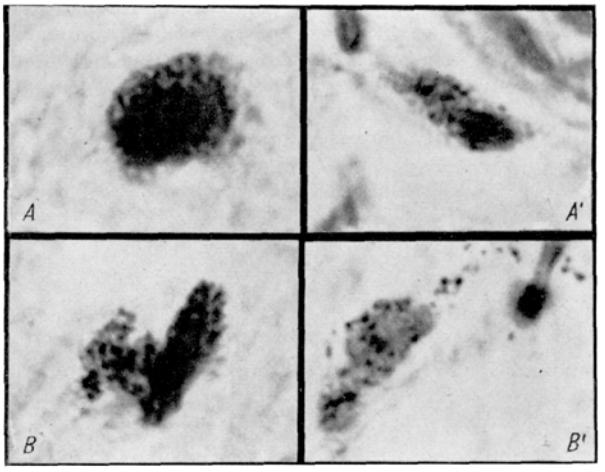

Fig. 2.—(*A–B*) Mast-cells in remission phase of asthma and (*A'-B'*) in asthma attack (same patients). Magnification ×2000.

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 authors[1] 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.

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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 desametazone 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.

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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

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interarterielle Anastomosen bei stenosierender Koronarsklerose und bei Kammerhypertrophie sehr häufig (70% der Fälle), bei normalen Herzen jedoch selten (7% der Fälle) sind. Es scheint, dass der pathologische Prozess selbst die Eröffnung bestehender oder die Bildung neuer Anastomosen zur Folge hat. Die Diskrepanz der Ansichten über die Häufigkeit dieser Anastomosen bei normalen Herzen bleibt ungeklärt. Es ist möglich, dass bei Verwendung unphysiologisch hoher Strömungsdrucke[3,7,9,10] künstliche Kommunikationen entstehen, welche Anastomosen vortäuschen.

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Medizinische Universitäts-Klinik Basel und Pathologisch-Anatomische Anstalt der Universität Basel, 4. April 1959.

Summary

Wax spheres with a diameter of 35–45 μ and 75–90 μ were injected into one coronary artery of 73 human hearts. The finding of spheres in the opposite coronary artery was considered as evidence for the presence of interarterial coronary anastomoses. In hearts with occlusive coronary sclerosis or ventricular hypertrophy anastomoses were found in approximately 70% of the cases. Of the 15 normal hearts only 1 (7%) was found to have anastomoses.

Chromosomes Deficiencies Induced with Myleran

In previous experiments[1,2], we reported the peculiar properties of one mesyloxy-alkane compound (Myleran) to break chromosomes without giving rise to a considerable number of rejoinings. In *Vicia faba*, the breaks induced with this substance were strictly localised in or near heterochromatic regions of the genome. This peculiarity considerably reduced the number of potential deficiencies obtainable with this chemical. It also enables us to analyse the dynamics of such chromosome deficiencies and their relationships with heterochromatin.

In *Vicia*, contrary to the pyrimidine derivative: maleic hydrazide[3,4] but similarly to other alkylating agents[5], Myleran produces a large amount of localised breaks in acrocentric chromosomes. We found another maximum of localised breaks in the zone of the nucleolar organiser body. This is similar to the effect of 8-Ethoxycoffeine[6,7]. However, in further investigations, this localisation proved to be less constant.

Another conclusion that we reached was that there is a shortening of the total length of chromosomes during the development of growing root tips.

Now, the question arises: is this shortening of the measured genome really due to chromosome deficiencies, or is it attributable to some quite different effect of coiling?

This work attempts to answer this question. For such a purpose, dried seeds of *Vicia faba* (commercial variety: Åkerböna Weibull) were treated at 21°C with 20 mg% of Myleran during $1^1/_2$ h (10 seeds/100 cm³ solution). After drying, they were germinated at the same temperature. Root tips were fixed at about 30 h intervals starting from the second day after sowing. Pretreatment with colchicine 0·05% for 2 h was used before fixing in acetic-alcohol 1:3 for 2 h and staining with Feulgen.

In our analysis, we tried to compare the frequencies of breaks (iso-chromatid and chromatid) with the frequencies of observed deficiencies. Since it was previously found that the average number of breaks decreases from the second mitosis after treatment, it was therefore indispensable to work with increased numbers of metaphases.

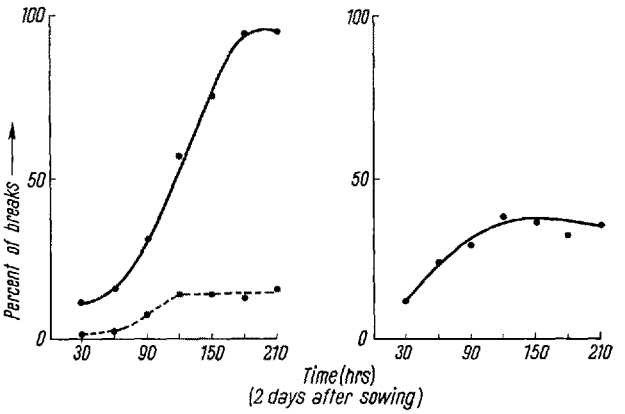

Fig. 1 Fig. 2

Fig. 1. – Observed chromosomes deficiencies per 100 breaks.
——— total number
----- number in chromosome *M*

Fig. 2. – Percent of breaks affecting *M*-chromosome.

The first graph shows an increase of deficiencies expressed as the percentage of breaks (total number of about 200 scored breaks). Even those cells which contain fragments degenerating into micronuclei were scored as deficiencies. This accumulation of deficient caryotypes is an indication of their variability. However, it is obvious that this viability must not be the same for all types of cells; it means that some breaks have a better chance of being selected. Therefore, a more careful comparison of breaks and deficiencies has to be made.

A first approach to this problem is the analysis of breaks and deficiencies observed in the metacentric chromosomes (*M*) and in the acrocentric (or subtelocentric) chromosomes (*S*).

Concerning breaks, Figure 2 shows that the proportion affecting *M*-chromosomes increases while that affecting *S*-chromosomes diminishes till they reach a certain equilibrium level 120 h after the first recording. The increased proportion of breaks in the *M*-chromosome was observed to be mainly due to an increased frequency of breaks in the nucleolar organiser. This is in close agreement with our previous work.

Concerning deficiencies, Figure 1 indicates that as the total amount of deficiencies increased, the amount of *M* and *S* deficiencies also increased but much more in *S* than in *M*. There is evidence that some deletions of the *M*-chromosome are not viable. This fact was predicted in previous experiments. However, some caryotypes with only one nucleolar constriction were observed. We can relate this to the increasing number of breaks in this zone. Both breaks and deficiencies reach equilibrium after a certain time. At this time, the number of deficiencies in

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