In the next section, four cell classes are presented, two of them normal, two neoplastic. In each class, the emphasis is put on a different expression of the interaction of motility, shape, and fibrillar organelles. Activated blood platelets show an intense stationary motility coupled with conspicuous shape changes and alterations of microfibrillar organelles. The main regulator of this characteristic series of events is Ca^{2+}. In no other cell type has the role of this cation been so extensively analyzed, and it appears timely to apply experience on calcium in thrombocytes to motile phenomena in other cells. For instance to the locomotion of fibroblasts. In these cells, studies on the interaction of adhesion and contraction for generating translocative motility are well advanced. They allow us to envisage a first rough concept of the mechanics of locomotion in this particular cell type. For other expressions of cell motility, e.g., the projection of growth cones in neuroblastoma cell, other mechanisms, based on polymerization–depolymerization processes rather then on contraction, must be taken into consideration. Studies on the interrelation of motility, shape and fibrillar organelles are particularly promising in leukemia. In leukemia cells processed for electron microscopy, the functional state—rest or locomotion—in the moment of fixation is revealed by the spherical or polarized configuration. Thus, electron microscopic findings concerning shape and fibrillar organelles of fixed cells can be directly related to the motile behavior of living cells.

Regulation of the Contractile System of Blood Platelets

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ALTHOUGH platelets generally do not show locomotion, they nevertheless contain large amounts of actomyosin, which is essential for many aspects of platelet activity [1]. Upon activation the disk-shaped platelet generally undergoes a fast transformation to a 'spiny sphere', whereby long filiform pseudopodia are formed. Activation is brought about by a wide variety of agents, such as thrombin, collagen and ADP, but also by surface and cell–cell interaction in closely packed platelets. 'Rapid shape change' involves the temporary disappearance of a ring of microtubules which is localized in close proximity to the inner surface of the plasma membrane. Under physiological conditions, shape change is linked to cell aggregation and followed by the contraction of formed pseudopodia. During this process, the aggregates are drawn together. With higher concentrations of inducer, aggregation is accompanied by the release of a variety of substances from at least two types of specific storage organelles.

By the use of ionophores for Ca^{2+} ions, such as A 23187, it has been possible to show that all manifestations of platelet activity are due to the mobilization, in the cytoplasm, of Ca^{2+} ions [2]. It is most likely that this mobilization occurs in three different steps, namely:

(1) From the stimulated plasma membrane [3]: the local appearance of small amounts of the cation would be causative for the induction of the contraction of a performed network of submembranous filaments leading to morphological changes ('rapid shape change') and to the disappearance of the microtubular ring. (2) Upon more extensive stimulation, Ca^{2+} ions are mobilized from cytoplasmic vesicles, termed the dense tubular system (DTS) and corresponding to the sarcoplasmic reticulum of muscle cells [4]. Concomitant with this step, large amounts of F-actin are formed throughout the cytoplasm from a dispersed precursor [5], and gross contractile activity sets in. Simultaneously, two other important manifestations of platelet activity are initiated: the release reaction and prostaglandin (PG) synthesis. (3) Concomitant with the release reaction the plasma membrane acquires permeability for Ca^{2+} ions. It must be noted, though, that this influx of Ca^{2+} is not the trigger for platelet activity: shape change and the release reaction occur also in Ca^{2+}-free systems.

Although the essential role of the Ca^{2+} ions is well established, very little is known about the nature of the membrane signal which induces their mobilization. Perhaps cluster formation of membrane constituents is the essential event [6]. Shape change and release-

linked 'second phase aggregation' in principle are reversible phenomena. This means that the platelet is able to remove again cytoplasmic Ca^{2+}. Platelet activation is prevented and reversal reactions are accelerated by all measures which increase the intracellular level of cyclic AMP. Some of the most powerful known blockers of platelet activity, such as PGE_1, PGI_2 (prostacyclin, synthesized by endothelial cells from PG-endoperoxides) and adenosine exert their action via the activation of adenylate cyclase. We have shown [7, 8] that the effect of cAMP on platelet activity consists in the stimulation, via a protein kinase, of a 'calcium pump'. This is responsible for the uptake into a vesicular system, most likely the DTS, of the cation from the cytoplasm and perhaps also for the extrusion of Ca^{2+} ions to the outside of the cell. This finding probably also explains earlier observations partly made on tumor cells on the effect of cAMP on cell motility [9].

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The Locomotory Machinery of Fibroblasts

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All substratum-based cell locomotion may be considered as the co-operative result of three essential processes: the cell must make adhesions to the substratum; must move its bulk in relation to these adhesions, must protrude new material forward in order to make new adhesions. If the cell is to remain in a 'steady-state', it must also de-adhere from the substratum at the rear and retract the rear end into the cell bulk. One conclusion to be drawn is that a portion of the locomotory machinery must cycle in position with respect to the moving cell. This portion includes all