A complete understanding of the biomechanics of cell division has been considered a noble goal for over a century. At any one time during that period, the number of investigators actively contributing new data to the field has been, by present standards, small, and progress has been slow. Cytokinesis in animal cells, the aspect of the process that will be the subject of this paper, has always been a subject of intense speculation. Many of the early cell and developmental biologists appeared very willing to publish their ideas on the subject, even though it might not be in their area of expertise. Speculation or theorization usually took the form of models that could be either physical or verbal. To be considered feasible, models had to replicate the normal events of cytokinesis and because of the apparent simplicity of the phenomenon, the same effect could be achieved by models based upon different physical mechanisms. In this circumstance, the workability of a model revealed little about the possibility that it accurately resembled the events that take place in the cell. There was also a near absence of information concerning the division-relatedness of the structures and activities and whether their roles were active or passive. In consequence, the number of possible mechanisms was immense. As late as 1928, Heilbrunn was moved to remark that, "Usually it is easier to make a new theory of cell division than to test an old one." In fact, by that time it was rather difficult to make a really new theory as the last major original theory was proposed about twenty-five years before. Most of what followed has been recycling with variations. But why that kind of statement, even though made in partial jest, should be accurate enough to cause some discomfort is presently unclear. Experimentation was then an accepted method of analysis and at that time several excellent micromanipulators were commercially available. It occasionally took a long time for the results of experimentation to affect cell division theory. Despite convincing demonstrations by Ziegler (1896) and by McClendon (1908) that cytokinesis in the absence of chromosomes is perfectly possible, essential roles for chromosomes were proposed decades later. It may be possible that this circumstance arose from simple ignorance of the literature. But it could also have resulted from a devout faith that the beauty, ingenuity and personally lovable qualities of the hypothesis or model would eventually triumph over a few ugly, inconvenient facts.
We carry a heavier burden than our predecessors. For those who wish to know the mechanism of cytokinesis that actually operates in the cell, the days of freewheeling speculation are over. Information derived from measurements and from experimentation has greatly reduced the number of possibilities. Every hypothesis has predictive properties, and the accuracy of the prediction can often be determined by forcing cells to divide under circumstances that were not specifically allowed for when the hypothesis was created. Because the cleaving echinoderm eggs that are used in experimental studies are all very similar, and because echinoderm eggs also serve as the pattern for models of typical animal cell division, the possibility that inconsistencies between the behavior of models and that of real cells can be attributed to species differences is small. The certainty that additional important information concerning the process will be forthcoming does not change the requirement that, when properly interpreted, all existing information must fit into a single, logically coherent pattern. No significant exceptions are possible.

In the study of cytokinesis, one of the current central problems is understanding the basis of the unequal distribution of active contractile components associated with the surface. Division activity requires previous interaction between the achromatic mitotic apparatus and the surface and, because we presently lack means for direct study of the local events that lead to or constitute the organizing activity that creates the division mechanism, it has been necessary instead to study the events and relations that can affect the process, both directly and indirectly.

**TIMING**

The causal chain of events that culminates in division follows a characteristic schedule. The cell's ability to divide when the mitotic apparatus is removed late in the mitotic cycle (Tatsu, 1912; Hiramoto, 1956) revealed not only the absence of any physical role for the mitotic apparatus, but also the existence of a pause between the time when the surface alteration elicited by the mitotic apparatus achieves irreversibility and the beginning of division mechanism function. The duration of the pause is about 4 min (Hamaguchi, 1975; Rappaport, 1981). The time when the interaction between the mitotic apparatus and the surface begins has not been determined. It has, however, been shown that an interaction period of about 1 min is sufficient to establish a furrow (Rappaport and Ebstein, 1965; Rappaport, 1965). The fact that precocious furrows form when the mitotic apparatus and the surface are located closer together than normal (Rappaport, 1975) strongly suggests that they are capable of interacting before they normally do.

Under experimental conditions, the mitotic apparatus can establish several furrows in succession (Harvey, 1935; Rappaport and Ebstein, 1965; Rappaport, 1975, 1985). This fact strongly suggests that the property of the mitotic apparatus that initiates surface contraction is preserved and functional after the time when the furrow first appears. In the sand dollar Echinarchnus parma, the mitotic apparatus has been shown to initiate as many as 13 successive furrows as it was pushed back and forth in a cylindrical cell over a 24.5 min period (Rappaport, 1985). After it has been shifted several times, its radiate structure appears diminished and the distance between the astral centers is abnormally large. Because both of these changes have been associated with reduced ability of the mitotic apparatus to establish furrows, it is not clear whether the shifted mitotic apparatus loses its ability because of an event associated with the normal cell cycle or because of the structural and geometrical changes caused by the manipulation. The cell's ability to form both precocious and belated furrows suggests that the precise