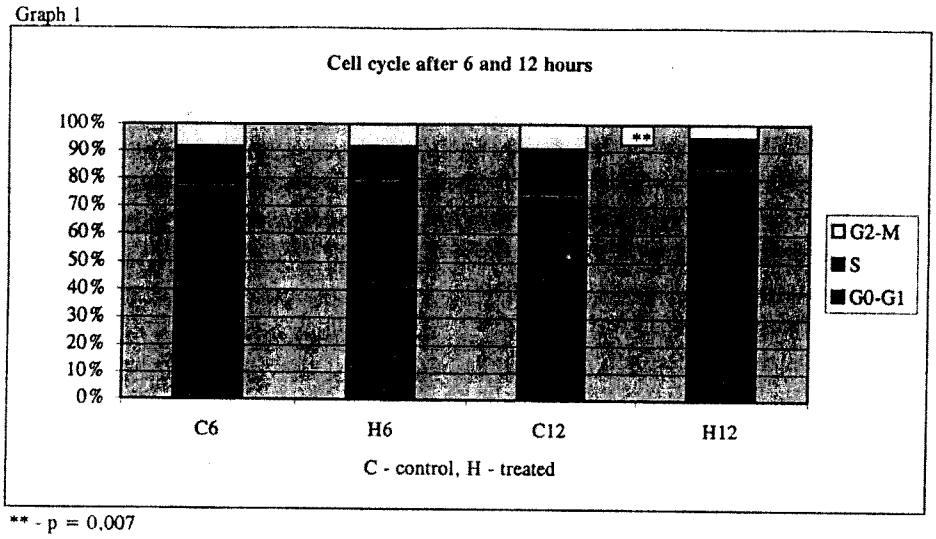

Fig. 6. Cell cycle after 6 and 12 h. Abscissa: C6, C12 – control groups after 6 (12) h, H6, H12 – treated groups (hydrocortisone) after 6 (12) h. Ordinate: % of cells, ** – $P < 0,01$.

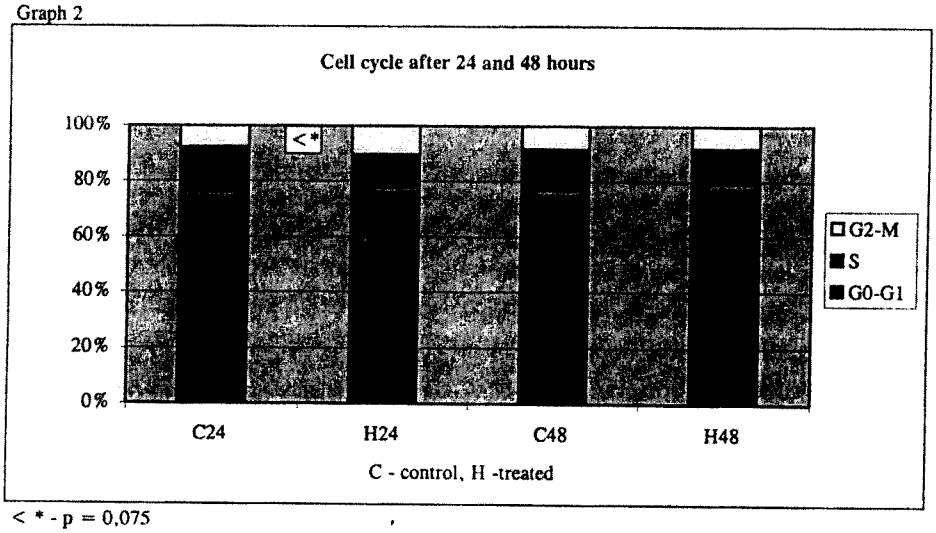

Fig. 7. Cell cycle after 24 and 48 h. Abscissa: C24, C48 – control groups after 24 (48) h, H24, H48 – treated groups (hydrocortisone) after 24 (48) h. Ordinate: % of cells, $<* - 0,1 > P > 0,05$.

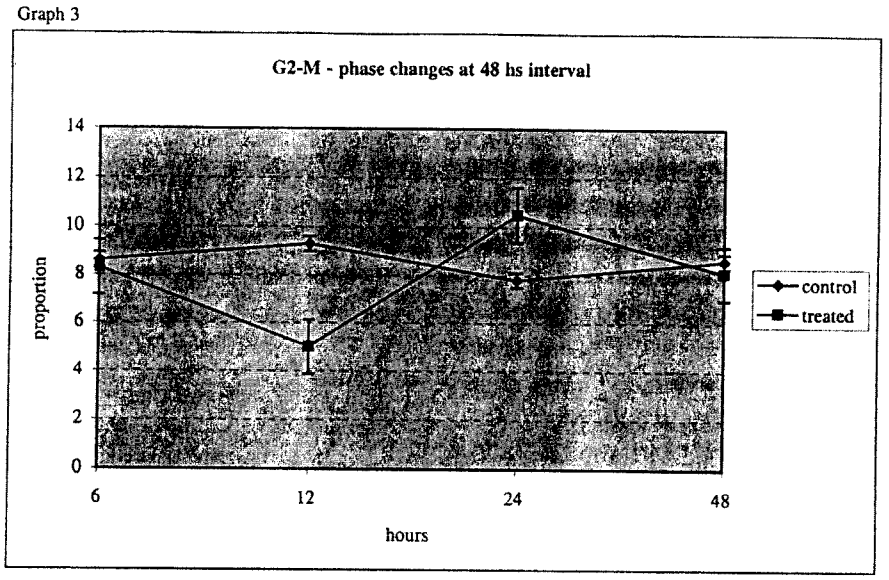

Fig. 8. G2-M phase. Abscissa: time interval in hours. Ordinate: proportion of cells in %.

Discussion

The term membrana reuniens (Vereinigungshaut, Rathke, 1838) designates the internal layer of the pericardial fold forming secondarily the ventral thoracic wall in the chick embryo (Steding and Klemeyer, 1969; Männer et al., 1995). We use the term MR despite of the fact that it was first used in mammalian embryology, and the origin of the pericardial cavity in birds and in mammals differs. The reason for revisiting the term is, besides the Rathke's priority, that the term suits perfectly, according to our findings, to the functional meaning of the structure.

The white ridge observed in the upper region of the ventral body wall in some embryos developing EC (see Results, Group 1), and always well constituted in the controls, originates from accumulation of both the mesenchyme and ectoderm in place where the chest wall closes. It is formed due to the cell crowding caused by morphogenetic movements (Seichert and Seichertová, 1998). A number of macrophages with engulfed remnants of apoptotic cell debris, which we have documented in the paper mentioned above, point to a process of cell reduction as known from other morphogenetic systems (Saunders et al., 1962 – chick wing etc.). This explains the remarkable reduction and even more frequent absence of this ridge in the embryos with the developing EC.

Using flow cytometry we succeeded to demonstrate in experimental embryos a significant lowering of proliferation activity 12 h after H administration (Fig. 8). Interestingly, the depression of mitoses failed to be caused by prolongation of the S phase as observed in embryos treated with cyclophosphamide (Heringová et al., 1998), but by blockage of entering mitosis. These findings are in consensus with the results of previous studies (Pavlík et al., 1986; Peterka et al., 1997). The mechanism of EC may be the following: normal body-wall morphogenesis requires a certain level of proliferation activity of PM. In case of the mitotic depression followed by lack of cellular material in the critical period of body-wall formation, an insufficient amount of PM is at disposal for bridging, under the guidance of MR, the developing heart. MR, therefore, remains thin, and the growing and pulsating heart brings about its further thinning and bulging. After the H levels sink down, proliferation rate rapidly normalizes and temporarily even overshoots. The deficient material becomes restituted; however, too late with respect to the critical period (Jelínek and Rychter, 1979). Enormously distended and vaulted MR affords for PM an insufficient guidance for crossing the already displaced heart.

We therefore conclude that the mechanism of EC development induced by H administration differs substantially from that triggered by mechanical disruption of MR. In the latter case, a structure is lacking necessary to guide the PM shift over the developing heart prominence. After H administration the structure is always present; however, during the critical period of body wall closure there is, in consequence of mitotic inhibition, an apparent lack of PM material for bridging the already dislocated heart.

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