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Models Showing Accumulation.

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Models consisting of a non-aqueous layer separating 2 aqueous solutions (the inner being more acid than the outer) show the following points of resemblance to *Valonia:*

(1) Penetration from the outer into the inner aqueous solution (artificial sap) is in the following order $K > Na > Mg > Ca$. Also $Cl > SO_4$.

(2) Instead of equilibrium a steady state is reached in which the artificial cell "sap" increases in volume but remains approximately constant in composition.

(3) Osmotic pressure and K-concentration become greater inside than outside.

(4) In certain models the "sap" contains CO_2. As potassium enters and $KHCO_3$ is formed the osmotic pressure rises. Although CO_2 is regarded as a waste product in organisms it acts in this case to increase the energy stored in the cell.

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Anginal Syndrome Induced by Gradual General Anoxemia.

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With individuals subject to attacks of chest pain, in whom there is no evidence by physical or electrocardiographic examination of myocardial (coronary) disease, it is often difficult to be certain of the origin of the pain. We wished to distinguish between those with pain due to impaired coronary circulation, and those in whom the pain arose otherwise. It occurred to us that if one were to produce a general anoxemia, and, therefore a local cardiac anoxemia, there might appear differentiating responses in these 2 groups.

By means of rebreathing, we were able to produce a state of general anoxemia in human subjects. The carbon dioxide was absorbed. It usually took about 10 minutes for the oxygen to become so low that the patient became uncomfortable.

Twenty-seven patients were subjected to the test. Fourteen patients were used as controls. The controls consisted of 4 patients with normal hearts. The remaining cases were patients with chronic valvular disease, paroxysmal auricular fibrillation, rheumatic fever, spondylitis, gall bladder disease, and cardiospasm. None of these patients developed pain. Thirteen were patients with clear-cut histories of attacks of precordial pain brought on by exertion, excitement, eating, or exposure to cold. Ten developed pain during the rebreathing test. Eight of these had no physical or electrocardiographic signs of myocardial disease. The pain appeared when the oxygen fell to about 9 to 10%. This ordinarily took about 8 to 10 minutes. Subjects were advised to raise their hands when they felt uncomfortable, and the experiment was stopped.

It is furthermore interesting to observe that 2 other patients with clinical angina and intra-ventricular block developed pain and additional electrocardiographic changes during the anoxemia. The changes found were depression of the R-T segments in Leads II and III, and in another instance in Leads I and III. Unfortunately before further tests could be made, the second patient died, but the patient with changes in II and III was observed further. We were unable to reproduce the electrocardiographic changes with (1) oxygen and carbon dioxide inhalations, (2) intravenous atropin, (3) adrenalin, (4) pitressin, and (5) amyl nitrate.

Three patients with clinical coronary artery disease, one with definite electrocardiographic changes, and two with no electrocardiographic changes, responded negatively to the test.

We feel, therefore, that it is possible to reproduce the pain of angina pectoris in susceptible subjects by inducing general anoxemia. We assume in the explanation that the coronary circulation, normally adequate, becomes inadequate during anoxemia. We are not prepared to state that anoxemia *per se* is responsible for the pain that appears during the experiment. It is possible that the explanation of angina as being due to a cardiac ischemia combined with a piling up of Lewis's P-factor explains the pain that appears during rebreathing. We feel that our work, although not conclusive, tends however to support the anoxemia theory of angina pectoris. This test promises to be of some value in differentiating the causation of chest pain.