SUMMARY AND CONCLUSIONS

1. The mechanical stress important in the localization of atherosclerosis is a stretching of the arterial wall. This is influenced by arterial blood pressure, surrounding tissue pressure, radius of lumen, curvature of artery and arterial fixation.

2. This mechanical stress is counteracted by the components of the arterial wall.

3. This stretching force may be linked with the stress which localizes lesions in scurvy.

4. Ascorbic acid deficiency in guinea pigs produces atherosclerosis regardless of whether the scurvy is acute or chronic.

5. Atherosclerosis of scurvy occurs at normal cholesterol levels and without deposits of lipid in the intima-media system. This form of atherosclerosis thus closely simulates the human form of the disease.


7. The ground substance disturbance associated with atherosclerosis is of major importance in the pathogenesis of arterial calcification, intimal hemorrhage and of thrombosis.

8. Cholesterol feeding interferes with ascorbic acid metabolism in rabbits and in guinea pigs receiving oral ascorbic acid.

9. Acute infection in guinea pigs produces minimal atherosclerosis in about 33% of guinea pigs studied.

10. Thyroid and cortisone have no inhibitory effect on the atherosclerosis of cholesterol feeding in guinea pigs in a small series nor do they inhibit the atherosclerosis of scurvy.

11. Massive doses of parenteral ascorbic acid may be of therapeutic value in the treatment of atherosclerosis and the prevention of intimal hemorrhage and of thrombosis.

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