of the aorta, we incline to the view that it is secondary to the inflammatory processes of the media. Our increasing familiarity with syphilitic mesaortitis indicates that the lesions in the arteries are of an infective nature and that this infection lies in the adventitia and media. With this infective process there is truly a destructive process in the media, but there is also a concurrent inflammation which we feel convinced affects the intima.

The effect of moderately increased work upon the arterial walls leads to an hypertrophy of the muscle elements, giving way not infrequently to a later degeneration. The degeneration becomes most evident in the musculo-elastic layer which gives rise to atheroma. The growth of connective tissue elements over this degeneration is secondary.

When increased work exceeds the capabilities of the arterial tissues, degenerative changes set in from the first and no intimal hyperplasia is evident.

CONCLUSIONS.

We agree with Jores and others that not one but many factors may be at work leading to intimal hyperplasia. Among these factors may be mentioned infection, bacterial toxins, organic poisons, inflammation and increased arterial tension.

The theory of Thoma that the connective tissue developed in the intima is compensatory cannot be sustained.

From the evidence which we have at hand it is not possible to state that the proliferative changes in the intima are uniformly secondary to the weakening of the media.

Common influences may act simultaneously upon the media and the intima.

Progressive medial degeneration of the peripheral arteries (Moenckeberg's sclerosis) is the result of muscle fatigue coupled with nutritional disturbance.

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