Sonic stress as one of the cardinal risk factors for atherosclerosis

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It is well recognized that stress and hyperlipidemia are cardinal risk factors in causing the atherosclerotic lesion. However, only a few reports have appeared on the interrelation between sonic stress (stress produced by sonic noise) and persisting abnormal lipid metabolism\(^1,2\)). The intent of this study is to clarify the effect of stress and lipid metabolism in the development of atherosclerosis. The experimental animals were divided into several groups, as shown in Fig. 1. They were subjected to changes in the serum lipid levels, deposition of a lipid substance in the aortic wall, and deviations in the weights of the several major visceral organs. All were fed for 20 weeks; and every 2 weeks blood specimens were collected and the serum lipid levels were determined. Sonic stress was produced by the method described by Hasegawa et al.\(^3\)). The animals were exposed to 600 Hz, 120 dB from a speaker continuously for 24 hours. Then the animals were sacrificed by decapitation. Part of the thoracic aorta was excised and fixed in 10% buffered formalin and studied under a conventional microscope. Particular attention was paid to the deposition of lipid streaks, using Oil Red O staining.

There were no significant changes in the weight of the major visceral organs. Changes in the serum lipid levels are shown in Fig. 1. The TC level increased in the sonic-stress group; however no significant difference between the groups was recognized. Similarly, the two groups showed no significant in the HDL-C and LDL-C levels.

The sonic-exposed groups showed a marked increase in the TG level (varying from 48 to 250 mg/dl), in comparison with the control (from 44 to 84 mg/dl), substantiating the premise that sonic-exposure may cause an increase in the blood TG content. The addition of γ-oryzanol suggested that this drug might have an inhibitory effect on TG content elevation, both in the experimental and control groups. This effect was more evident in the sonic-exposed TG elevation. The thoracic aorta was excised at the end of the experiment and stained with Oil Red O stain for histologic examination of the fatty streaks. The involvement of atheromatous plaques in each groups was quantitatively demonstrated or visualized in Fig. 2, where the lesions were delineated by a tracing method. The ratios are; 62.0 ± 9.5% for the sonic-exposed group and 42.0 ± 6.9% for the control groups.

It was shown that sonic stress can enhance lipid deposition on the walls of the thoracic aorta. The addition of γ-oryzanol was very effective in preventing lipid deposition; and this therapeutic
effect was much more conspicuous in the sonic-exposed group. In this study, particular attention was paid to the interrelation between abnormal lipid metabolism and progression of atherosclerosis. For stress, we have adopted sonic-exposure, instead of the more widely used water immersion method. At the moment, we do not have a concrete evidence to assume that sonic stress is a direct cause for progression of the atherosclerotic process; however, it is reasonable to believe that this may be a contributing factor for induction of atherosclerosis. In a preliminary experiment, this sonic method caused an increase in blood catecholamine and corticosteroid levels (69% and 140%, respectively). The histological study disclosed atrophy of the thymus gland and compensatory hypertrophy of the adrenal glands. Thus it was proven that sonic exposure may serve as one of the most versatile methods to produce a stress state in animals.

In the present investigation, sonic stress caused a marked increase in the TG level in the blood; however, the TC and LDL-C levels remained normal or failed to show significant changes. The histological observation revealed that the sonic stress may enhance lipid deposition in the thoracic aorta. However, it may be difficult to explain the development of fatty streaks simply on a pathological basis. It should be noted that the stimuli which may cause stress tend to raise the blood pressure and trigger exaggerated platelet aggregation through the action of thromboxane A2. Participation of these contributory factors is also considered significant in the development of this pathological condition.

Moreover, the results of our study showed that γ-oryzanol may possess an effect to deter the
development of the atherosclerotic process by sonic stress. This may indicate that the drug possesses a distinct inhibitory action on the development of an atherosclerotic lesion through the stabilization of the autonomic nervous and endocrine systems.

To prove that sonic stress is one of the cardinal contributory factors for the development of an atherosclerotic lesion, further detailed investigations are needed. It is meaningful, however, to re-evaluate stress as one of the most important contributing factors in the progression of atherosclerosis, which is one of the most serious adult diseases today.

References


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