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Citation: *The Journal of the Acoustical Society of America* **27**, 1142 (1955); doi: 10.1121/1.1908143

View online: <http://dx.doi.org/10.1121/1.1908143>

View Table of Contents: <http://asa.scitation.org/toc/jas/27/6>

Published by the *Acoustical Society of America*

Mechanical Mechanism of Destructive Effects of Sound on Tissue*

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(Received July 6, 1955)

A mechanical mechanism is presented to explain some of the nonthermal, noncavitation effects of high-intensity sound on tissue. The theory proposes that the observed effects result from unidirectional forces produced by the acoustic wave, and that these forces cause elastic failure in the system. Some experimental verification is presented from measurements on irradiated frog muscle tissue and on irradiated frog spinal cord.

INTRODUCTION

SOUND waves propagated in tissue can produce damage by a variety of mechanisms. The conversion of mechanical energy into thermal energy which takes place in absorptive tissue can give rise to temperatures which are injurious to the tissue.¹ The formation, growth, and subsequent collapse of gaseous and vapor cavities under the action of high-amplitude sound waves can produce destructive forces and high temperatures which are also injurious to the tissue.² There have been a number of experiments carried out in which sound waves have produced damage in biological systems under conditions which appear to make thermal and cavitation mechanisms unlikely.³⁻⁵ It is the purpose of this paper to develop a mechanical mechanism which can explain some of these observed nonthermal, noncavitation effects of high-intensity sound on tissue.

Sound is a mechanical phenomenon, and when it is propagated in a medium various oscillatory and unidirectional forces result. If a sound wave in a fluid is oscillatory in nature, then any structure imbedded in this fluid is subject to an oscillatory viscous force. In addition, because of the complex nature of propagation of high-amplitude sound waves, the structure is also acted upon by unidirectional forces such as radiation pressure, Oseen forces, and mass streaming viscous forces. In the following development these unidirectional forces will be lumped together as a group and called higher order forces, since they do not show up mathematically from a consideration of the infinitesimal wave theory. The mechanism to be discussed is based upon an effect caused by these unidirectional forces. It is assumed that they are large enough to cause a

structural member of the tissue involved to stretch beyond normal limits.

ANALYSIS OF MECHANISM

The analysis of particle motion in the sound field is carried out for a traveling wave field since most of the quantitative data available is from traveling wave field experiments.

Consider a particle suspended in a fluid in which sound is propagated. The equation of motion of this particle under the influence of the linear viscous force due to the sound in the medium can be obtained from the differential equation.⁶

$$M\ddot{r} + R\dot{r} = m(\partial^2\xi/\partial t^2) + R\dot{\xi} \quad (1)$$

where ξ is the particle velocity of the medium, \dot{r} is the velocity of the particle, M is the effective mass of the particle, m is the effective mass of the displaced fluid, and R is the viscous force coefficient.

If the particle is under an elastic restraint, and if a unidirectional constant force caused by higher order effects is taken into account, then Eq. (1) becomes

$$\ddot{r} + \frac{R}{M}\dot{r} + \frac{K}{M}r = -\frac{m}{M}(\partial^2\xi/\partial t^2) + \frac{R}{M}\dot{\xi} + \frac{F}{M} \quad (2)$$

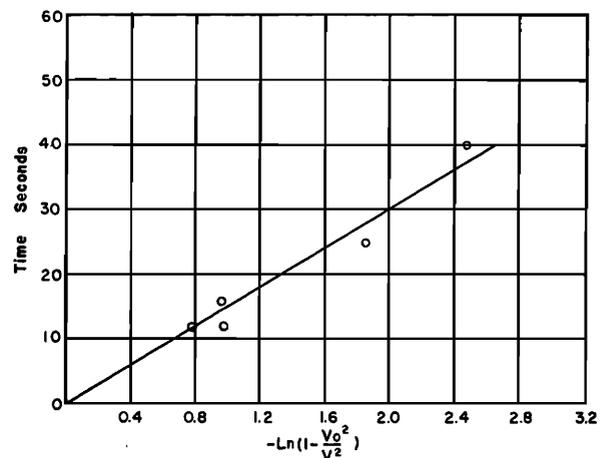


FIG. 1. Muscle data plotted in accordance with theory, $t_B = -(R/K) \ln[1 - (V_0^2/V^2)]$. Atmospheric pressure, initial temperature 18–20 degrees centigrade.

* See, for example, W. J. Fry, J. Acoust. Soc. Am. 24, 412 (1952).

* Submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at the University of Illinois, 1954.

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¹ See for example J. F. Lehmann, J. Acoust. Soc. Am. 25, 17 (1953).

² For a discussion of bubble formation in tissue see Harvey, Whiteley, McElroy, Pease, and Barnes, J. Cell. Comp. Physiol. 24, 23 (1944).

³ Fry, Wulff, Tucker, and Fry, J. Acoust. Soc. Am. 22, 867 (1950).

⁴ Fry, Tucker, Fry, and Wulff, J. Acoust. Soc. Am. 23, 364 (1951).

⁵ Welkowitz and Fry, J. Cellular Comp. Physiol. (submitted for publication).

where K is an elastic constant and F is the constant unidirectional force.

If the particle velocity of the traveling wave propagated in the medium is taken as

$$\xi = u \sin(\omega t + \varphi) \tag{3}$$

in the region of the moving particle (this assumes that r is small compared to the wavelength), then Eq. (2) can be solved to yield

$$r = C_1 e^{\alpha_1 t} + C_2 e^{\alpha_2 t} + A \cos \omega t + (F/K) \tag{4}$$

where

$$\alpha_1 = - (R/2M) + \left(\frac{R^2}{4M^2} - \frac{K}{M} \right)^{\frac{1}{2}}$$

$$\alpha_2 = - (R/2M) - \left(\frac{R^2}{4M^2} - \frac{K}{M} \right)^{\frac{1}{2}}$$

$$A = u \left[(m/R) \sin \varphi - (1/\omega) \cos \varphi \right]$$

$$\varphi = \tan^{-1} \frac{\left[(\omega m/M) + (1/\omega) \left(\frac{K}{M} - \omega^2 \right) \right]}{\left[(m/R) \left(\frac{K}{M} - \omega^2 \right) - (R/M) \right]}$$

The physical picture being considered is one wherein, under the action of the unidirectional force, in addition to the vibration due to the linear viscous force, the particle moves unidirectionally away from its equilibrium position to a point where its elastic restraining member is stretched beyond normal limits and is permanently damaged. This deformation is assumed to be much greater than the amplitude of the oscillatory motion due to the linear viscous force. The physical condition of the system corresponding to this picture is that the elastic restraining force is small compared to the viscous damping force. The mathematical condition is that

$$(K/M) \ll (R^2/4M^2). \tag{5}$$

Within the framework of this physical picture and under the initial conditions that at

$$t=0, \quad r=0, \quad \dot{r}=0,$$

Eq. (4) reduces to the approximate solution

$$r = A \cos \omega t + (F/K)(1 - e^{-(K/R)t}). \tag{6}$$

Damage is assumed to occur when the deformation r is equal to some value r_B where $r_B \gg A$. From Eq. (6), it can be seen that the irradiation time required for damage to occur is approximately given by

$$t_B = - (R/K) \ln [1 - (r_B K/F)]. \tag{7}$$

It was previously indicated that, F , the unidirectional force, was due to higher order effects. For many of

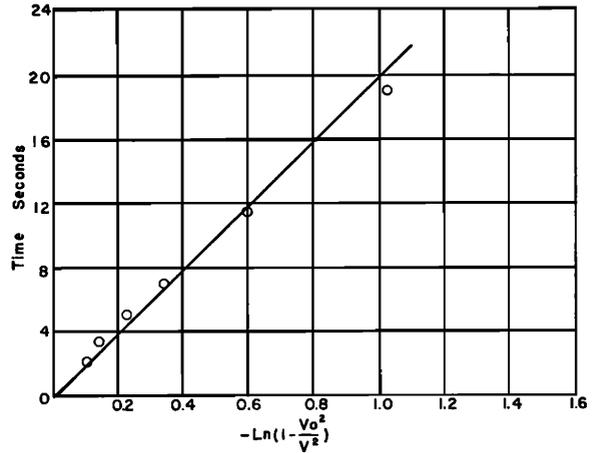


FIG. 2. Frog paralysis data plotted in accordance with theory, $t_B = - (R/K) \ln [1 - (V_0^2/V^2)]$. Atmospheric pressure, initial temperature 0-1 degree centigrade.

these it can be considered proportional to the square of the particle velocity amplitude.

$$F = fu^2. \tag{8}$$

Under these conditions

$$t_B = - (R/K) \ln [1 - (r_B K/fu^2)]. \tag{9}$$

It is apparent from Eq. (6) that unless a certain threshold force is reached, damage never results. In terms of the square of the particle velocity amplitude this threshold is

$$u_0^2 = (r_B K/f). \tag{10}$$

The irradiation time required to produce damage can then be expressed in terms of the threshold as

$$t_B = - (R/K) \ln [1 - (u_0^2/u^2)], \tag{11}$$

or in terms of the driving voltage on the transducer (a readily measurable parameter) as

$$t_B = - (R/K) \ln [1 - (V_0^2/V^2)], \tag{12}$$

where V_0 is the threshold crystal voltage.

EXPERIMENTAL VERIFICATION

From some previous work by Fry and associates,⁴ there are data available on the minimum irradiation time required for paralysis of the hind legs of frogs irradiated in the lumbar enlargement region of the spinal cord. Tests indicated that these results were due to neither excessive heating nor cavitation. Similarly from some work on muscle tissue by Welkowitz and Fry⁵ data are available for minimum irradiation time required for a 10% permanent reduction of the action potential. In this case, also, tests indicated that the observed effects were due to neither heating nor cavitation. According to Eq. (12), if the theory proposed explains the measurements, then a plot of the time required for the observed effect against

$-\ln[1 - (V_0^2/V^2)]$ should yield a straight line through the origin. The slope of the resultant line is then equal to (R/K) . Figures 1 and 2 show the data plotted as indicated. In both cases, the voltages corresponding to 100 seconds of irradiation time were used as threshold voltages V_0 . The measurements appear to agree quite reasonably with the theory. For the case of the spinal cord data, a value of (R/K) of 20 seconds is obtained from the resultant straight line. For the case of the muscle tissue the value obtained is about 15 seconds. It appears plausible that the two types of frog tissue (spinal cord and muscle) would have about the same order of magnitude of mechanical constants, with small differences accounted for by the tissue differences and the different environmental temperatures used during the experiments.

DISCUSSION

A theory to explain some of the effects of high-intensity sound on biological tissue has been advanced.

This analysis is based upon the destructive mechanical action of the unidirectional forces that are a concomitant of high amplitude sound waves. Agreement with measurements made on irradiated frog muscle tissue and frog spinal cord tissue is reasonable. The theory was derived with reference to a single particle. It is naturally meant to apply, in terms of observed effects, to a large number of similar particles. Experimental work suitable for verification of the theory thus far has been confined to traveling wave sound fields. Standing wave field experiments would be an important check. The dependency of unidirectional forces upon particle velocity is much different for standing wave fields from that for traveling wave fields and therefore, according to Eq. (10), the threshold required to produce an effect should be much different.

ACKNOWLEDGMENTS

The author wishes to thank Professor W. J. Fry for helpful suggestions on the preparation of the manuscript.

Effects of Noise on the Blood Eosinophil Levels and Adrenals of Mice*

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(Received May 23, 1955)

Physiological changes are described following exposure of mice to single and intermittent noise stimulation (110 db *re* 0.0002 dyne/cm², 10–20 kc) for varying lengths of time. Attention is focused on the degree of adreno-cortical activation as measured by cytological changes in the adrenal gland and fall in the number of circulating eosinophils. Since the observed changes were transient, of short duration and no evidence of systemic pathology could be detected, the noise was described as not harmful. The tendency of certain investigators to regard noise as an injurious, nonspecific stress stimulus without specifying the exact nature of the noise situation does not seem justified.

THERE has been growing interest in possible harmful effects of sound in animals.¹ In a recent study attempts were made to evaluate damaging effects of intense sound independent of the auditory pathway.² The physical or mechanical effects of sound on tissues were found to be negligible (as far as permanent damage to the skin or endocrines was concerned) unless the sound was intense enough to result in the conversion of sound energy into heat at the animal's skin surface.

The present study deals with tissue changes following

exposure of mice to moderately intense sound (approximately 110-db sound pressure level in a 10- to 20-kc frequency band). Several earlier reports suggest that sound may act as a severe stress stimulus in animals, but offer only scant data on the exact degree to which auditory stimulation may be harmful.^{3–7}

For this reason, experiments were set up to determine changes in eosinophils, adrenals and other tissues which may occur following exposure of mice to specified and controlled noise fields. We hoped that these studies would help clarify the extent to which intense noise acts

* Authorized for publication on January 27, 1955 as Paper No. 1944 in the Journal Series of the Pennsylvania Experiment Station. This work was sponsored in part by the Aero Medical Laboratory, Wright-Patterson Air Force Base, Dayton, Ohio under contracts No. AF 33(038)–786 and No. 33(616)–2505. We wish to thank Dr. Hubert Frings for his helpful suggestions during the course of this work and Philip R. Sheridan who measured and monitored the sound fields.

¹ H. O. Parrack, *Noise-Causes, Effects, Measurements, Costs* (School of Public Health, University of Michigan Press, Ann Arbor, 1952), pp. 45–53.

² Adam Anthony and P. A. Danner, (to be published).

³ P. Bugard, *Rapports du II^e Congres Technique National de Securite et d'Hygiene du Travail, La Baule, 1–14, September 27–30, 1951.*

⁴ Bugard, Souvras, Valade, Coste, and Salle, *La Semaine de Hospitaux de Paris*, 29, No. 65, October 26, 1953.

⁵ C. Fortier, *Endocrinology* 49, 782–788 (1951).

⁶ R. Miline and O. Kochak, *Compt. rend. assoc. anatomistes, 38^e Reunion-Nancy, 1–12, March 19–21, 1951.*

⁷ Day, Fletcher, Naimark, and Mosher, *J. Aviation Med.* 22, 316–318 (1951).