Standing Wave Problems in Acoustic Behavioral Testing of Animals

H. van den Ende

Follow this and additional works at: https://scholarlycommons.henryford.com/hfhmedjournal

Part of the Life Sciences Commons, Medical Specialties Commons, and the Public Health Commons

Recommended Citation
Available at: https://scholarlycommons.henryford.com/hfhmedjournal/vol9/iss4/4

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons.
Introduction

Two audiometric testing rooms are presently in use, for acoustic behavioral testing of animals, at the Otological Research Laboratory. Acoustic stimuli consisting of pure tones are introduced by means of loudspeakers.

Speaking of rooms for pure tone audiometry, Beranek remarks, "When audiometers which employ earphones are used, it is usually sufficient to keep ambient noise at a low level, without regard to the nature of the interior of the room. If a loudspeaker is used, however, the interior of the room becomes extremely important, since the presence of sound-reflecting surfaces will materially affect the nature of the sound that reaches the listener's ear. In particular, the level of the sound will vary from point to point in the room, giving rise to "dead spots" and "loud spots". In a companion paper we reported sound pressure level patterns obtained in one of our rooms by means of a microphone traveling in vertical paths. The speaker was thereby driven with a signal of constant frequency and constant amplitude. The pressure from one point to another differed by as much as 30 db. at each of the commonly used test frequencies 250, 1,000, 4,000 and 16,000 c/s. The four walls and ceiling of this testing room were constructed of 4 inch thick acoustic panels, the sound absorption coefficients of which are listed in Table I as specified by the manufacturer.

<table>
<thead>
<tr>
<th>Frequency (Hz)</th>
<th>250</th>
<th>1,000</th>
<th>4,000</th>
<th>16,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absorption Coefficient</td>
<td>0.99</td>
<td>0.99</td>
<td>0.83</td>
<td><strong>n.s.</strong></td>
</tr>
</tbody>
</table>

Table 1

This absorption was not obtained at the floor of the room which was covered with wall to wall carpeting, at a window in one of the walls, or at the margins of the panels. It seems surprising that the above results are compatible with such high absorption coefficients, but less so when it is realized that an absorption coefficient of 0.99 means a pressure amplitude of reflection of 0.1 of the incident sound.

The pattern of loud spots and dead spots shifts with frequency. This means that at a stationary point of observation the sound pressure level will change when the frequency of the driving signal is changed. This condition is undesirable in experiments designed to determine frequency discrimination abilities of animals.

For such experiments the ideal placement of the animal would be in an area where the walls do not reflect sound energy at all. Such an area can be obtained almost perfectly by the installation of wedges and other structures on the walls. The amplitude reflection coefficient then decreases with increasing frequency over a wide range. The frequency at which the amplitude reflection coefficient assumes the
value 0.1 is called “cut-off frequency”. Such a room is then called anechoic (that is without echo) for frequencies above the cut-off frequency. To render our existing facilities (the audiometric testing rooms measure 10 x 10 x 6½ ft.) anechoic for frequencies above 250 cps would require the installation of wedges with a wedge height of one foot. Such an addition was prohibitive because of the cost (estimated total cost per room $3,000). Cut-off frequencies lower than 250 cps would require
Acoustic Testing of Animals

wedge height in excess of one foot which would not be practical. As an experiment, a less costly acoustical treatment was installed in the existing facilities in an attempt to render the room as anechoic as possible for frequencies above 250 cps.

Installation of Acoustic Material

One roll of a flexible, blanket type fiberglas, "Aerocor", 72 inches wide and 100 foot long was acquired. The sound absorption properties of this material are indicated in Table II.

SOUND ABSORPTION COEFFICIENT OF "AEROCOR"

| Aerocor Blanket Material (Owens Corning PF — 399) |
|---------------------------------|----------|
| Thickness: 1 inch               | Density: 2 pcf* |

<table>
<thead>
<tr>
<th>Frequency cps</th>
<th>125</th>
<th>250</th>
<th>500</th>
<th>1,000</th>
<th>2,000</th>
<th>4,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absorption Coefficient</td>
<td>0.10</td>
<td>0.51</td>
<td>0.63</td>
<td>0.94</td>
<td>0.91</td>
<td>0.89</td>
</tr>
</tbody>
</table>

*pcf = pounds per cube foot.

Table II

Studs 2 by 1½ inches were erected 1½ inches from the existing walls without defacing the walls with screws, see Fig. 1. The ¼ inch plywood triangles, with pieces of carton adhered over the points to prevent puncturing of the blanket, permitted folding the blanket to form horizontal wedges as shown in Fig. 1.

The triangles measure 8 inches at the base and were 10 inches high, resulting in a treatment depth of 14 inches.

The ceiling was treated likewise (See Fig. 2.) and a rack with wedges was hung on the door. All area was so treated except for the floor, an opening of 2 by 3 feet to permit viewing through the testroom window, and an opening at the center of the ceiling for illumination.

It took two men two days to install this structure, and the cost of the materials was approximately $200.00.

Method

The arrangement of electrical equipment (for a list of equipment see page 525) is shown in Fig. 3. The motor of the recorder was mechanically coupled to the oscillator so that it would simultaneously drive the frequency dial of the oscillator and the recording paper, both at a predetermined speed. While thus scanning a selected frequency range, the voltage applied to the speaker did not vary appreciably, whereas the sound pressure at the microphone varied considerably.
The experiment consisted of recording the sound pressure variation as a function of frequency, while keeping the microphone stationary 3 feet above the floor and pointing toward the sound source for

a) Bare walls.

b) Aerocor installed on walls and ceiling (as described under Installation of Acoustic Material, see Fig. 1 and 2).

Frequency scanning rather than the method of the traveling microphone (see Ref. 1) was used because the effect of the Aerocor acoustical treatment is much more clearly demonstrated in this manner.

Results

The tracings of Fig. 4 were obtained with an arrangement as shown in Fig. 3. The sound source, an eight inch speaker mounted in a 3/4 inch plywood baffle 23 by 30 inches, was placed on the floor as shown in the situation sketch (see Fig. 4).

The oscillator and amplifier supplied a sinusoidal signal to the speaker with an amplitude of 1 volt and a frequency which was scanned from 200 to 5000 cps in a period of 230 seconds.

A wide band pass filter was inserted in the circuit which allowed frequencies from 75 to 9,600 cps to pass to the recorder. This was necessary to eliminate any low frequency components from distorting the results. The recording paper moved
Acoustic Testing of Animals

at a rate of 3 mm/sec. Fig. 4 compares the recorded sound pressure level before and after installation of the aerocor on the walls.

The upper tracing was obtained before installation of acoustical material whereas the lower tracing portrays the sound pressure level variations with wedged aerocor blanket installed on the 4 walls and ceiling. In both cases the floor was covered with wall to wall carpeting.

In both tracings an irregular line composed of peaks and valleys is seen. These valleys and peaks represent changes in sound pressure levels with changes in frequency in a designated area of the test room as received by the stationary microphone. The lowest point of a valley is designated as a sound pressure "minimum". The number of minima and the depths of the valleys are greater in the untreated room when compared to the acoustically treated room. From this we infer that sound reflections from the surfaces of the acoustically treated room were less than those of the non-treated room.

Tracings were also obtained for frequencies from 5000 to 30,000 cps employing a high frequency speaker with the microphone directed toward and away from the speaker. The results were similar to those of Fig. 4, except for the effect of directivity which the microphone displays at high frequencies.
Conclusions

An audiometric testing room was converted into a semi-anechoic room. This was accomplished by installing on the existing walls and ceiling long wedges of a flexible blanket type fiberglas (Aerocor) 1 inch thick with a density of 2 pounds per cubic foot. Following such treatment, variations in sound pressure levels due to wall reflections were reduced considerably. To what extent the remaining variations are due to wall reflections and to what extent due to speaker characteristics or diffraction phenomena has not been determined.

The author is indebted to Dr. T. M. McGee for helpful discussions and to Mr. L. B. Lowery for his assistance during this investigation.

REFERENCES


7. Thiesen, G. J. (National Research Council, Canada): Personal communication.

List of Equipment

Oscillator: B & K* Type 1013.
Amplifier: McIntosh - Model MC 30.
Vacuum tube Voltmeter: Balantine Model 300.
Speaker: James B. Lansing Type D - 208 mounted on a ¾ inch plywood baffle 23 by 30 inches.
Microphone, Cartridge: B & K Type 4131.
Cathode Follower: B & K Type 2615.
Microphone Amplifier: B & K Type 2603.
Filter: Allison Model 2A.
Sound Level Recorder: B & K Type 2304.
Audiometric Testing Room: Industrial Acoustics Company Model 1200 SP.

*B & K Instruments, Inc.
THE EFFECT OF GROWTH HORMONE ON THE
INCORPORATION OF N\textsuperscript{15} FROM AMMONIUM CITRATE,
GLYCINE, L-ASPARTIC ACID, L-ALANINE AND L-GLUTAMIC
ACID INTO AMINO ACIDS OF LIVER PROTEIN*

TRIESTE G. VITTI

Procedures developed in recently published studies\textsuperscript{1} on the metabolism of N\textsuperscript{15}
from four individual amino acids in untreated and growth hormone-treated hypo­
physectomized rats have been utilized to extend the scope of the former study,
and to carry out a similar one dealing with incorporation of N\textsuperscript{15} from ammonium
citrate into proteins of liver, heart, muscle, kidney, and spleen, and with distribution
of the isotope among nine amino acids and amide nitrogen isolated from liver protein.

One group of seven untreated hypophysectomized rats, and a second group,
similar in size and number, but treated daily for seven days with 100 \textmu g.
intraperitoneal injections of growth hormone, were both given N\textsuperscript{15}-labeled ammonium
citrate containing 0.126 meq N\textsuperscript{15} per 100 g. of body weight, on the fifth experi­
mental day. All animals were sacrificed 48 hours after injection of the isotope.
Proteins from homogenates of liver, heart, kidney, and spleen were precipitated
with TCA, washed free of lipid material with alcohol-ether and also of nucleic
acids with hot TCA\textsuperscript{3}. Quadriceps muscles were processed to yield soluble sarco­
plasmic and myofibrillar protein fractions, and an insoluble collagen-elastin fraction\textsuperscript{3}.
Amide nitrogen and nine amino acids were isolated from hydrolysates of gram­
amounts of TCA-liver protein samples by ion exchange chromatography\textsuperscript{4}. Relative
amounts of N\textsuperscript{15} in nitrogen from protein and amino acid samples were calculated
from 29:28 ratios obtained by mass spectrometry\textsuperscript{5}.

Similar N\textsuperscript{15} analyses were conducted on proteins, and their constituent amino
acids, in liver tissue saved from the four series of animals used in the preceding
study\textsuperscript{1} in which the incorporation of N\textsuperscript{15} from glycine, alanine, aspartic acid, and
 glutamic acid into muscle proteins, and its transfer to their component amino acids,
was investigated. Finally, distribution of N\textsuperscript{15} from alanine among amino acids of
liver protein was determined in control and treated groups of rats sacrificed 24,
48, and 72 hours after administration of the N\textsuperscript{15} compound.

Growth hormone increased the incorporation of N\textsuperscript{15} from ammonium citrate
into proteins of all tissues examined. Increases in weight and nitrogen content in
growth hormone-treated hypophysectomized rats in the ammonium-N\textsuperscript{15} citrate experi­
ment were much smaller for liver than for muscle, but N\textsuperscript{15} incorporation into liver
protein was nearly twice as great as for muscle protein. Utilization of N\textsuperscript{15} from

\*From the Biochemistry Department of the Edsel B. Ford Institute for Medical Research and
the Chemistry Department of Wayne State University. This is an abstract of a thesis presented
to the Graduate School of Wayne State University in partial fulfillment of the requirements
for the degree of Doctor of Philosophy. The degree was conferred in June 1961. The research
work was done in the Biochemistry Department of the Institute under the supervision of Dr.
O. H. Gaebler. This work was supported in part by research grant A-1362-Endo from the
National Institutes of Health, U. S. Public Health Service.

526
ammonium citrate in protein fractions of skeletal muscle was smaller than that previously reported for N\(^{15}\) from glycine, alanine, aspartic acid, and glutamic acid, but was close to that of glutamic acid-N\(^{15}\).

Excepting the ammonium-N\(^{15}\) citrate experiment, the augmenting effect of growth hormone on incorporation of N\(^{15}\) was more striking in muscle than in liver proteins, due to the fact that while proteins in control livers were much more heavily labeled than corresponding control muscle protein fractions, concentrations of isotope in proteins of the two tissues were comparable in treated animals.

Although metabolism of nitrogen from aspartic acid and ammonia has been regarded as quite similar, present experiments demonstrate that the extent of incorporation and distribution of N\(^{15}\) in amino acids of liver protein are more nearly alike when ammonium citrate and glycine are compared as sources of the isotope, than when ammonium-N\(^{15}\) citrate is compared with aspartic-N\(^{15}\).

Growth hormone caused an increased incorporation of N\(^{15}\) from all four of the N\(^{15}\)-labeled amino acids, into arginine of liver protein. This effect was even more evident when ammonium citrate was the source of N\(^{15}\), and obviously offers no support to the idea that interference of growth hormone with urea formation might occur somewhere in the pathway leading to formation of arginine. These findings on arginine, together with the delay in disappearance of N\(^{15}\) from liver protein amino acids in treated animals, support the concept that the overall anabolic effect of growth hormone is due in part to its suppression of protein breakdown to amino acids.

REFERENCES


