VERIFICATION OF SIMMONS AND DIXON'S SUMMATION LOUDNESS DECREMENT PRINCIPLE

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Loudness information is coded by the cochlea and the auditory nerve. This coding is explained on the basis of two operational mechanisms. The first is essentially a *Place Principle,* wherein the nerve fibers excited by outer hair cells require a less intense stimulus than do the. fibers excited by the inner hair cells (Harris, 1953). Traditionally, defects in the coding mechanism have been associated with Fowlers recruitment phenomenon (Simmons and Dixon, 1966). When the more sensitive outer hair cells (or related structures) are damped, auditory threshold is elevated. However, when the undamaged inner hair cells are excited as a function of intensity raise, the resulting loudness sensation eventually equals that of the undamaged ear.

The second mechanism for loudness depends upon a *Summation Principle,* the total number of nerve fibers excited (Harris, 1953, Wever, 1949). More intense sounds excite a larger area of the cochlea and ultimately more nerve fibers. An important feature of this code is its distribution within the cochlea: *(a)* as intensity increases, most of the additional energy is distributed toward the basal end; *(b)* low frequencies spread further than high frequencies. The audiological consequences of these features have been studied in normal and are clinically recognised in masking phenomenon.

To study the consequences of summation loudness defects, Simmons and Dixon (1966) chose two typical unilateral high frequency sensorineural hearing loss cases with following audiometric configurations.

Case 1: Unilateral right ear high frequency sensorineural loss of sudden onset.
A'C Thresholds

	Frequency (in Hz)						
	250	500	1000	1500	2000	4000	8000
Left ear	10	5	5	10	20	5	5 dB HL
Right ear	10	10	10	10	60	95	NR dB HL

Case 2: Right ear Meniere's Syndrome
A/C Thresholds

	Frequency (in Hz)					
	250	500	1000	2000	4000	8000
Left ear	15	10	15	10	10	30 dB HL
Right ear	30	25	15	25	55	60 dB HL

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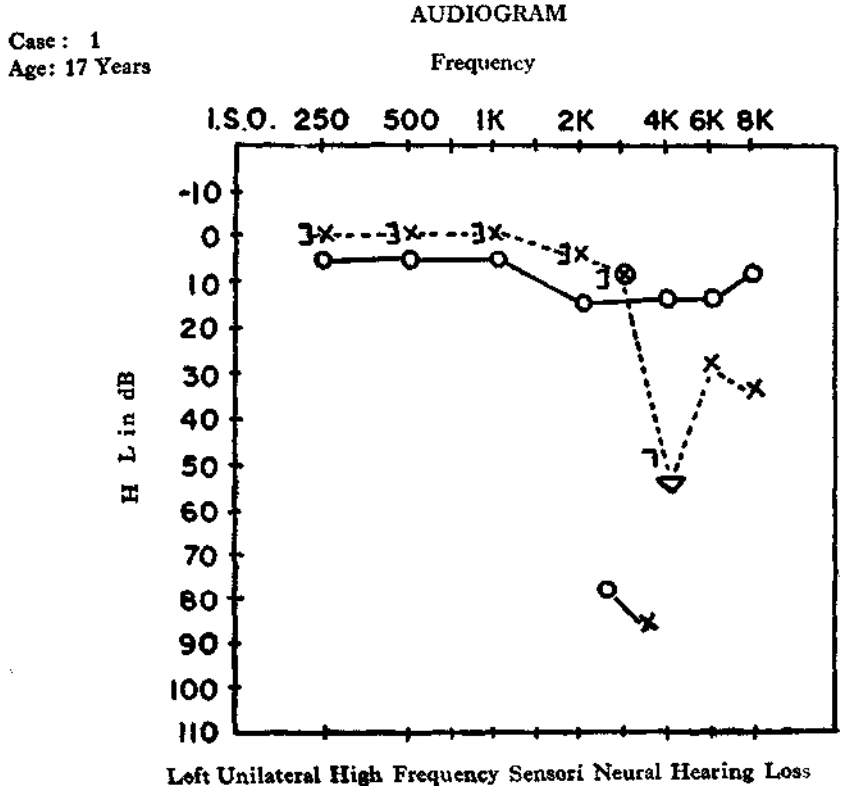
In case 1, when ABLB was administered at 1000 or 1500 Hz where the threshold was normal, an abnormally slow loudness growth occurred in the damaged ear. In case 2, abnormally slow growth of loudness occurred when the test Frequency (1000 Hz) was normal and below the region of the hearing loss.

Simmons and Dixon (1966) stated that case 2 is important because it demonstrates that summation loudness defects are not tied up inexplicably to 8th nerve defects (in pure cochlear portion of the nerve), but can also be observed in classically recruiting ears.

These two cases were selected because both had cochlear losses and because they had the necessary threshold contours for demonstrating the frequency-dependent nature of the summation loudness.

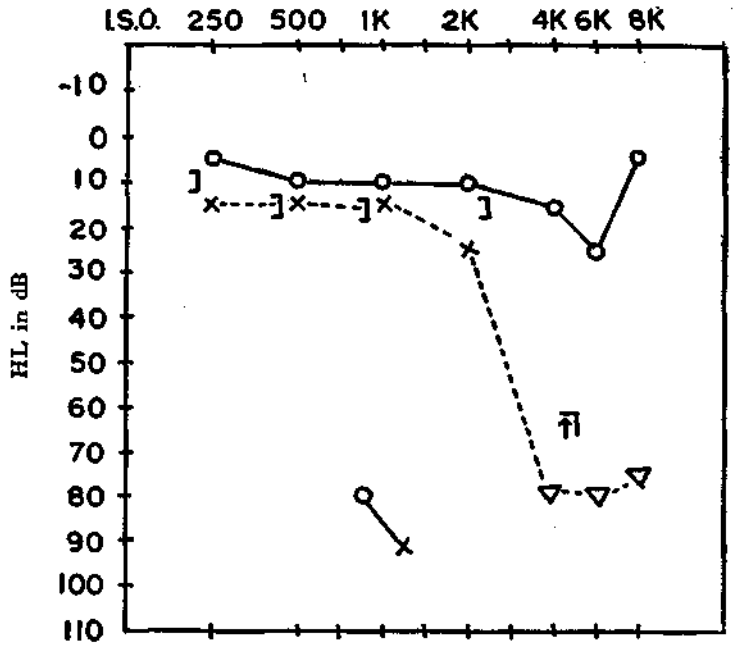
Simmons and Dixon (1966) illustrated that the frequency dependence of summation loudness results from the shape and spread of the cochlear travelling wave. Near threshold, the travelling wave excites neuroepithelial tissue over a small area with low frequencies centred more apically than high frequencies. All frequencies excite about the same number of nerve fibers. As stimulus intensity increases, more and more energy is distributed toward the basal end (Schuknecht, 1960). Thus the additional fibers stimulated as intensity increases will always be those which innervates hair cells whose most sensitive (threshold) frequencies are higher than the frequency of the stimulating tone. If the basal



Left Unilateral High Frequency Sensori Neural Hearing Loss

AUDIOGRAM

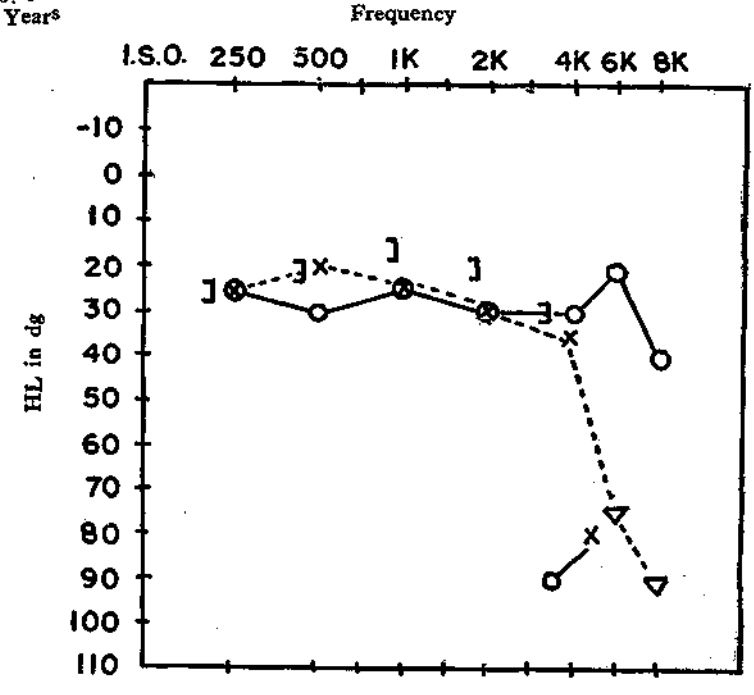
Case No. 2
Age 18 Years



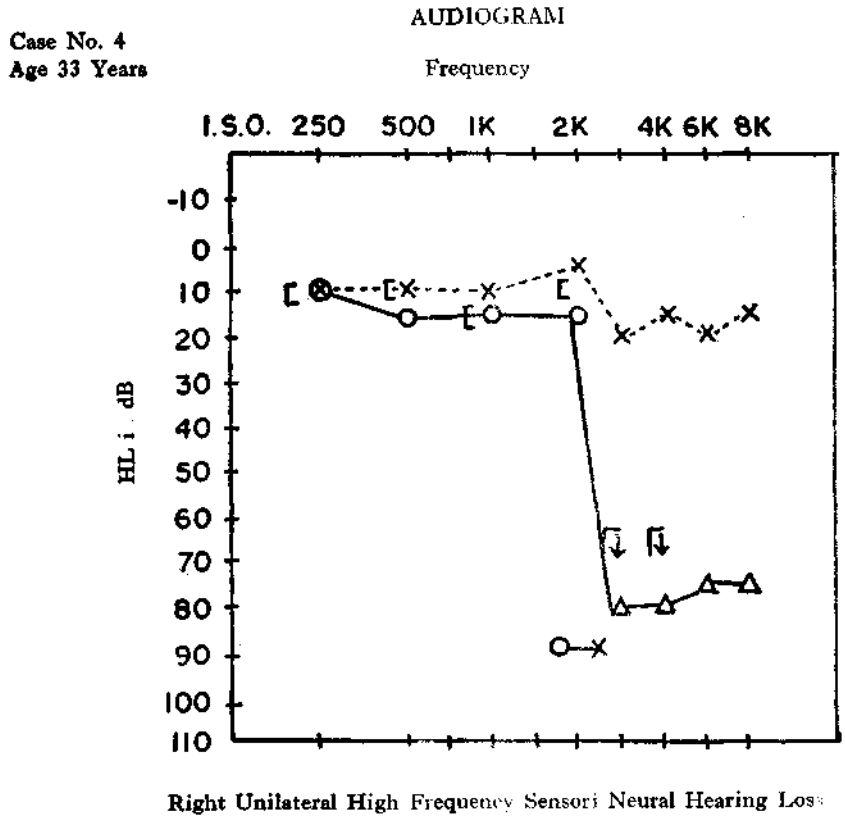
Left Unilateral High Frequency Sensori Neural Hearing Loss

AUDIOGRAM

Case No. 3
Age 42 Years



Left Unilateral High Frequency Sensori Neural Hearing Loss



Right Unilateral High Frequency Sensori Neural Hearing Loss

cochlear tissue (or nerve fibers) responsible for higher frequency is damaged or destroyed, the number of nerve fibers excited will increase at a slower rate than the normal ear (e.g. case 1). Compared to the normal ear, the relative number of discharging fibers decreases as the wave spreads into the damaged region of the cochlea.

According to Simmons and Dixon's (1966) findings in case 1, the growth of loudness does not stop entirely beyond the beginning point of damage (in the vicinity of 1500 Hz in the example). Instead, loudness grows more slowly. A higher intensity is required to gain equal status with the opposite ear. Loudness by the Place Principle (outer, inner hair cells, etc) still functions in non-damaged areas.

In case 2, Place Principle sensitive cells within the damaged region may also have been functioning at high intensities, thus causing loss of loudness decrement (Simmons and Dixon, 1966). A summation loudness decrement can occur in several places, just as long as increasing stimulus intensity leads to spreading of excitation from relatively normal into more damaged regions (Simmons **and** Dixon, 1966).

Methodology

Administering screening ABLB (Tillman, 1969) test for cases with unilateral high frequency sensorineural hearing loss. Thus by finding out the hearing level at which a pure tone in the normal ear sounds equally loud to the reference tone of 90 to 100 dB HL presented to the affected ear. This test was administered at the highest bilateral normal hearing frequency. The interaural intensity difference at the point of balance was determined.

Subjects: Four adult males with unilateral high frequency sensorineural hearing loss served as subjects for this experiment.

Procedure: The following measures were obtained for each subject.

1. Pure tone thresholds of both ears at all audiometric frequencies from 250 Hz to 8000 Hz.
2. Screening ABLB test (Tillman, 1969).

Modified Hughson and Westlake (Jerger and Carhart, 1959) procedure wa8 used for obtaining pure tone thresholds. Screening ABLB as reported in literature was administered. The procedure was as follows:

Instructions to the subjects: ' You are going to hear pure tones in your ears alternately. The tone will be at constant intensity in the poorer ear and the intensity of the tone in the better ear will vary. Hold your right hand (if right ear is poorer) at constant level and vary the height of the left hand (if left ear is normal). If the loudness of the tones in the two ears is equal, hold the two hands at equal level. If the loudness in the left ear (normal) is more, hold the left hand at a higher level than the right hand. If the loudness in the left ear (normal) is less, hold the left hand at a lower level than the right hand.

After giving the instructions, the subjects were asked to repeat the instructions to make sure whether they understood the instructions.

Procedure: The frequency adjacent to the impaired frequency of the affected ear, having normal hearing was chosen for loudness balancing. At about 100 dB HL the tone was presented to the poorer ear. The same tone was presented alternately to the two ears for brief intervals (autopresentation). The intensity of the tone in the better ear was varied until the subject reported equal loudness. The hearing levels at which the subjects reported equal loudness was noted. The experiment was repeated thrice to check the reliability of the loudness judgements.

The interaural intensity difference at threshold and interaural intensity difference at the point of balance were computed. For example,

Interaural intensity difference at threshold=X dB

Interaural intensity difference at the point of balance =Y dB

Y-X>10 dB was considered as an indicator of decruitment.

Equipments used and Calibration

Madsen (Model 4251) audiometer with TDH-39 ear phones calibrated to ISO (1964) standards was used for obtaining thresholds. Beltone 15CX Model equipped with TDH 39 earphones was used for screening ABLB test. The audiometric output was measured using Artificial ear (Bruel and Kjaer Type 4152) with Condensor microphone (Brueland Kjaer Type 4144) and AF Analyse (Bruel and Kjaer Type 2106). Beltone 15 CX Audiometer's output was measured for ABLB setting for each channel. The experiment was conducted in a sound treated room.

Results and Discussion

An attempt is made to verify Simmons and Dixon's (1966) ' Summation Loudness Decrement' principle to explain the phenomenon of decruitment in four unilateral high frequency sensori neural hearing loss cases (see audiograms).

Table I shows the SPL values required to perceive the pure tone equally loudly in both the ears, when screening ABLB was administered at the normal frequency in 4 subjects with unilateral high frequency sensorineural hearing loss. Interaural intensity difference at threshold and at the point of balance are compared and no summation loudness decrement (Decruitment) is observed.

TABLE I

Subjects	Test Frequency in Hz	SPL value in the pathological ear at the point of balance	SPL value in the normal ear at the point balance	Interaural intensity difference at threshold <P-N)*	Interaural intensity difference at the point of balance (P-N)*	(Y-X)	Inter-pretation
1	3000	86 dB	80 dB	0 dB	6 dB	6 dB	Not greater than 10. So no Decruit-ment
2	1000	91.5dB	80 dB	5 dB	11.5 dB	6.5dB	-do -
3	4000	80 dB	90 dB	5 dB	—10 dB	—15 dB	-do-
4	2000	90 dB	90 dB	10 dB	0 dB	—10 dB	-do-

•(P-N)=in (Pathological ear—Normal ear).

The column (Y-X) in Table I gives the difference between the interaural intensity difference at the point of balance and interaural intensity difference at the threshold. Decruitment is said to be present if (Y-X) value exceeds 10 dB. But even the maximum obtained (Y-X) value is 6.5 dB.

The absence of loudneas decruitment in these four typical unilateral high frequency sensorineural hearing loss cases questions the Simmons and Dixon's Summation Loudness Decrement Principle. The present observation refutes the above authors findings. This contradictory observation warrants further experimentation.

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