

# Course of Hearing Recovery According to Frequency in Patients with Acute Acoustic Sensorineural Hearing Loss

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**Abstract:** Through pure-tone audiometry, we studied the course of hearing recovery in 24 ears of 20 men (ages 18–48 years) who had acute acoustic sensorineural hearing loss (ASHL). All subjects were members of the Japanese Self-Defense Force. The hearing level in 5 ears returned to normal, the hearing level of 13 ears recovered but was not within the normal range, and the hearing level of 6 ears was unchanged. The time from noise exposure to presentation was longer in patients with unchanged hearing than in other patients. Recovery of hearing was poorest at 4,000 Hz, followed by 8,000 and 2,000 Hz. We concluded that hearing in patients with acute ASHL is likely to return to normal when the hearing level at 4,000 Hz recovers gradually; partial recovery of hearing is expected when the hearing level at 4,000 Hz reaches an early plateau.

**Key Words:** acute acoustic sensorineural hearing loss; hearing recovery; pure-tone audiometry

Acute acoustic sensorineural hearing loss (ASHL) caused by exposure to extremely loud noises, such as gunshots, and associated with subsequent hearing loss and ear ringing is infrequently encountered in general practice. Because early initiation of treatment may improve or cure such hearing loss, prompt diagnosis and appropriate therapy are imperative [1,2].

Detailed examination of the course of hearing recovery in patients with acute ASHL may improve the accuracy of prognosis. However, few studies have evaluated the process leading to recovery of hearing in such patients. We studied the time course of recovery of pure-tone hearing in members of the Japanese Self-Defense Force who had acute ASHL caused by shooting guns.

## SUBJECTS AND METHODS

From January 1998 through May 2005, we examined the course of hearing recovery by pure-tone audiometry in 24 ears of 20 men (ages 18–48 years; mean age,

26.7 years) who had acute ASHL. Six patients were in their teens, nine were in their twenties, one was in his thirties, and four were in their forties. Most patients were in their early twenties. We confirmed by interview that all patients had shot guns and had no history of ear diseases, such as acoustic trauma or hearing loss. Pure-tone audiometric examinations revealed that hearing levels at all frequencies were within normal range (25 dB HL or less) in the unaffected ear. The causes of hearing loss were gunshots from a rifle in 18 patients, a machine gun in 1, and an air gun in 1. The right ear was affected in 5 patients, the left ear in 11, and both ears in 4. Standard therapy for sudden hearing loss, including adrenocortical hormones (prednisolone, 60 mg), vasodilators (10% low-molecular-weight dextran, 500 ml), and vitamin B<sub>12</sub> (1,500 µg), was given to the patients from the day of presentation. Recovery of hearing was defined as follows: If the hearing level at all frequencies returned to 25 dB HL or less or returned to a level similar to that of the unaffected ear, the patient's hearing loss was evaluated as being "cured"; if the hearing level at one frequency or more returned to 25 dB HL or less or improved 30 dB or more, the patient was said to have "recovered"; if none of the described conditions was met, the evaluation was considered "unchanged."

We graphically depicted the time course of recovery of pure-tone hearing by plotting the number of days

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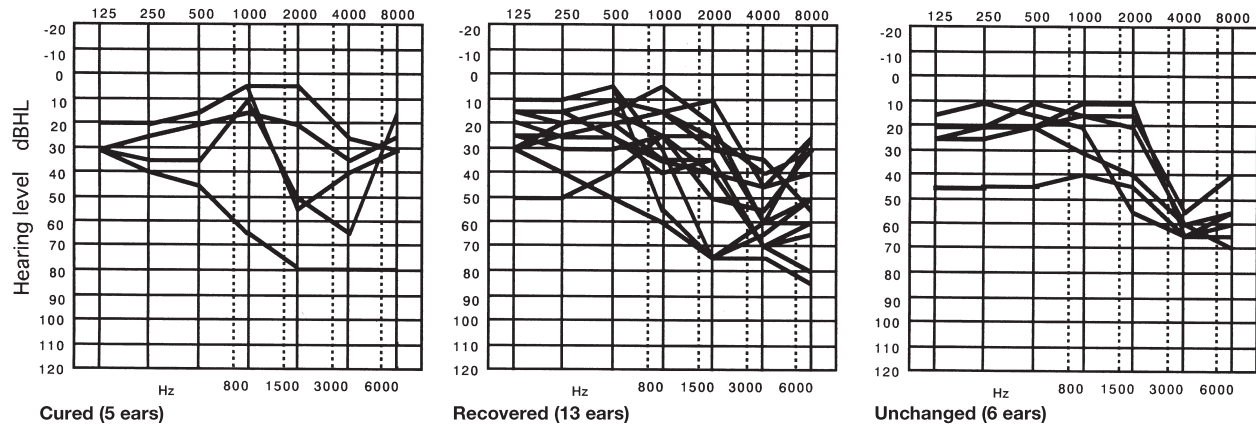


Figure 1. Pure-tone audiometry at presentation. Overlapping audiograms by group—cured, recovered, and unchanged.

after noise exposure on the horizontal axis and the hearing level on the vertical axis. As control, we compared the time course of hearing recovery in our patients with that in 12 patients who were treated between 1998 and 2005 for sudden hearing loss (5 men and 7 women; ages 23–64 years; mean age, 49.7 years).

RESULTS

Among our patients with acute ASHL, evaluation revealed cure in 5 ears, recovery in 13 ears, and unchanged

status in 6 ears. The results of pure-tone audiometry at presentation are shown as overlapping audiograms by group—cured, recovered, and unchanged (Fig. 1). We analyzed hearing level according to frequency with the Kruskal-Wallis test. No significant difference was noted in hearing level according to frequency among the three groups (125 Hz,  $p = .36$ ; 250 Hz,  $p = .42$ ; 500 Hz,  $p = .55$ ; 1,000 Hz,  $p = .24$ ; 2,000 Hz,  $p = .70$ ; 4,000 Hz,  $p = .66$ ; and 8,000 Hz,  $p = .35$ ). We briefly describe two patients from each group below. The time course of recovery of pure-tone hearing is shown in Figure 2.

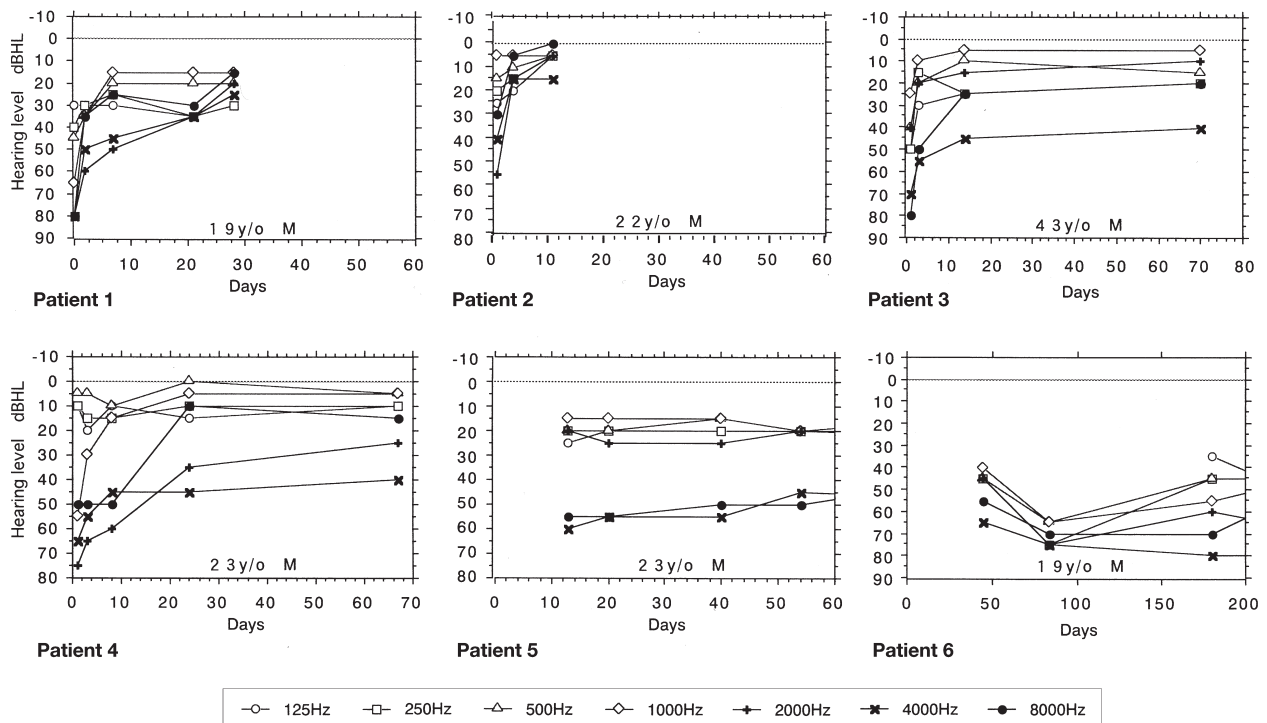


Figure 2. The course of recovery of pure-tone hearing in patients with acute acoustic sensorineural hearing loss. The time course of recovery of pure-tone hearing was graphically depicted by plotting the number of days after noise exposure on the horizontal axis and the hearing level on the vertical axis (two patients from each group).

The number of days after noise exposure is indicated on the horizontal axis.

## Cured Group

### *Patient 1*

A 19-year-old patient experienced ringing in the left ear and hearing loss after shooting a rifle without earplugs on July 29, 1999. He was examined on the same day and was admitted to the hospital because we diagnosed moderate, gradual high-tone sensorineural hearing loss. Vasodilators and steroids were given by continuous intravenous infusion, and vitamin B<sub>12</sub> was given orally.

The patient's hearing improved gradually, but a pure-tone audiometric examination on August 5 revealed that hearing levels at 2,000 and 4,000 Hz had not returned to normal. On August 26, his hearing level returned to normal, and ear ringing disappeared.

### *Patient 2*

A 22-year-old patient experienced ringing in his right ear after shooting a rifle without earplugs on the night of July 8, 2003. We diagnosed abrupt high-tone sensorineural hearing loss (from 2,000 Hz) at presentation on July 9. Steroids and vitamin B<sub>12</sub> were given orally. An audiometric examination on July 12 showed that the hearing level had returned to normal.

## Recovered Group

### *Patient 3*

A 43-year-old man experienced ringing in his right ear and a feeling of ear closure after shooting a rifle without earplugs on June 12, 2000. At presentation the next day, we diagnosed abrupt high-tone sensorineural hearing loss (shown from 2,000 Hz). Vasodilators were given by continuous intravenous infusion, and steroids were given orally. Hearing level at 8,000 Hz was 80 dB HL at presentation but returned to normal in 2 weeks. Hearing level at 4,000 Hz reached a plateau 2 weeks later and then did not recover. Hearing levels at all frequencies except 4,000 Hz returned to normal.

### *Patient 4*

A 23-year-old patient experienced ringing and hearing loss in both ears because his earplugs were dislodged when he had shot a rifle on March 3, 2005. At presentation the next day, we diagnosed moderate, gradual high-tone sensorineural hearing loss (from 1,000 Hz), and he was admitted to the hospital. Vasodilators and steroids were given by continuous intravenous infusion. Hearing levels at 1,000, 2,000, and 8,000 Hz returned to normal some 3 weeks later. However, the hearing level at 4,000 Hz did not improve in either ear after 7 or more days of treatment; hearing loss persisted.

## Unchanged Group

### *Patient 5*

A 23-year-old patient experienced ringing in his left ear after shooting a rifle without earplugs on November 27, 2004. We diagnosed moderate hearing loss at 4,000 and 8,000 Hz at presentation on December 10. Pure-tone hearing was unchanged despite treatment with vitamin B<sub>12</sub>.

### *Patient 6*

A 19-year-old man experienced hearing loss in the left ear and ear ringing after shooting a machine gun without earplugs on August 3, 1998. He presented because of persistent ear ringing on September 17. He was admitted to the hospital because of moderate sensorineural hearing loss. Pure-tone hearing was unchanged despite the continuous intravenous infusion of vasodilators and steroids.

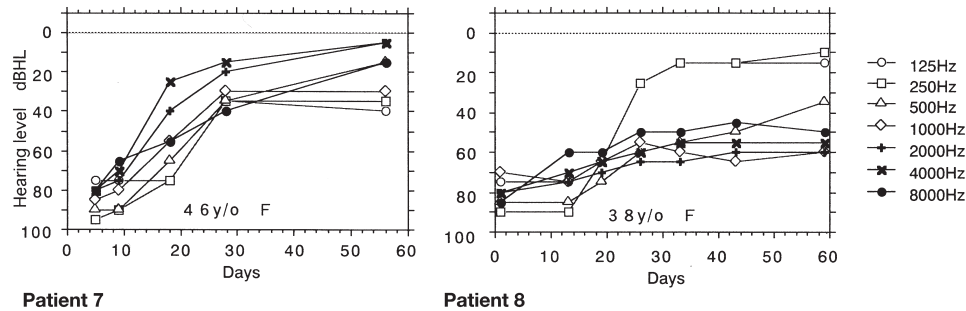
## Comparison of Group Data

Those patients in the cured group were younger (mean age, 20.2 years) than those in the other groups and were treated soon after injury (on average, 3 days after noise exposure). The sources of noise were rifles and an air gun. Earplugs were used in only three of five ears by those in the cured group. The patients in the recovered group were older (mean age, 28.2 years) than those in the cured group. There was no difference in the time from noise exposure to the start of treatment (mean, 4 days) between the recovered group and the cured group. Earplugs were used in only 6 of 13 ears in the recovered group. Of the 13 ears in that group, the number of ears in which hearing returned to normal, according to frequency, was as follows: 125 Hz, six ears; 250 Hz, five ears; 500 Hz, four ears; 1,000 Hz, eight ears; 2,000 Hz, seven ears; 4,000 Hz, one ear; and 8,000 Hz, seven ears.

The number of ears in which hearing did not return to normal, according to frequency, was as follows: 1,000 Hz, 2 ears; 2,000 Hz, 3 ears; 4,000 Hz, 12 ears; and 8,000 Hz, 6 ears. Hearing levels at 125–500 Hz returned to normal in all ears, but the hearing level at 4,000 Hz did not improve in 12 of 13 ears. Those in the unchanged group were older (mean age, 28.6 years) than those in the cured group, and the time from noise exposure to presentation was longer in the unchanged group (mean, 48 days) than in the cured group. Earplugs were used in only two of six ears in the unchanged group. Hearing loss at 4,000 and 8,000 Hz was noted at presentation in all patients in the cured group and did not respond to treatment.

## ASHL Versus Sudden Hearing Loss

Outcomes in patients with sudden hearing loss, classified according to the criteria used to evaluate recovery,



**Figure 3.** The course of recovery of pure-tone hearing in patients with sudden hearing loss. As a control, we compared the time course of hearing recovery in our patients who had sudden hearing loss and were treated between 1991 and 1997 (two patients from the recovered group).

were as follows: cured, three patients; recovered, eight patients; and unchanged, one patient. The course of recovery of pure-tone hearing in two patients from the recovered group is shown in Figure 3.

#### Patient 7

A 46-year-old woman experienced sudden hearing loss in the left ear on June 27, 1998. She presented at our clinic on July 2. Sensorineural hearing loss with a flat audiometric pattern (mean hearing level, 85 dB HL) was diagnosed by quartering. We administered steroids orally and started stellate ganglion blockade. After approximately 1 month, hearing levels improved in all but the low-tone range.

#### Patient 8

A 38-year-old patient experienced a sensation of left-ear obstruction on awakening on the morning of August 16, 2003. She was examined the next day and was given a diagnosis of sensorineural hearing loss with a flat audiometric pattern (mean hearing level in the left ear, 80 dB HL). We treated her on an outpatient basis and administered steroids with stellate ganglion blockade. Hearing at 125 and 250 Hz returned to normal on September 10, and hearing at the other frequencies improved slightly.

Of eight ears in the recovered group of patients with sudden hearing loss, the number of ears in which hearing did not return to normal was as follows, according to frequency: 125 Hz, three ears; 250 Hz, three ears; 500 Hz, three ears; 1,000 Hz, five ears; 2,000 Hz, two ears; 4,000 Hz, six ears; and 8,000 Hz, seven ears. Unlike the action in acute ASHL, hearing levels at some frequencies other than 4,000 Hz did not recover to normal.

## DISCUSSION

Acute ASHL arises from disturbances of the cochlear structure caused by rapid penetration of the inner ear by

extremely loud noises, such as gunshots (peak sound pressure, 150–170 dB; duration, 0.3–0.4 msec; main frequency, 1,000 Hz) [3]. We examined the time course of hearing recovery by means of pure-tone audiometry in 20 patients with acute ASHL. The outcome in our patients was registered as cured in 5 ears (21%), recovered in 13 (54%), and unchanged in 6 (25%). Disturbances of pure-tone hearing were cured in approximately 20% of our patients and recovered partially in 75%.

Pilgramm [4] suggested that in rifle-related injuries, the right ear sustains minimal damage because it is protected by the right forearm, shoulder, and gunstock. We also found that few patients had hearing loss in their right ears.

Cure may be likely in patients who have acute ASHL and mild sensorineural impairment. However, we found at presentation no significant difference in pure-tone hearing at any frequency among patients in the cured, recovered, and unchanged groups (analysis of variance). Two patients did not use earplugs in the cured group. The reason for the absence of significant difference in hearing level at presentation may be the small size of the cured group (five patients). Those in the cured group were younger, and the time until the start of treatment was shorter (mean, 3 days) than in the other groups.

The recovered group (mean age, 28.2 years) was slightly older than the cured group. However, no significant difference between the recovered and cured groups was seen in the time until the start of treatment. Hearing level at 500 Hz or less returned to normal in all 13 ears, but hearing level at 4,000 Hz returned to normal in only 1 of 13 ears. Hearing level at 4,000 Hz reached a plateau after approximately 7 days of treatment and did not return to normal in most patients. Hearing at frequencies other than 4,000 Hz may have undergone a temporary threshold shift (TTS), whereas that at 4,000 Hz may have undergone a permanent threshold shift (PTS). Pure-tone hearing levels at 2,000 and 8,000 Hz returned to normal in some patients whose presentation hearing levels at 2,000 and 8,000 Hz were worse than that at

4,000 Hz. Thus, the presence of severe hearing loss at 4,000 Hz is not likely responsible for the poor response at this frequency.

In the unchanged group, all patients had hearing loss at 4,000 and 8,000 Hz at presentation. Hearing loss at 4,000 Hz did not diminish after treatment in any patient. Most patients were examined a considerable number of days after noise exposure. In the unchanged group, some patients' hearing may have recovered somewhat during the period from noise exposure to presentation. Such patients may have been examined after their hearing level had already become fixed. With the exception of those in the cured group, recovery of hearing level was poorest at 4,000 Hz, followed by 8,000 and 2,000 Hz.

Experimental studies of ASHL in monkeys and chinchillas reported that sensory cells (hair cells) in the basal turn are damaged up to several millimeters from the cochlear window [5,6]. In humans, damage occurs 5–10 mm from the cochlear window [7,8]. These findings could explain the audiometric pattern at presentation (c5dip) and the abrupt onset of high-tone sensorineural hearing loss.

Transmission electron microscopical studies by Libermann and Dodds [9] have shown that shortening of basal rootlets in the cuticula plate in stereocilia initially causes a TTS. As damage intensifies, the basal rootlets are broken, leading to a PTS. Hawkins [10] studied the effects of extremely loud noise on endocochlear direct current potential, which occurs in vascular stria cells. He reported that extremely loud noise causes vasoconstriction, irrespective of noise frequency. Duvall et al. [11] found that blood sludge in cochlear arteries causes microvascular ischemia, resulting in acute ASHL. Depending on the duration of ischemia, either a TTS or a PTS develops.

At the frequency range of 3,000–6,000 Hz, the operating area of the basal plate in the human cochlea is considered to lie at the junction between the proper cochlear artery and the cochlear branch of the vestibulocochlear artery. The vascular changes described earlier and hemodynamic disturbances usually occur at the end of this bifurcation, leading to a dip at 4,000 Hz and delayed recovery of hearing level.

Our results indicate that extremely loud noise caused the microangiopathic and organic disturbances in the cochlea, irrespective of noise frequency. Reversible disturbances resolved gradually, but damage in the operating area around 4,000 Hz was irreversible and persisted.

Unlike the outcome in ASHL, in which only the hearing level at 4,000 Hz did not readily return to normal, hearing levels at all frequencies in patients with sudden hearing loss did not return to normal. The cause of sudden hearing loss is unknown but may be associated with many factors, such as viral infection, circulatory distur-

bances in the inner ear, rhexis of the membranous labyrinth, and metabolic disorders. Thus, various audiometric patterns were seen in patients with sudden hearing loss.

Probst et al. [12] showed that the hearing gain in ASHL patients who received dextran was similar to that of patients who had sudden hearing loss and received dextran. However, those authors measured the mean hearing level and did not evaluate changes in hearing levels at different frequencies.

In conclusion, our results suggest that hearing in patients with acute ASHL is likely to return to normal when the hearing level at 4,000 Hz show signs of gradual improvement. In contrast, only partial recovery is likely when the hearing level at 4,000 Hz reaches an early plateau. Patients who seek medical attention 14 days or more after noise exposure may have fixed hearing levels that are unlikely to recover to normal.

## REFERENCES

1. Harada H, Shiraishi K, Kato T. Prognosis of acute acoustic trauma: A retrospective study using logistic regression analysis. *Auris Nasus Larynx* 28:117–120, 2001.
2. Tsuiki T, Ando K, Jun K M. Treatment of acute hearing loss due to acoustic trauma. *J Otolaringol Jpn* 84:740–748, 1981.
3. Yulikoski J. Audiometric configurations in acute acoustic trauma caused by firearms. *Scand Audiol* 16:115–120, 1987.
4. Pilgramm M. Clinical and animal experiment studies to optimise the therapy for acoustic trauma. *Scand Audiol* 20(suppl 34):103–122, 1991.
5. Hamernik RP, Turrentine G, Robert M. Anatomical correlates of impulse noise-induced mechanical damage in the cochlea. *Hear Res* 13:229–247, 1991.
6. Jordan VM, Pinheiro MI, Jimenez CK. Cochlear pathology in monkeys exposed to impulse noise. *Acta Otolaryngl Suppl* 312:16–30, 1973.
7. Igarashi M, Schuknecht HF, Myers EN. Cochlear pathology in humans with stimulation deafness. *J Laryngol Otol* 78:115–123, 1964.
8. Johnsson LG, Hawkins JE. Degeneration patterns in human ears exposed to noise. *Ann Otol* 85:725–739, 1976.
9. Libermann MC, Dodds LW. Acute ultrastructural changes in acoustic trauma: Serial section reconstruction of stereocilia and cuticular plates. *Hear Res* 26:45–64, 1987.
10. Hawkins JE. The role of vasoconstriction in noise induced hearing loss. *Ann Otol Rhinol Laryngol* 80:903–914, 1971.
11. Duvall A, Ward W, Lanhara K. Stria ultrastructure and vessel transport in acoustic trauma. *Ann Otol Rhinol Laryngol* 83:498–514, 1974.
12. Probst R, Tschopp K, Lüdin E, et al. A randomized, double-blind, placebo-controlled study of dextran/pentoxifylline medication in acute acoustic trauma and sudden hearing loss. *Acta Otolaryngol (Stockh)* 112:435–443, 1992.