# Auditory Hallucinations After Right Temporal Gyri Resection

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*The authors present a case study on the development of auditory hallucinations secondary to right temporal lobe damage. Surgical resection in the study patient was of the right superficial middle and inferior temporal gyri. Carbamazepine at a dosage of 800 mg daily was the most effective medication used. A multidisciplinary approach involving the neurosurgeon, psychiatrist, family, and rehabilitation specialist is necessary in managing the psychiatric sequelae of brain injury.*

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**Case Report**

The patient presented as a 36-year-old, married, father of three children. He had no family or personal psychiatric history.

The patient sustained a right-sided skull fracture, secondary to a fall, and he subsequently required a right temporal craniotomy for a burst temporal lobe, with evacuation of an acute extradural hematoma and subdural hematoma. The middle cerebral veins in the sylvian fissure were coagulated, and the right superficial middle temporal gyri were resected to approximately 4 cm. The inferior temporal gyrus was resected to approximately 6 cm.

The patient was unable to recollect any details of the incident surrounding his head injury. He remained an inpatient in a neurosurgical ward for 8 weeks, postoperatively. During this period, several episodes were noted during which the patient displayed confused, agitated behavior. These were interspersed with lucid periods. The patient expressed fixed ideas, feeling as though he was being "detained by courts for refusing to take part in executions," and he had repeated thoughts of electrocution. The possibility of seizure activity was considered, and he commenced carbamazepine prior to discharge.

During the next few weeks, the patient's mood was labile, but his agitation gradually improved. Carbamazepine was gradually reduced and eventually discontinued after 3 months.

Following the discontinuation of carbamazepine, the patient's mood changed dramatically, varying from crying hysterically to laughing inappropriately. He was commenced on Cipramil. During this period, his wife also noted that he was jerking a lot in his sleep.

One month later, the patient displayed several inappropriate outbursts, during which he was verbally aggressive and threatened to kill himself. During one episode, he threatened to crash the car while his wife was driving and he was in the passenger seat. After this, he informed her that he had heard "a deep male voice" telling him to grab the steering wheel and crash the car. He stated, "They were telling me to do it." On the same day, he shouted to his wife, "Cover your neck!" He later stated that he had heard "vampires telling [him] to bite [her]." These auditory hallucinations persisted for several days, and he was admitted to an acute psychiatric ward. On admission to the ward, he saw several men working on the roof of the building and said that he heard a male voice telling him to go on to the roof and jump.

The patient was commenced on haloperidol on admission. However, there continued to be evidence of auditory hallucinations, and his mood remained labile. On one occasion, he was upset following a disagreement with another patient and reported hearing a voice saying, "Do not take that abuse from anyone; go and sort it out." When he did not act on this, the voice asked, "Why are you not doing what I said?"

As his length of stay in the hospital extended, there were increasingly frequent occasions when he became extremely distressed and agitated. He would hit his own head with his fist and state that he wanted to die. He became violent and attempted to throw furniture

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around the ward. Later he would be unable to recall his actions and would apologize for his behavior. There was no obvious trigger to these outbursts. Following most of these episodes he revealed that he had experienced auditory hallucinations during them.

The patient was subsequently transferred to a specialist unit for further investigation.

Electroencephalogram (EEG) showed a moderate right-sided abnormality. It revealed a responsive symmetrical alpha rhythm at 10–12 Hz of 32–1000 uV amplitude. This was accompanied by similar amplitude slow activity at 1–3 Hz seen only on the right side, maximal in the right mid temporal region. Hyperventilation was performed for 3 minutes and produced increase in the slow activity from the right hemisphere. There were no clearly defined epileptiform discharges observed.

Computed tomography (CT) revealed volume loss within the right anterior temporal lobe with associated dilatation of the right frontal horn. There was also prominence of the cerebrospinal fluid (CSF) space in the floor of the right middle crania fossa and reduction in the volume of the right middle temporal gyrus.

Magnetic resonance imaging (MRI) showed T2 hyperintensity and volume loss around the right temporal lobe and brain stem that were likely to be related to the previous surgery. It also showed further focal signal areas that were likely to be vascular in origin, with marked ischemic damage in the corticospinal tracts.

IQ testing revealed that the patient was functioning in the borderline range of general intellectual ability. The patient had significant difficulty acquiring new information, but once acquired he was able to retain what had been learned.

The patient was difficult to contain in the specialist unit and constantly demanded to go home. This meant that a single photon emission computed tomography (SPECT) test could not be carried out. During his stay, he was thought to have a seizure episode where he lost consciousness for a few seconds and was unresponsive. Postictal prolactin levels were normal, and antipsychotics were gradually withdrawn and eventually stopped.

On return to his original ward, the patient was commenced on carbamazepine. His behavior gradually settled, and there were no aggressive outbursts. He was eventually discharged, and outpatient follow-up for 6 months has revealed remarkable mental stability, with very few further outbursts and auditory hallucinations. He still shows neuropsychiatric cognitive deficits and mild instability of gait.

## DISCUSSION

Traumatic brain injury (TBI) is known to have a wide spectrum of possible consequences that are demonstrated by varying degrees of emotional, intellectual, and behavioral disturbance. Presentations vary greatly and, as in this case, can have elements of disorder of mood, behavior, and psychosis. This can make finding correct and appropriate management difficult.

Four percent to 8.9% of individuals who sustain TBI are reported to develop some degree of psychosis,[1,2] and attempts have been made to identify possible risk factors. Male gender, previous psychological disturbance, family history of psychotic illness, learning difficulties, and premorbid neurological abnormalities are all said to increase the chance of developing postinjury psychotic symptoms.[2,3,4]

Symptoms can range from delusions or hallucinations to expression of odd ideas or aggressive behavior and may or may not be persistent.[5]

Persecutory or paranoid delusions are the most common psychotic symptoms and are said to be present in up to 80% of patients. While auditory hallucinations are observed frequently, visual hallucinations, negative symptoms, and formal thought disorder are rare.[6]

The age at which psychosis secondary to TBI can present is variable, but most studies have shown the mean age to be in the mid-twenties to mid-thirties.[3] The latent period between head injury and onset of symptoms also varies, but research indicates that the mean is approximately 4 years.[1,3,7] Despite this, a substantial number of patients display symptoms within 1 year of injury.[8]

It has also been noted that epilepsy—particularly temporal lobe epilepsy—can present concurrently with psychotic symptoms postbrain injury.[9] The disorder appears to accompany an open-head injury more often than a closed-head injury.[7] Rates range from 9% to 34%.

Research on localizing injuries in patients presenting with psychotic symptoms postbrain injury consistently reports abnormal findings in the temporal lobe.[6,8]

As mentioned, some patients' symptoms may be acute and short-lived, while others develop a more chronic course. Response to treatment is variable. One study.[16] in 2002 reported that neuroleptics were the most commonly used treatment, followed by anticonvulsants and lithium. In general these patients are extremely sensitive to the anticholinergic and sedating properties of medications, and all treatment should be initiated at a low dose and increased gradually.

There have been few randomized control trials of the

neuropsychiatric consequences of a brain injury, and thus several treatments may have to be undertaken before an effective one is found. Anticonvulsants are useful in treating impulsivity, aggression, seizure disorder, and mood lability. What is well established, however, is that management of the psychiatric sequelae of brain injury requires a multidisciplinary approach involving the neurosurgeon, psychiatrist, family, and rehabilitation specialists.[10]

The case presented in this study adds to the literature on development of auditory hallucinations secondary to right temporal lobe damage. It is germane that the surgical resection in this patient was of the right superficial middle and inferior temporal gyri. It is also relevant to note that the most effective medication in this case was carbamazepine at a dosage of 800 mg daily.

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