

What caused George Gershwin's untimely death?

Gregory D Sloop

New Orleans, Louisiana, USA

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I suggest that the noted American composer George Gershwin (1898–1937) died of a pilocytic astrocytoma, not a glioblastoma multiforme, as previously diagnosed. The aetiology of Gershwin's chronic gastrointestinal complaints, long thought to be somatization, is re-examined in this new light.

Case history

On 9 July 1937, George Gershwin presented to Cedars of Lebanon Hospital, Los Angeles, with signs of central herniation¹, including progressive loss of consciousness, small, slightly reactive pupils, loss of upward gaze, and papilloedema with fresh retinal haemorrhages². One month earlier, he had developed right-sided headaches, which awakened him in the morning. He was initially hospitalized on 23 June, at which time a neurological consultant noted right-sided hypsomnia³ and photophobia². No papilloedema was seen. Gershwin refused lumbar puncture, which then was important in the evaluation of suspected brain tumours. Despite the incomplete work-up, the discharge diagnosis was "most likely hysteria"², the condition now known as somatization disorder⁴.

Gershwin's initial symptom of central nervous system disease was an olfactory hallucination in January 1934, three and a half years before his death⁵. Additional episodes, described as an unpleasant smell of burning rubber, occurred in February and April 1937. Unlike the initial episode, these were associated with altered consciousness.

Near the end of his life, Gershwin experienced two episodes that were probably complex partial temporal lobe seizures⁶. These can present as impaired consciousness associated with involuntary motor activity, known as automatisms. Reactive automatisms are provoked by external

stimuli, and may be inappropriate and disturbing to others⁷. In the first episode, Gershwin opened the driver's-side door of a moving car and attempted to push out his chauffeur. Asked why he did this, Gershwin replied, "I don't know." In the second, in response to the gift of a box of chocolates, Gershwin crushed the contents into a mass, which he smeared over his body.

On his final admission, lumbar puncture revealed clear fluid with an opening pressure of 400 mmHg². With the history of olfactory hallucination³, these findings suggested a temporal lobe tumour to the attending neurosurgeon, Dr Carl Rand. Gershwin then underwent ventriculography, which showed both lateral ventricles were displaced to the left, and the temporal horn of the right ventricle did not fill, confirming a right temporal lobe tumour⁸. Subsequently, a gliomatous cyst of the right temporal lobe, which contained an ounce of dark yellow fluid, was removed². The cyst wall was smooth, except for a mural nodule on the medial side. Postoperatively, Gershwin's temperature rose rapidly to 106.5°F, pulse to 180 beats per minute, and respirations to 45 per minute. He died hours later, without having regained consciousness, at 10.35 a.m. on 11 July 1937.

According to the microscopic description of 1937, Gershwin's tumour was composed of loosely placed small cells with small, deeply stained nuclei, and fine cytoplasmic processes that connected with those of adjoining cells. Marked variation in nuclear size and occasional multinucleated cells were noted. No mention was made of necrosis, endothelial proliferation, or characteristic mitotic activity. The tumour was diagnosed as "spongioblastoma multiforme", the tumour today known as glioblastoma multiforme².

Re-evaluation of the diagnosis

The differential diagnosis of cystic neoplasia in the brain is limited⁹. The differential diagnosis of cysts with mural nodules includes pleomorphic xanthoastrocytoma, ganglioglioma, pilocytic astrocytoma, and haemangioblastoma. Only the first three are likely in the cerebrum. Examination of

Correspondence to: Gregory D Sloop MD, Department of Pathology, Louisiana State University School of Medicine in New Orleans, 1901 Perdido Street, New Orleans, LA 70112, USA (E-mail: gsloop@lsuhsc.edu)

the three previously published photomicrographs of Gershwin's tumour^{2,10}, and a fourth, previously unpublished photomicrograph (Figure 1), reveals a tumour composed of spindle cells with small, moderately pleomorphic, hyperchromatic nuclei in a background of delicate, hair-like cytoplasmic processes. No mitoses or necrosis are identified. The absence of xanthomatous change or neuronal differentiation weighs against pleomorphic xanthoastrocytoma or ganglioglioma, respectively. The hair-like cytoplasmic processes, distinctive gross tumour morphology, and long clinical history suggest Gershwin's tumour was a pilocytic astrocytoma, the lowest-grade astrocytoma¹¹. Cellular pleomorphism is present in both pilocytic astrocytoma and glioblastoma multiforme, and may have led to the mistaken diagnosis, which has been accepted for many years. However, Gershwin's three-and-a-half-year history of neurological symptoms is not consistent with a high-grade tumour. Based on the long clinical history and pathological findings, malignant glioma is virtually eliminated from the differential diagnosis.

Biology of pilocytic astrocytoma

Because it is not known how thoroughly Gershwin's tumour was examined histopathologically, focal malignant degeneration, although rare in pilocytic astrocytoma⁹, cannot be excluded. Even without malignant degeneration, cystic enlargement of the tumour, a common occurrence in pilocytic astrocytoma¹², could have caused Gershwin's clinical deterioration. The temporal lobe is the most common supratentorial site of pilocytic astrocytoma^{11,13}. The most common symptoms are seizures and headache^{13,14}. Mean age at the onset of symptoms is reported to be 22 years¹⁴. An exceptionally long duration of symptoms before diagnosis is noted occasionally. A duration of 30.5 years has been reported in association with a supratentorial pilocytic astrocytoma¹³. Durations of 38 years to at least 55 years have been reported with cerebellar examples¹⁵. Limited data suggest the biological behaviour of supratentorial and cerebellar pilocytic astrocytomas is similar¹¹.

Re-evaluation of Gershwin's gastrointestinal complaints

Beginning at age 23, Gershwin suffered from occasional attacks of vague stomach pain and constipation. After multiple normal physical examinations, these were attributed to somatization, the process in which unexplained or amplified symptoms are linked to psychological factors or conflicts¹⁶. Gershwin's symptoms do not fulfil modern criteria for somatization disorder⁴. However, somatization can exist by itself as a

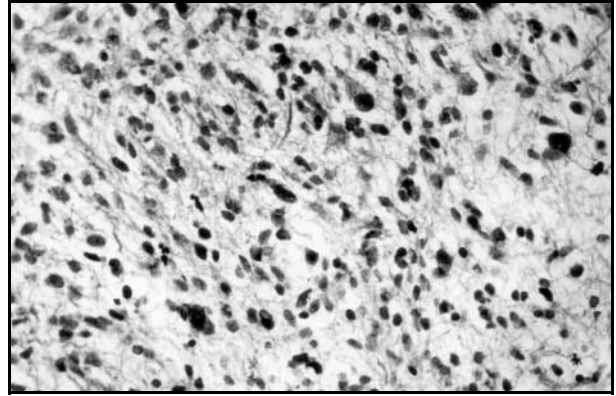


Figure 1. Photomicrograph of Gershwin's glioma (probably haematoxylin-eosin stained, original magnification unknown).

common, less severe expression of somatization disorder¹⁷.

Gershwin's case is not a classic example of somatization for several reasons. A childhood history of abuse, a dysfunctional or substance-abusing family background, or a work history characterized by excessive disruptions because of medical problems and interpersonal difficulties is common in somatization disorder¹⁸. None was present in Gershwin's case. Patients with somatization disorder can exhibit histrionic or overly dramatic behaviour¹⁸. The depiction of Gershwin by noted drama critic Alexander Woollcott, written in 1933, suggests the opposite:

The first time I ever met George Gershwin, he came to dine with me at my hotel in Atlantic City.... He began by apologizing for the eccentric dinner he would have to order. "You see," he explained, "I have terrible trouble with my stomach."

...Like you and me, Master Gershwin was profoundly interested in himself, but unlike most of us he had no habit of pretense. He was beyond, and, to my notion, above, posing. He said exactly what he thought, without window dressing it to make an impression, favorable or otherwise.... Well, if I...could have but one adjective for George Gershwin, that adjective would be "ingenuous."¹⁹

Several other observations argue against somatization in Gershwin's case. Somatization is more common in women⁴. Gershwin received no secondary benefit from the sick role. Some even scoffed at his complaints, believing them to be manufactured to receive attention²⁰. In fact, Gershwin reportedly said, "No one believes me when I say I'm sick."³ Furthermore, Gershwin gave up a long-term addiction to tobacco in 1931, trying to find relief. The sacrifice and emotional resources needed to effect such a positive change seem unusual for a patient with prominent somatic complaints. Gershwin also manipulated his diet and kept a diary of his food intake, in the vain hope that he could identify the cause of his gastrointestinal pain. Finally, unlike typical patients with somatization disorders¹⁶, Gershwin sought psychiatric therapy. It is noteworthy that men diagnosed with somatization disorder are more

likely later to be diagnosed with a medical disorder that explains the symptoms¹⁸.

A primary gastrointestinal illness should also be considered in explaining Gershwin's gastrointestinal complaints. However, common, objective evidence of gastrointestinal pathology, such as gastrointestinal bleeding or weight loss, is not mentioned in biographical material about Gershwin. Indeed, his symptoms might not have been diagnosed as somatization given objective evidence of gastrointestinal pathology. This absence of evidence argues against peptic ulcer disease, diverticulitis, inflammatory bowel disease, and some infections. Further, many of these entities were well known in Gershwin's day, and could be diagnosed. The long duration of symptoms weighs against a progressive disease, which might have "declared itself" over time. Finally, Gershwin's dietary experiments could have uncovered lactose intolerance.

In this setting, an unusual presentation of a non-gastrointestinal illness should also be considered. Simple partial temporal lobe seizures can present as olfactory or autonomic symptoms, including abdominal discomfort⁷. With an indolent tumour such as pilocytic astrocytoma, seizures could have been present for years. Thus, Gershwin's gastrointestinal symptoms could have been simple partial temporal lobe seizures, as originally suggested by Ljunggren⁸. Supporting this possibility, Gershwin definitely experienced one type of simple partial temporal lobe seizure, olfactory hallucinations. Also, explaining Gershwin's neurological and gastrointestinal symptoms with a single diagnosis satisfies the principle of simplicity (Occam's razor). Nevertheless, a combination of pilocytic astrocytoma and either somatization or undiagnosed gastrointestinal illness is possible, and it is not possible today to determine the cause of Gershwin's gastrointestinal illness with certainty.

Conclusions

Modern neuroimaging would have allowed identification of Gershwin's tumour earlier, when it could have been safely removed. However, even in 1937, Gershwin's prognosis might have been good if he had been treated before herniation. In a series of 42 patients with cerebral pilocytic astrocytomas treated in Sweden between 1926 and 1957²¹, only six died of their tumour or complications of surgery. Of the 34 patients alive at the time of the report, 27 had full capacity for work, three had partial capacity for work but were self-supporting, and four were completely disabled. Twenty-seven subjects had been observed for more than 10 years, and 19 more than 20 years.

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