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*AJNR Am J Neuroradiol* 1983, 4 (3) 446-449

<http://www.ajnr.org/content/4/3/446>

This information is current as  
of September 13, 2025.

# Evaluation of the Brainstem with High-Resolution CT in Cerebellar Atrophic Processes

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The authors studied the usefulness of computed tomography (CT) for evaluation of the brainstem in cerebellar atrophic processes. Twenty adult subjects without posterior fossa lesion were used for normal CT measurements of the brainstem. The measured values with CT corresponded to those with pneumotomography. Also reviewed were 49 patients with cerebellar atrophy which included spinocerebellar degeneration (25 patients), Shy-Drager syndrome (five), progressive supranuclear palsy (three), chronic phenytoin usage (10), and chronic alcoholism (six). All but the chronic alcoholism group showed atrophy of the brainstem at all locations of measurement when compared with normal controls ( $p < 0.05$ ). In addition, the patients with progressive supranuclear palsy had significantly more pronounced midbrain atrophy. In the chronic alcoholism group the measurements of the brachium pontis, the medulla, and the fourth ventricle differed significantly from those of normal controls ( $p < 0.05$ ).

Before the introduction of computed tomography (CT), patients suspected of cerebellar atrophy were subjected to pneumoencephalography (PEG) [1–4]. CT is accepted as a valuable procedure for evaluation of cerebellar atrophy [5–7]. The brainstem evaluation has been performed mostly with PEG or CT cisternography [8, 9], but analysis of atrophy of the brainstem with plain CT has not been attempted systematically. The authors discuss the usefulness of CT with sagittal and coronal reconstruction for evaluation of the brainstem in cerebellar atrophic processes. An attempt is also made to establish metric criteria for determining the normality of the brainstem.

## Subjects and Methods

CT was performed with the Siemens Somatom 2, using a pulsed x-ray source and 520 cesium-iodide detectors that rotate through 360°, and an image monitor with a matrix of 256 × 256. A zoom factor of 4 was used, so each pixel represented about 0.5 × 0.5 mm of the object. Collimation allowed us to obtain contiguous slices 2 mm in thickness, but in a few cases the 4 mm slice mode was selected. These CT techniques furnished sufficient data to permit coronal and sagittal reconstruction. The scanning plane was parallel to the orbitomeatal line.

The CT criteria for the evaluation of cerebellar atrophy are similar to those that have been identified by PEG and include: (1) enlargement of the cerebellar sulci, (2) enlargement of the cerebellopontine cisterns, (3) enlargement of the superior cerebellar cistern, and (4) enlargement of the fourth ventricle [5].

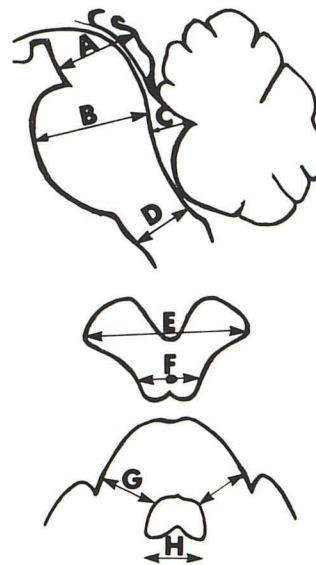


Fig. 1.—Normal brainstem measurements. Mean measurements (in mm) ± SD were: A, 17.5 ± 1.2; B, 22.3 ± 1.8; C, 10.8 ± 0.9; D, 13.1 ± 1.2; E, 34.8 ± 2.2; F, 17.6 ± 1.4; G, 16.5 ± 1.1; and H, 14.7 ± 2.7.

TABLE 1: Cases of Brainstem Measurements with CT

| Clinical Group                 | No. of Cases | Gender | Age (years) |         |
|--------------------------------|--------------|--------|-------------|---------|
|                                |              | M:F    | Range       | Average |
| Normal controls                | 20           | 12:8   | 30–60       | 47      |
| Spinocerebellar degeneration   | 25           | 14:11  | 20–72       | 50      |
| Shy-Drager syndrome            | 5            | 4:1    | 36–55       | 46      |
| Progressive supranuclear palsy | 3            | 2:1    | 51–67       | 60      |
| Chronic phenytoin usage        | 10           | 4:6    | 11–58       | 34      |
| Chronic alcoholism             | 6            | 5:1    | 25–63       | 49      |

Note.—Duration of phenytoin use was 8–32 years (average, 19 years); duration of alcoholism was 6–30 years (average, 18 years).

Twenty patients without posterior fossa lesions, ranging in age from 30–60 years (average, 47 years), were used as controls. Forty-nine patients with cerebellar atrophy on CT were also reviewed. Patients with an acquired lesion known to cause cerebellar atrophy (such as operative defects and vascular lesions of cerebellar hemispheres) were excluded. Brainstem measurements were made using a light-pen on the viewing console. From axial images,

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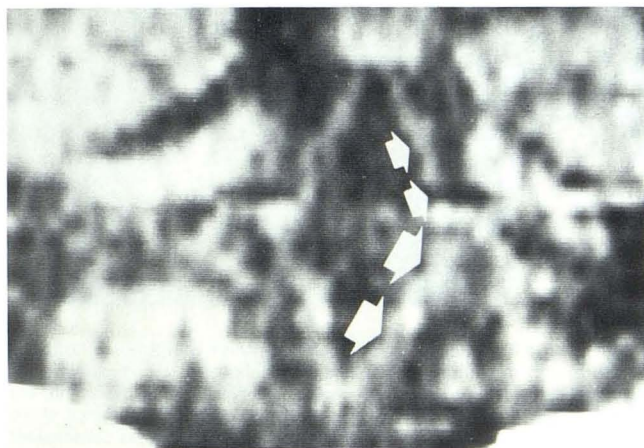
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TABLE 2: Brainstem Measurements (Mean  $\pm$  SD in mm)

| Clinical Group                           | Midbrain              |                   |                 | Pons            |                | Medulla        | Fourth Ventricle |                |
|------------------------------------------|-----------------------|-------------------|-----------------|-----------------|----------------|----------------|------------------|----------------|
|                                          | Transverse Peduncular | Transverse Tectal | AP              | Brachium Pontis | AP             | AP             | Height           | Width          |
| Normal controls . . . . .                | 34.8 $\pm$ 2.2        | 17.6 $\pm$ 1.4    | 17.5 $\pm$ 1.2  | 16.5 $\pm$ 1.1  | 22.3 $\pm$ 1.8 | 13.1 $\pm$ 1.2 | 10.8 $\pm$ 0.9   | 14.7 $\pm$ 2.7 |
| Spinocerebellar degeneration . . . . .   | 31.4 $\pm$ 2.7        | 15.8 $\pm$ 1.4    | 14.5 $\pm$ 1.2  | 12.0 $\pm$ 2.2  | 18.6 $\pm$ 2.0 | 10.5 $\pm$ 1.2 | 13.8 $\pm$ 2.3   | 19.5 $\pm$ 3.6 |
| Shy-Drager syndrome . . . . .            | 30.8 $\pm$ 4.6        | 15.8 $\pm$ 2.3    | 14.0 $\pm$ 2.6  | 11.5 $\pm$ 3.8  | 17.4 $\pm$ 3.7 | 9.6 $\pm$ 1.4  | 13.2 $\pm$ 2.2   | 22.3 $\pm$ 0.6 |
| Progressive supranuclear palsy . . . . . | 31.0 $\pm$ 1.4        | 13.0 $\pm$ 1.6    | 13.0 $\pm$ 0.8  | 12.3 $\pm$ 1.0  | 18.7 $\pm$ 1.0 | 10.3 $\pm$ 0.6 | 13.3 $\pm$ 0.6   | 19.0 $\pm$ 1.0 |
| Chronic phenytoin usage . . . . .        | 30.8 $\pm$ 2.1        | 15.8 $\pm$ 1.4    | 15.6 $\pm$ 1.6  | 13.5 $\pm$ 1.5  | 19.9 $\pm$ 1.7 | 11.1 $\pm$ 1.2 | 13.9 $\pm$ 1.9   | 19.9 $\pm$ 2.8 |
| Chronic alcoholism . . . . .             | 33.4 $\pm$ 2.6        | 15.8 $\pm$ 1.3    | 16.0 $\pm$ 1.14 | 13.6 $\pm$ 1.5  | 21.4 $\pm$ 0.6 | 11.0 $\pm$ 1.2 | 13.8 $\pm$ 2.4   | 20.0 $\pm$ 2.0 |



A



B

Fig. 2.—Shy-Drager syndrome in 59-year-old man with 10 year history of severe orthostatic hypotension and impotence. A, Midsagittal reconstructed image revealed very marked atrophy of midbrain, pons, medulla, and cerebellum. B, Coronal view. Widened fourth ventricle with atrophy of superior (small arrows) and inferior (large arrows) cerebellar peduncles.

transverse measurements of the cerebral peduncles, the tectum, the fourth ventricle, and the brachium pontis were made. From reconstructed sagittal images, anteroposterior (AP) measurements of the midbrain, the pons, the medulla, and the fourth ventricle were made (fig. 1). Statistical analysis was performed using the Student *t* test.



Fig. 3.—Spinocerebellar degeneration in 39-year-old man with 6 years of ataxic gait disturbance; impaired speech characterized by dysphonia, dysarthria, and perseveration; and a positive family history of olivopontocerebellar atrophy. Reconstructed midsagittal image revealed evidence of pontine atrophy as well as vermian atrophy.

## Results

From the etiology of cerebellar atrophy, 49 patients could be classified into five groups (table 1). Measurements of the brainstem in normal controls and cerebellar atrophic processes are listed in table 2.

All but the chronic alcoholism group showed atrophy of the brainstem at all locations of the measurements ( $p < 0.05$ ). Patients with Shy-Drager syndrome (fig. 2) showed somewhat more severe atrophy of the cerebellum than the other groups, but measurements of their brainstems did not differ significantly from those of spinocerebellar degeneration (fig. 3).

Compared with the rest of the series, patients with progressive supranuclear palsy showed significantly more severe atrophy in the midbrain, except for the cerebral peduncles (figs. 4A and 4B). One of three patients with supranuclear palsy died of pneumonia several months after CT examination. The midsagittal section of this autopsy specimen showed a good correspondence with CT (fig. 4C).

In chronic phenytoin usage, all patients had a long history of phenytoin treatment (of 8–32 years; average, 19 years). All of the measurements of their brainstems differed from those of normal controls. Seven of them showed some cerebellar signs. No specific anatomic pattern was evident to correlate with clinical findings. Compared with the other groups, however, many patients with chronic phenytoin usage revealed prominent atrophy in the posterior and inferior aspects of the vermis and the hemispheres. The atrophy of the posterior vermis was equal to that of the anterior vermis in three cases and more severe in four cases. Eight of the ten showed a mild to moderate degree of cerebral atrophy.

In the chronic alcoholism group, AP diameters of the midbrain

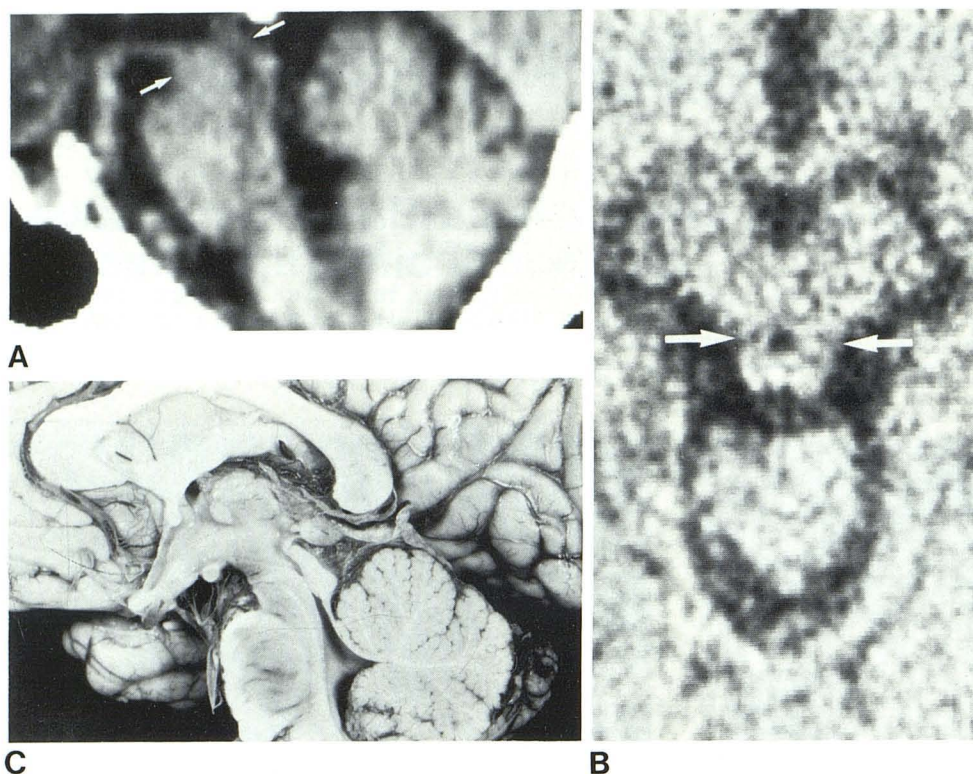


Fig. 4.—Progressive supranuclear palsy in 68-year-old man with progressive diplopia, dysarthria, and mental deterioration. He had difficulty walking, and upward and downward gaze were severely restricted, although doll's-head eye movements were preserved. He also showed masked face, bradykinesia, and neck and upper trunk rigidity. A, CT revealed atrophy of the midbrain (arrows) and cerebellum. Superior colliculi are probably smaller than inferior colliculi. B, Axial image. Marked atrophy of tectum of midbrain (arrows). Enlargement of aqueduct and quadrigeminal and ambient cisterns. C, Died of pneumonia several months after CT. Midsagittal section of brain showed good correlation with CT images.

and the pons did not differ from normal controls, but their measurements of the brachium pontis, the medulla, and the fourth ventricle differed significantly ( $p < 0.05$ ). There was a known 6–30 year history of alcoholism in six cases. All but one showed some cerebellar signs such as tremor and ataxia of stance and gait. All patients showed relatively greater midline cerebellar atrophy as the most prominent CT feature (fig. 5). All six cases had some degree of supratentorial cortical atrophy as well.

## Discussion

Our measurements of the brainstem led to some conclusions that differ from previous reports. Measurement on a reconstructed sagittal image seems to have the advantage of less influence of scan angulation. Recently Steele and Hoffman [9] reported brainstem evaluation with CT cisternography. Their normal brainstem measurements showed a rather large standard deviation. They measured from hard copy and corrected for minification. Their transverse diameter for the cerebral peduncles was much smaller than ours. As they stated, this diameter tends to decrease as the peduncles descend to the pons. We measured this diameter at a higher level than they did.

As for the height of the fourth ventricle, Amundsen and Grimsrud [10] reported its mean value as 12.5 mm with 1 SD, 1.14 mm in normal PEG. But there are changes in the ventricular size during PEG. Lim et al. [11] reported the average increase in the height of the fourth ventricle between the first and last fraction of air was 2.5 mm. Considering this change, our normal measurements correspond well with those with PEG.

Although CT cisternography with metrizamide depicts the brainstem and cerebellum quite well, we believe that CT cisternography is not necessary to evaluate atrophy of the brainstem, nor are air



Fig. 5.—Chronic alcoholism in 55-year-old man with 30 year history of excessive drinking, during which time he reached a reduced mental state. Neurologically he showed a marked tremor, cerebellar ataxia, dysarthria, and gait disturbance. CT revealed atrophy of vermis and hemispheres of the cerebellum. Anterior and superior aspects of vermis showed conspicuous atrophy, depicted well in this midsagittal reconstructed image.

studies. In the patients with atrophic diseases, widened subarachnoid space serves to delineate surrounding structures.

From the pathogenesis, brainstem atrophy in the degenerative groups is considered as primary neuronal degeneration. All patients with these degenerative diseases showed some cerebellar signs. While the overall estimates of the brainstem measurements for the spinocerebellar degeneration group differ from the controls, they do not differ from the other types of cerebellar atrophic processes.

Patients with Shy-Drager syndrome showed somewhat more severe atrophy of the cerebellum, but measurements of their brainstems did not differ significantly from those of spinocerebellar degeneration. Patients with progressive supranuclear palsy showed atrophy of the brainstem, cerebellum, and cerebral hemispheres on CT. Also, the atrophy of the midbrain (excepting cerebral peduncles) was very marked for this group, and differed significantly from other groups of cerebellar atrophy ( $p < 0.05$ ). This finding corresponds with the previous reports by other authors [8, 12].

In chronic phenytoin usage, the mechanism of the cerebellar syndrome is controversial, because it is difficult to separate the effect of phenytoin from the cumulative effect of anoxia occurring with repeated convulsions [13–15]. Both are thought to play a role. Iivanainen et al. [16] reported that PEG examination revealed atrophy of the brainstem or cerebellum or both in 36 of 131 phenytoin-treated mentally retarded epileptics. McLain et al. [17] reported five patients who developed cerebellar degeneration and irreversible cerebellar symptoms while taking phenytoin. In contrast to that report, Koller et al. [6] reported that all eight patients they studied had normal cerebellar function despite the existence of cerebellar atrophy on CT. They concluded that cerebellar degeneration on CT caused by phenytoin may be observed while patients are asymptomatic, which indicates that CT may be helpful in the preclinical detection of phenytoin-induced cerebellar degenerations.

The relatively pronounced midline cerebellar atrophy on CT (which characterizes most of our patients with chronic alcoholism) and other cerebellar signs are consistent with known histopathology, with the degeneration of all neurocellular elements of the cerebellar cortex (particularly of the Purkinje cells), and strikingly restricted to the anterior lobe and the superior vermis [18]. Selective midline cerebellar atrophy on CT in alcoholism has been observed by others [5–7]; however, they did not comment on the brainstem. In their classic study of cerebellar cortical degeneration in alcoholic patients, Victor et al. [18] autopsied 11 cases and found that the olivary nuclei were almost always involved. This finding may well explain the atrophy of the brainstem we have reported in the chronic alcoholism group.

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