

Subcortical Anarthria: A Case Report

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A 56-year-old right-handed male with a history of hypertension and diabetes presented two episodes of stroke: The first affected territory was the left anterior coroidal artery (capsular and paracapsular infarcts at the level of the genu and posterior arm of the internal capsule) and the second was the right thalamus, due to a hematoma. Following the first stroke, the patient developed severe dysarthria and after the second stroke remained anarthric. The pathophysiology of the disorder is discussed, and the role of the left and right thalamus as far as speech is concerned is reviewed. © 2001 Academic Press

INTRODUCTION

From the time that Pierre Marie described subcortical aphasias, in 1906, many doubts have been raised regarding the participation of subcortical white matter, the striatum, and the thalamus in the mechanisms of speech. In our clinical practice we attended a patient with a double subcortical lesion. Initially there was an infarct on the left side and later there was a hemorrhage on the right side so that our patient first presented dysarthria and then anarthria. In this article, our patient's case is described and relevant comments are made.

CASE DESCRIPTION

A 56-year-old, right-handed, hypertensive, diabetic male was admitted to the Lesit Clinic, Buenos Aires, Argentina in May, 1997. He had developed a right facio-brachio-crural hemiplegia with severe dysarthria, secondary to an infarct in the anterior coroidal territory, involving the left capsular region at the genu and posterior arm, in addition to the adjacent portion of the globus pallidus (Fig. 1).

Two months later the neurological picture worsened with the appearance of anarthria as a result of a right thalamic hematoma involving the thalamic mass but sparing the pulvinar area, with ventricular leakage signs disclosed by a CT (computed tomography) of the brain at that time (Fig. 2). Four months later, the hemorrhage was reabsorbed (Fig. 3).

On examination, the patient was found to be alert. He demonstrated comprehension of the spoken language, executing simple and complex orders. However, upon attempting to speak, he could only issue inarticulate monosyllables and unintelligible

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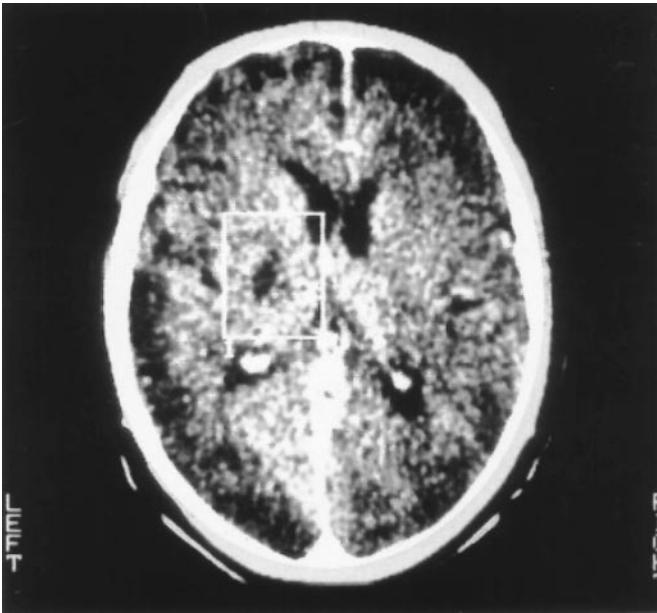


FIG. 1. Brain CT scan in transverse section showing hypodensity, indicated by lines that delimit it at the level of the genu and posterior arm of the left internal capsule involving the medial portion of globus pallidus.

utterances. He presented a right facio-brachio-crural hemiparesis which was mainly brachial in nature, with enhanced osteotendinous reflexes and indifferent cutaneo-plantar responses. While there was a slight spasticity on his right side, there were no sensory disorders. He also displayed a mild left crural paresia with preserved muscular tone, a flexor plantar response, and slowing of movement on his left side.

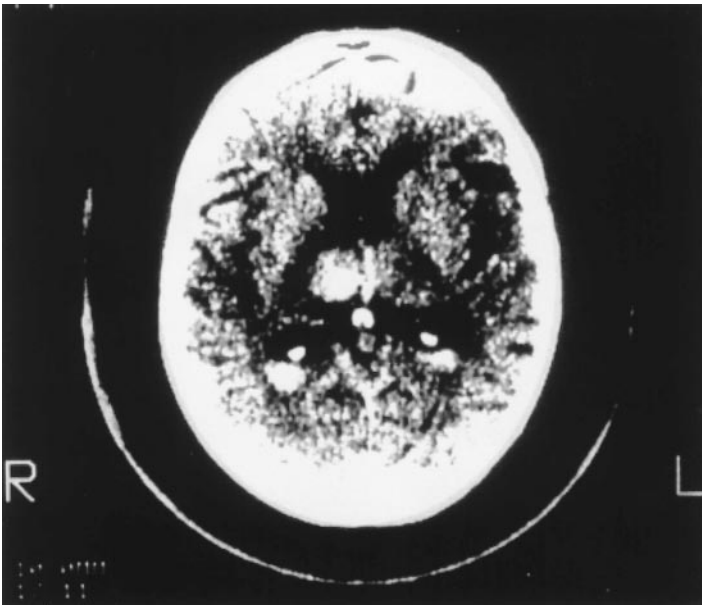


FIG. 2. Two months later there is an added hemorrhage, developed at the level of the right thalamus and sparing the pulvinar, as observed in a transverse section of the CT scan.

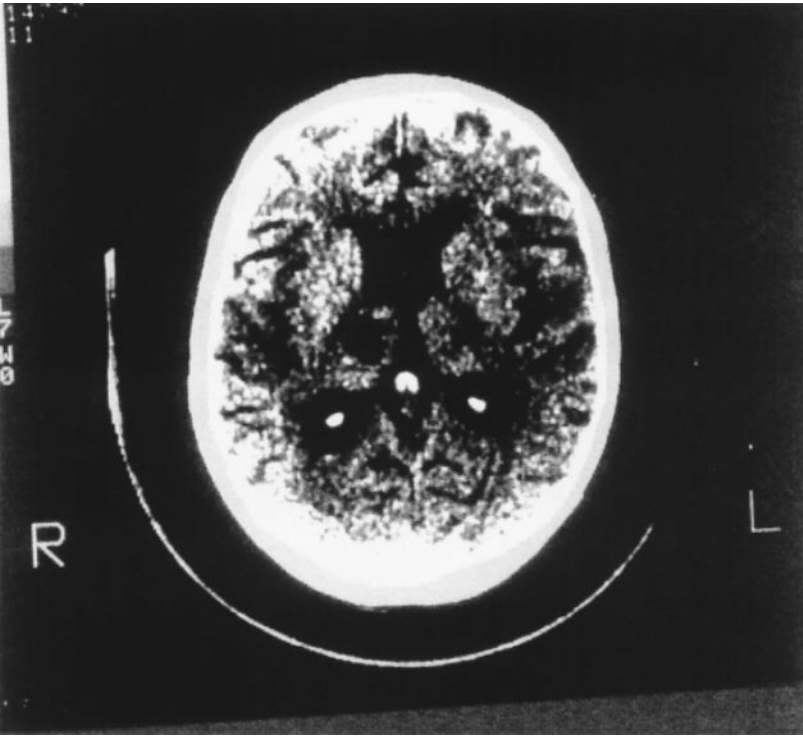


FIG. 3. Four months later, blood has been reabsorbed, leaving a cavity at the level of the right thalamic hematoma. Brain CT scan, transverse section.

Neither dysphagia nor velopalatine paresia was found. The patient moved his tongue slowly, while the mentonian reflex was increased and the velopalatine reflex was preserved. Directional hypokinesia was observed in his upward gaze, which was limited and could only be sustained briefly. He occasionally suffered sudden episodes of hypersomnia which did not show the typical EEG pattern of narcoleptic attacks.

During his neuropsychological examination, the patient exhibited a lack of short-term memory recall. When he was requested to memorize a series of names and record them in two different series, he evoked names from the first series in the second (proactive interference). To date, the patient remains anarthric, but communicates by means of a typewriter using his left hypokinetic hand.

In examining a patient we needed to consider the distinction between (a) an aphasic disorder, (b) speech apraxia, (c) aphemia, (d) mutism, or (e) extrapyramidal hypokinesia of articulatory speech mechanisms. These possibilities are summarized as follows:

(a) *Thalamic aphasia*, which has been described by Penfield and Roberts in 1959 and Ojemann in 1977, is characterized by its fluent dysnomia with frequent perseverations, but with intact comprehension and repetition. According to Cambier et al. (1982) this disorder features the presence of paraphasias and incoherences with normal repetition, which was lacking in our patient. Crosson (1984) added the presence of jargonaphasia, which was also absent. In keeping with the above-mentioned criteria, there were no comprehension deficits and there was a loss of spontaneity in verbal expression. However, our patient, who was anarthric, found speech articulation almost impossible, and this behavior exceeded the hypophonia and/or articulatory fluency deficits described in most thalamic aphasia cases.

Thalamic aphasia has been described in thalamic tumors (Smythe & Stern, 1938), thalamotomies (Waltz et al., 1966; Selby, 1967; Bell, 1968; Samra et al., 1969; Ojemann, 1975), and in the surgical removal of a thalamic arteriovenous malformation (Luria, 1977). Postmortem studies of thalamotomized patients show that small lesions in the left ventrolateral nucleus may be associated with mild but persistent aphasic disorders (Samra et al., 1969). Kirshner and Kistler (1982) reported a case of thalamic aphasia secondary to a right thalamic hemorrhage in a left-handed patient. Bell (1968) also found thalamic dysarthrias when he performed right thalamotomies.

Thalamic aphasias, secondary to ischemic lesions, may occur in the left tuberothalamic or in the paramedian territory. As a rule, it is bilateral in nature (Nadeau & Crosson, 1997). Aphasias, secondary to vascular lesions, last longer than those secondary to thalamotomies. Kameyama (1976–1977) documented two cases of thalamic aphasias due to left thalamic hemorrhages which involved the posterolateral ventral nucleus and pulvinar.

(b) In *anarthria* there may be discerned a paretic feature with weakness of the muscles dependent on the ambiguous and major hypoglossal nuclei attributed to damage to the upper motor neuron and/or its descending pathways, which are mostly bilateral. The apraxic feature belongs to the *buccolinguofacialis apraxia*, which implies disturbances in mouth opening and closing, whistling, licking, kissing, smiling, and so on. Alajouanine and Lhermitte (1963) have observed this type of apraxia in patients without anarthria. If our patient had developed this type of apraxia, (i) it would be regarded as a subcortical phenomenon because of the absence of opercular lesions in the CT of his brain and (ii) it would not justify the existence of his anarthria.

(c) *Aphemia*, characterized by Schiff et al. (1983), is a severe, scarcely reversible dysarthria without aphasic disorders. It is linked to lesions of the left hemisphere: in the lower half of the primary motor cortex, in the posterior portion of the inferior frontal gyrus, and in the underlying white matter and anterior arm of the internal capsule. This concept may apply to the first lesion (left sided) developed by our patient, but it was surpassed by the second (right-sided) lesion.

(d) *Mutism* is the inability to speak which is linked to attentional-intentional deficits (Watson et al., 1981); due to periaqueductal gray matter lesions (Botez and Barbeau, 1971), bilateral lesions of the medial thalamus (Brage et al., 1961), the globus pallidus (Denny-Brown, 1962), or the medial areas of the frontal lobes (Nielsen and Jacobs, 1951); or with psychotic defects in speech production (i.e., schizophrenia). Our patient, lacking a psychiatric history, made great efforts to speak, thereby failing to show the lack of intention to speak that characterizes mutism. His desire to communicate was documented when he successfully typed out a message using a manual typewriter with his hypokinetic left hand.

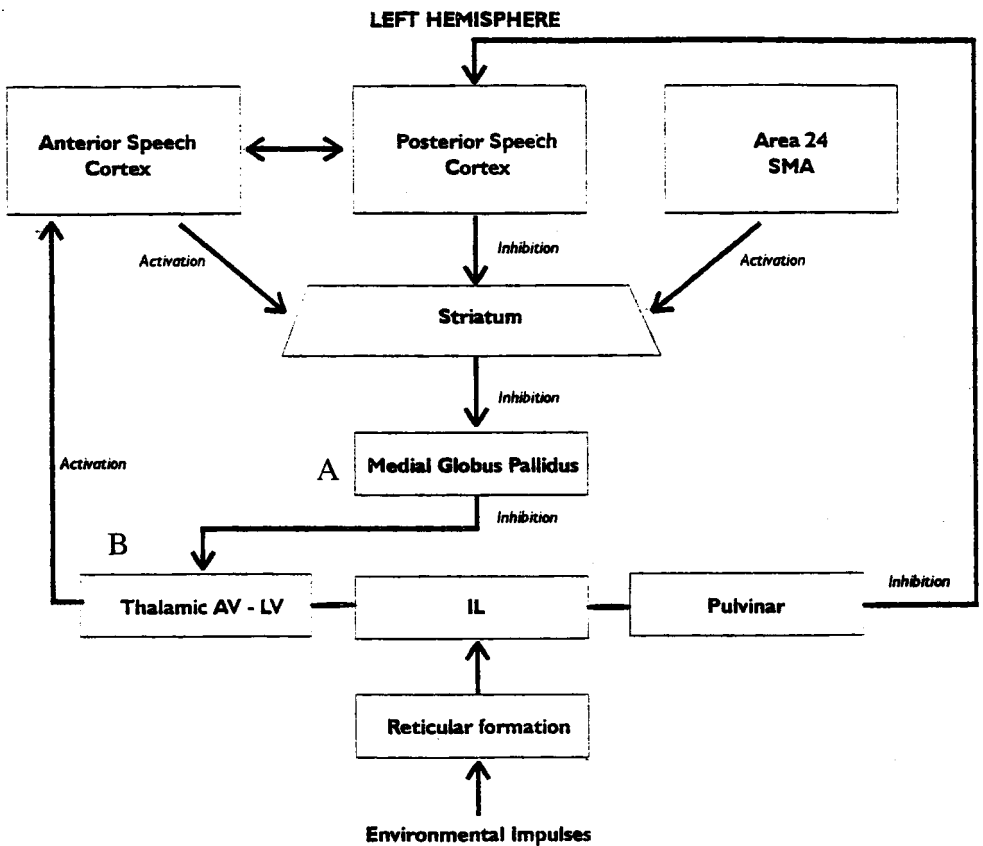
Mutism is also linked with lesions of the left anterior arm of the internal capsule (Milhaud et al., 1994). These lesions may include fibers descending from the frontal associative cortex toward the contralateral cerebellar hemisphere. There are cases of mutism arising from (a) cerebellar lesions (Rekate et al., 1985), (b) damaged connections between the ventrolateral thalamic nucleus and the motor cortex, or (c) damaged pathways implicated in speech initiation which would include the medial subcallosal fascicle that carries the fibers from the areas 24 and AMS to the caudate nucleus.

(e) Finally, the slowing down in tongue movements presented by our patient seemed to be a component of a generalized *hypokinesia* instead of a specifically apraxic disorder. This hypokinesia can be linked to the extrapyramidal system derangement and was seen after the second lesion had interrupted the thalamo-cortical extrapyramidal projections.

DISCUSSION

Riklan et al. (1969) observed that in bilateral lesions of the thalamus, a lesion on the right side added to a previous one on the left side more commonly led to dysarthria than a left-side lesion added to a right-sided previous one. These facts address the importance of additional bilateral effects and have not received accurate explanation or interpretation at this time.

It is a theoretical challenge to discuss the role of the different regulatory relays for subcortical speech functions. The anatomical relationships among these different regulatory relays have been outlined by Wallesch and Papagno (1988) (Fig. 4 shows the site of the functional interruption of the circuits in our patient's case).



AV - LV: Anteroventral and Lateroventral Thalamic Nuclei
IL: Intralaminar Nuclei

FIG. 4. The anterior and posterior speech cortex exert a modulatory influence on the striato-pallido-thalamic loop, which in turn regulates the motor releasing of speech. Area 24 and SMA contribute to endoevoked speech, providing motivational drive as the intralaminar thalamic nuclei contribute to exoevoked speech receiving environmental impulses. (A) The left-sided lesion of the patient producing interference with the pyramidal function (damaging the speech motor output itself at the capsular level) but also with the extrapyramidal sites at the globus pallidus level. (B) The proposed thalamic extrapyramidal disturbance debt to the second lesion placed at the right side implies certain degree of bilateral thalamic function in order to activate the speech cortex.

Crosson (1992) contended that while the cortico-striatal-pallidal-thalamo-cortical loop regulates the release of cortically formulated speech segments, the thalamus activates the anterior speech cortex and cortico-thalamo-cortical pathways, which would transfer information from the anterior speech cortex toward the posterior and vice versa. This same author also developed a theoretical model according to which the dominant temporal lobe normally maintains an inhibition on the head of the dominant caudate nucleus, which in turn generates hypoactivity of the inhibiting circuit passing through the medial pallidus. The latter could therefore exert its inhibitory action on the anteroventral thalamic nucleus, arresting the activation exerted on the anterior speech cortex (Crosson, 1985). The participation of the medial pallidus in speech production has been indicated by various authors (Svennilson et al., 1960; Wallesch, 1985).

Completing the concepts advanced by Crosson, a double integrative adjustment may be proposed: (a) Phasic changes on the anterior speech cortex would release speech segments ready for their motor programming, while (b) a tonic flow on the anterior speech cortex would maintain it if it responded to the demands originating from the outer world or from the same subject. Goldberg (1985) calls these speech forms endo- or exo-evoked according to whether the initiative to speak originates from the outer world or from within the subject, showing that the thalamus is primarily implicated in endo-evoked speech.

In our patient's case, hypokinesia of articulatory speech mechanisms was attributed to the interruption of thalamic impulses relayed by the anteroventral and lateroventral thalamic (AV and LV) nuclei of the right thalamus, which was affected by the hematoma. This in turn increased the damage induced by the left capsular and pallidal infarct that had involved the corticobulbar pyramidal fibers. It is possible that the subcortical anarthria could be a form of speech articulation akinesia, preventing or hindering the initiation and/or continuation of speech.

It can be said that these additional effects appeared when the effect of the left-sided lesion (damaging the motor output of speech by interrupting the fibres at the level of the capsular genu and damaging the extrapyramidal regulation of the speech cortex activation by pallidal derangement) was made worse by the destruction of the extrapyramidal sector of the right thalamus. This produced a failure to activate the cortical language zones that were possibly relevant in compensating the previous left-sided deficit.

Naeser et al. (1982) and Damasio et al. (1982) have observed that the capsular lesions extending forwardly and upwardly into the white matter induce dysarthria with preservation of syntax and speech comprehension. This lesion type interrupts the descending projection from the speech motor area and from the inferior motor cortex. In our patient's case, the capsular lesion extended toward the posterior arm of the internal capsule involving the globus pallidus. The role of the basal ganglia in speech production is linked more closely to processes regulating its motor execution than to the formulation of its content. According to Kornhuber (1971) and DeLong and Strick (1974), the basal ganglia generate slow and uniform movements of a voluntary nature which are more specialized in ramp than ballistic movements.

Finally, with regard to the participation of the thalamus in the induction of anarthria, Van Buren (1975) found that the thalamus can serve to integrate respiratory mechanisms necessary for speech vocalization and linguistic functions. Our patient had developed dysarthria due to the left capsular and pallidal lesions. His ability to vocalize and his phoneme expression decreased dramatically after the right thalamic lesion. The findings of Ojemann and Fedio (1968), who have described disturbances in verbal material recall in the short-term memory, were also observed in our patient. According to Schaltebrand (1975), the role of the thalamus in speech may be the

release or silencing of preformed speech fragments, whether phonemes, words, or phrases. Metter et al. (1988) suggest that the thalamus, as well as the basal ganglia, has a direct relationship with speech fluency, more than with its comprehension. Various nuclei, including the anterior, anteroventral, lateroventral, dorsomedial, intralaminar, and pulvinar, have been incriminated in their relationship to speech production (Allen et al., 1966; Bell, 1968; Ciemans, 1970; Kameyama, 1976–1977; Ojemann, 1977; Brown, 1977; Crosson, 1985; Crosson, 1986; Graff-Radford et al., 1985; Schaltebrand, 1975; Gorelick et al., 1984).

By comparison, most studies indicate that aphasic disorders occur more frequently when a single thalamus is intervened if the operation has been performed on the left side (Cooper, 1958; Allen et al., 1966; Selby, 1967; Bell, 1968; Asso et al., 1969; Samra et al., 1969; Darley et al., 1975). According to Ojemann (1977), the left thalamus is involved in the processes that provide the mechanical basis to speech: the respiratory substrate and control of oral-facial muscles. On the other hand, the right thalamus serves attentional processes bound to visuospatiality. Ojemann (1983) also suggested that the thalamus participates in the attentional mechanisms related to speech. In either case, the intralaminar/reticular nuclei could participate by selectively disinhibiting thalamocortical circuits used for speech production (Nadeau & Crosson, 1997). According to Crosson (1985), antero-cortical speech mechanisms are activated by the thalamic anteroventral nucleus that would regulate the excitatory flow originating in the reticular formation. Friston et al. (1993) showed activation of both thalami in PET studies on subjects receiving verbal fluency tests. Our hypothesis contemplates the disruption of the mechanisms of speech activation because the location of the left-sided lesion damaged the globus pallidus. An additional effect was found when the right extrapyramidal thalamus (AV and LV nuclei) was also affected.

It is necessary to make the distinction between (a) motor speech disorders as dysarthria, aphemia, or anarthria observed in bilateral thalamic lesions (in our case this was a combined right thalamic hemorrhage and left striatocapsular infarction) or (b) motor aphasia; speech fluency disorders due to programming disturbances and their control which is secondary to the cortical and/or subcortical lesions of the dominant hemisphere.

Vertical gaze impairment tends to accompany amnesic disturbances and motor slowing, as in paramedian diencephalic syndrome (Boggouslavsky et al., 1968), which may also present aphasic disorders (Fensore et al., 1988). These patients may develop sharp cognitive fluctuations, as in our patient, who presented sudden episodes of hypersomnia. Nevertheless, the paramedian diencephalic syndrome is due to infarcts affecting both thalami bilaterally. In our case, the thalamic lesion was unilateral and caused by a hemorrhage that did not affect the tectal region directly, so that oculomotor disorders were incomplete and reversible.

CONCLUSION

This case report illustrates the existence of anarthria due to subcortical lesions located outside the rolandic operculum and not accompanied by bucco-linguo-facial apraxia.

In our patient's case, an initial left pallidocapsular lesion of ischemic origin was followed two months later by a right thalamic hemorrhage sparing the pulvinar but involving other nuclei, such as the anteroventral, the lateroventral, the dorsomedial, and the intralaminar, all of which were linked to the regulation of speech production.

We have emphasized in this article that our patient's speech was hypokinetic, especially after the second lesion, without truly aphasic disturbances. Interestingly, our

patient's case (1) had a completely subcortical location; (2) there was no attentional-intentional disorder indicating mutism, since the patient desired to communicate with us; and (3) the lesion responsible for the anarthria occurred in the right thalamus. In agreement with findings reported by Riklan et al., a right thalamic lesion played a role in initiating and/or sustaining the speech rhythm. PET activation studies (Friston et al., 1993) seem to confirm this role. Further studies are needed in this area in order to clarify the relation between the right thalamus and language.

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