



## Brain tumor revealed by atrial sinus block

### Tumeur cérébrale révélée par un bloc sino-atriale

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#### ABSTRACT

Bradycardia is rare in children and may be asymptomatic or cause fatigue or discomfort leading to syncope. It may be a warning sign of underlying pathology. A check-up for a cardiac or extra-cardiac organic cause should be performed. We report the case of an 8-year-old child admitted to the emergency room for bradycardia with a sino-atrial block that caused syncope. The assessment has objectified a brain tumor. We will discuss the different mechanisms that can explain the occurrence of bradycardia during a brain tumor, and the specificities of the management.

**Keywords:** Brain tumor, bradycardia, vagal hyperactivity, intracranial hypertension.

#### RÉSUMÉ

La bradycardie est rare chez les enfants et peut être asymptomatique ou provoquer une fatigue ou un malaise conduisant à une syncope. Elle peut être un signe d'alerte d'une pathologie sous-jacente. Un bilan à la recherche d'une cause organique cardiaque ou extra-cardiaque doit être réalisé. Nous rapportons le cas d'un enfant de 8 ans admis aux urgences pour une bradycardie avec un bloc sino-atrial à l'origine d'une syncope, le bilan a objectivé une tumeur cérébrale, nous allons discuter les différents mécanismes qui peuvent expliquer la survenue de bradycardie lors d'une tumeur cérébrale, et les particularités de la prise en charge.

**Mots clés :** tumeur cérébrale, bradycardie, hyperactivité vagale, hypertension intracrânienne

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## INTRODUCTION

Bradycardia is defined by a heart rate below 60 beats per minute, It can be physiological in the athletic or elderly subject, as it can be secondary to an underlying cardiac pathology (sinus node anomaly, conduction disorder) or extracardiac such as a metabolic disorder or dysthyroidism, or after taking some medicines (betablocker, bradycardia calcium channel blockers). It can also be related to neurological causes as seen in the reported case (1).

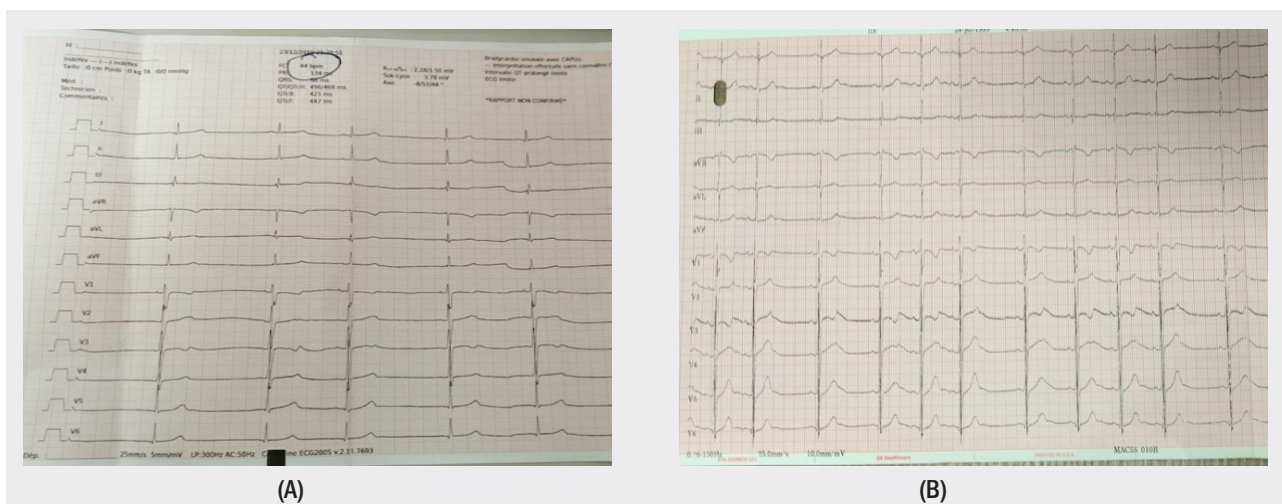
We will discuss through this clinical case, the different neurological causes that can lead to bradycardia.

## CLINICAL CASE

Eight-year-old child with no specific pathological history, admitted to the emergency department for significant

fatigability for one week. The child didn't report any chest pain, dyspnea, neurological or infectious signs.

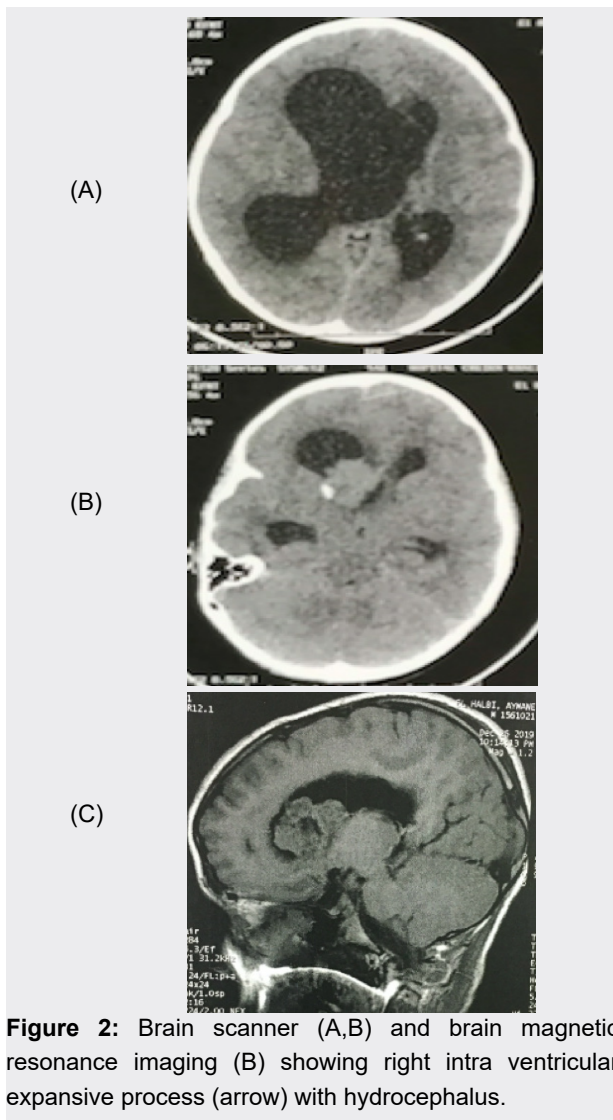
On clinical examination, he was conscious, his conjunctiva was well colored, afebrile ( $T=36,5^{\circ}\text{C}$ ), his heart rate was 43 beats per minute with a blood pressure of 120/60 mmHg. His cardiovascular examination has shown regular bradycardia without any audible murmur or signs of heart failure. The neurological examination was normal. No motor or sensory deficit was observed, no meningeal stiffness and reflexes were preserved. Blood tests were normal (blood glucose, hemoglobin, thyroid blood tests, blood ionogram, magnesemia, and serum calcium), infectious check-up was negative. The electrocardiogram revealed an atrial sinus block 2/1 with a heart rate at 43 beats per minute. A deep breathing test was performed (figure 1).



**Figure 1.** First electrocardiogram (A) showing an atrial sinus block 2/1 (calibration 25mm/s, 5mm/mV) and Second ECG (B) after deep breathing (calibration 25mm/s, 10mm/mV)

An acceleration of the heart rate with a variability of 65% was found, and vagal hyperactivity was detected. The echocardiography was normal. The patient was hospitalized for clinical monitoring. During his hospitalization, he had sinus bradycardia at 35 beats per minute with syncopal unconsciousness of a few seconds. The post-syncopal clinical examination was unchanged. A few hours later, he made an abnormal movement with his lower lip. Given this evolution, a brain scanner was performed to eliminate an intracranial cause. It revealed an expansive supratentorial intra-ventricular right process with hydrocephalus

and signs of intracranial hypertension (Figure 2). The patient was transferred to the neurosurgery department and an external ventricular shunt was emergently performed. A brain magnetic resonance imaging showed a 22.5/42/32 mm right ventricular expansive process of lobulated contours blowing into the left ventricle (figure 2). We planned to perform a lumpectomy. Unfortunately, the patient presented with severe bradycardia and profuse sweating followed by a drop in blood pressure and asystole that could not be recovered despite resuscitation measures.



**Figure 2:** Brain scanner (A,B) and brain magnetic resonance imaging (B) showing right intra ventricular expansive process (arrow) with hydrocephalus.

## DISCUSSION

Brain tumors may manifest as headaches, seizures, clinical signs of intracranial hypertension (nausea, vomiting, visual disturbances), or motor deficit. Cardiac manifestations of brain tumors are non-specific, with a few rare cases of bradycardia described in the literature as warning signs (2). Many investigations done allowed us to understand the mechanisms of bradycardia of so-called central origin (in the case of a brain tumor or haematoma, or a spinal cord tumor, or during neurosurgery) (3).

The "Cushing's reflex" is characterized by the development of systemic hypertension, bradycardia and apnea secondary to an increase in intracranial

pressure responsible for pressure and/or stretching of the brainstem. It is a protective and effective action of the brain that preserve an adequate cerebral perfusion pressure despite an increased intracranial pressure. It has been demonstrated that the haemodynamic features of classical Cushing's response can be produced by electrical stimulation or distension of tissues within a thin strip of brain along the floor of the fourth ventricle in the rostral medulla and caudal pons. The efferent limb of the bradycardic response being the vagus nerve (4). On the other hand, the elevation of intracranial pressure with the subsequent decrease of cerebral perfusion pressure significantly induced a vigorous cerebro-protective neuroendocrine system activation that can lead to the neurogenic stunned myocardium, which is defined as myocardial injury and dysfunction occurring after diverse types of acute brain injury as a result of imbalance of the autonomic nervous system (5). Another cause that may explain bradycardia in brain tumors is its location. Cases of localization of tumors in the temporal or frontal lobe are associated with bradycardia (6). Tumor location next to the regulatory heart centers (thalamus, hypothalamus and tonsil) may be accompanied by bradycardia (7). A localization also in the posterior part of the cerebellum (sub-lobule IX-b of the cerebellar vermis) is associated with a decrease in sympathetic activity, responsible for bradycardia and hypotension (8).

Epileptic seizures can also lead to changes in the autonomic nervous system. Tachycardia is the most common manifestation, but bradycardia and cardiac asystole may occasionally occur with temporal lobe seizures. Bradycardia and many other types of arrhythmias have also been described during episodes of focal or generalized epilepsy (8). Other mechanisms described in the literature can also explain bradycardia such as trigeminocardiac Reflex (9), ventricular catheter obstruction in cases of hydrocephalus or preganglionic sympathetic or parasympathetic nerve fibers injury (10). Finally, all these mechanisms can coexist and lead to bradycardia as in the case of our patient, his tumor located in the right ventricle, with a large mass effect responsible for intracranial hypertension. He first presented bradycardia, with syncope, and then signs of vagal hyperactivity, with an epileptic seizure.

If bradycardia and signs of vagal hyperactivity is present with normal cardiac workup, further investigation is needed to find a central neurological cause, which may require

urgent management.

Management of these patients include identification and treatment of the underlying cause of bradycardia, anti-cholinergics and if necessary cardiac pacing. Nevertheless, other causes of haemodynamic changes (i.e. anesthetic drugs, tumor manipulation) should also be considered and managed accordingly. We believe that this knowledge and understanding will help to identify the patients at risk and will also help in the management of neurosurgical patients with bradycardia (10).

### CONCLUSION

Bradycardia may be the only sign of a brain tumor with intracranial hypertension. It must lead us to perform an etiologic assessment with blood test, cardiac and also cerebral imaging, especially if there are other signs of vagal hyperactivity. Once diagnosed, the brain tumor must be urgently and effectively managed.

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