

## What Caused Jeanne-Francoise Champollion, Decipherer of the Ancient Egyptian Scripts, Premature Death?

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### Abstract

A possible relationship between so recurrent collapse/syncope events hit Jeanne-Francoise Champollion (JFC)'s and his being forty-one years old when he got the premature killing stroke, is engaging and worth investigation. Champollion (JFC) was too young and the first scholar committed to the unbearable mission of disclosing the language system behind the ancient Egyptian scripts from 4000-5000 B.C., despite Diabetes Mellitus, Gout disease, recurrent Syncope, chronic Tinnitus, Headaches, and later unilateral Gaze deviation, from whom he suffered. The etiology behind JFC's premature death is unestablished, possibly because his family refused to allow a postmortem dissection. Along with those facts and absence of lab tests, we will try analyzing the exact cause of death by discussing the differential diagnosis in its classical method: the main complaint, family medical history and hints of somatic signs in reliable literature. JFC's medical history was marked by frequent faints, part of them were neural mediated or vagal responsiveness. One of these collapses happened to him at eighteen years old, shortly after he was shocked by the news that Lenoire had published the first decipherment of Hieroglyphs. Other collapses recorded in literature were not vagal, by definition. Most striking was the collapse attacked him in 1822, in a moment of elation when he got the idea of the language system behind the Hieroglyphs and ran breathlessly into his brother's office and shout: "je tiens l'affaire!", "I found it", and immediately fell unconscious for few days. Two years after his return to Paris from Egypt, with the unequivocal proof of the Hieroglyphic language system in his hands, 'death was waiting for him', as he drifted into and out unconsciousness. We suppose, with enough confidence, that a vascular disorder was the etiology behind JFC early death, basically, cerebral arteries' dilatations, that finally had dissected and torn.

**Keywords:** Champollion; Collapse; Tinnitus; Stroke; Berry aneurysm; Strabismus; Ancient Egypt

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### Background

After last words of ancient Egypt have been sculpted on the Rosetta stone, around 196 B.C., anybody was able to read these scripts on the walls of temples, tombs, statues and papyrus [1,2].

Greek researchers initially believed that the abandoned magnificent Egyptian manuscripts are only a mixture of decorations and symbols. This belief prevailed until 1822 years after Christianity, same as the belief of Ptolemy's geocentric theory was, concerning the earth as the center of our galaxy, a

theory that lasted near one thousand years. Few scholars, tried to treat these symbols as a kind of a real written language, and made some progress.

Jeanne-Francois Champollion, who was able, at earlier age, to read write and speak twelve languages including Hebrew, Arabic and Coptic, swore to be the first to decipher the Egyptian scripts [1,3]. Obsessively, like in any great achievement, in spite of extreme health concerns, he found that these symbols were a real readable language, thus, refuting the prevailing Greek theory

about their symbolism, exactly as Galileo Galilei opposed the geocentric model of the Ptolemaic system. Unfortunately, a brain stroke killed him at forty-one years of age [2,3]. The exact etiology behind his premature death by a brain stroke, in an era with male average life expectancy of sixty to sixty five years [Wikipedia], remained unknown for more than one century and half.

Champollion had several health concerns. In the relevant literature, it is clear that Champollion suffered from recurrent collapse, part of these events were thought to be even his last moments of life. The idea of a possible relationship between recurrent collapse events, and between Champollion premature exitus, deserves investigation.

### Findings and case history

Champollion was diagnosed by his family physician, Dr. Janine, as suffering from Gout and Diabetes mellitus [3]. The exact etiology behind his premature death by a brain stroke, in an era with male average life expectancy of sixty to sixty five years [Wikipedia], remained unknown for more than hundred and fifty years.

Champollion medical history was marked by frequent faint fits, part of them were neural-mediated vaso-vagal reflexes as that happened to him after emotional distress at the start of the summer holiday, when he thought that his school would shut its doors behind him forever. Another collapse occurred at eighteen years of age, shortly after he was shocked by the news that a man named Lenore Alexander, claimed, erroneously after the event, that he is the first to decipher the ancient Egypt writings [1].

Other collapse events were not neural-mediated. The most striking collapse was described in several sources, was as follows:

While working, in Paris since early morning, on the latest inscription he got from an envoy who visited in Abu Simbel, his face suddenly were lit by the moment of discovery of the language system behind the Hieroglyphs. At this instant of elation, in 1822, he felt he must soon tell somebody, so he covered the distance of the neighboring street in the shortest time possible [4], breathless just as he burst into his brother's office, and shouted: 'je tiens l'affaire!' or 'I found it', and immediately fell down unconscious. For moments, his brother feared he was dead [1-3].

In his passport, Champollion was reported as: A twenty-five years old, one meter and seventy centimeters tall, black hair and eyes, brown complexion, a large forehead, flat nose and his face was lightly marked by smallpox [3]. His letters to his brother tell he suffered from recurrent headaches, constantly teasing tinnitus 4, which led him to have volcanic temperament and a furious impatience. He also mentioned the remitting swelling of his feet, described as follows: 'I mounted a donkey, for I have a severe painful swelling in my left foot. I was transported over the shoulders of four men, for the slope was almost perpendicular. The gout disappeared after a few days, leaving me free to work on monuments'.

The apparent Champollion's eye squint laterally, as it is seen in the painting drawn by Giuseppe Angelelli, when they visited Egypt, Thebes, in 1829, along with the appearance, by the way, of Champollion as an obese person as he was "filling his seat"; were important hints for the horrible to come [3]. Few months before

his death, Champollion fell in a semi-coma. His family prevented people and friends to see his miserable state, especially his peculiar face.

### Family history

Dr. Janine wrote, in 1830, about Champollion family: "Champollion's mother Jeanne, an illiterate woman, suffered from rheumatism in her forties, cured by a local peasant by hot massages. She born Champollion, the last of seven children, in 1790, and raised him in humble circumstances. His father was an impoverished bookseller. Two of Champollion's brothers died during their infancy; the bachelor child, a son, died within hours after birth, and the other son died prematurely at two years old [2,3] from unknown cause. Eleven years after Champollion married Rosine Blank, Zoraide, their only daughter, was born. She lived seventy years (1824-1894).

### Differential diagnosis

The main complaint that Champollion had suffered since his childhood, was countless episodes of stroke-like collapse; some of which were accompanied by unilateral weakness or transient paralysis. Together with this major complaint, he suffered diseases mentioned above: Diabetes Mellitus, etc.

Obviously, the etiology behind these events did not affect his intelligence nor his enthusiasm to crack the ancient Egyptian language. A chronic and remitting/relapsing disease or diseases of some areas of the CNS, which began at ten years of his age, possibly were the common denominator that might attach together these episodes of collapse [1,4].

Finding the etiology of Champollion's early death in line with the above-mentioned main Complaint will be achieved through the organ system review, as the following:

### Heart and vascular disease

Champollion early death was not a sudden death. Thus, we can exclude immediately several etiologies during which there are recurrent collapses and ultimate sudden cardiac death, particularly, dangerous arrhythmias, as due to Long QT Syndrome, 'fast, broad and irregular' arrhythmias of WPW syndrome, for instance.

The "je tiens l'affaire" event could be representing a left ventricular outlet obstruction; however, this defect is almost daily symptomatic illness of childhood and adolescence. Left outlet obstruction will not allow Champollion to travel to Italy and Egypt, navigate easily walk or running over long distances, while roaming from one location to another. In some topographies man cannot ride donkeys. Sudden syncope, After all or cardiac death, which is induced by an abrupt physical stress, as might occur in cases of catecholaminergic polymorphic ventricular tachyarrhythmia, [5,6] could be excluded immediately, for that Champollion death was not sudden, but was expected.

Neural-mediated collapses, or vaso-vagal faint events from which Champollion suffered [3,4,6], although they are usually non-killing accidents, does not rule out other organic causes of recurrent syncope in the same person, such as coronary atherosclerosis and occlusion, which could be symptomatic under several physical and spiritual conditions that pressed as clutch on Champollion's chest [7].

Symptoms related to coronary artery disease were absent; this is evidenced in his letters sent from Thebes, Egypt: "We managed to climb the mountain of Horemheb, and, when we got to the plateau at the top we were astonished from the degree of devastation" [3].

Cardiac amyloidosis [8] causes heart failure and presents with signs and symptoms of progressive dyspnea that does not fit our case.

Atrial Fibrillation, when episodic or chronic, has a low probability as a cause of Champollion's collapses and strokes. The risk of obstruction of the cerebral arteries by thrombi, as a cause of stroke, could be calculated in the case of Champollion [9], by the abbreviated CHADS2. It equals 2-3/6 in the Champollion case.

Cardiac myxomas are more common in women. Symptoms may occur at any time, but nothing is mentioned about collapses accompanying a change of body's position [10]. Therefore, Champollion's death cause was not his heart.

### CNS and CNS vascular diseases

MS is not included in the list of etiologies of stroke; neither the 'Jean-Martin Charcot Triad' fits our case. MS is excluded, in spite it is a cause of abrupt collapses, or stroke-like events, weakness, hemi-paresis and even hemiplegia [10,11].

Cranial Tumors and pseudo-tumor symptoms, mainly headache, are never remittent, but steady and progressive in their course. Champollion periodic fainting beginning at his teenage, with long recovery periods could not be due to intracranial benign tumor, therefore.

Compression of internal carotid artery by an anomalous hyoid bone, and its consequent syncope, which can ensue after head turning [12], is extremely rare and we have no hints of its existence in the literature.

'Hereditary Hemiplegic migraine' in the absence of preceding prolonged aura (sometimes for several days), or convulsions, really makes it an irrelevant possibility.

One day, Champollion requested unexpectedly to remain alone in his room [3]. Three weeks later, while he was talking to a friend about ancient Egyptian astronomy, he suddenly cried out and moved his hand to his head, and soon fell in a very serious collapse. This left him almost completely paralyzed for one week, but he could still speak later.

Assuming that Champollion might have had chronic systemic hypertension, relying on his obese appearance in several paintings, then, his recurrent complaints of headache accompanied by Diabetes mellitus would have affected him as follows:

A fall in blood pressure during sleep can lead to marked hypo-perfusion in the previously narrowed small vessels of the brain leading to ischemic stroke in the morning. Conversely, a sudden rise in blood pressure due to excitation during the daytime can cause a tear in blood vessels, so a hemorrhagic stroke ensues at evening. Stroke symptoms typically start suddenly, during seconds to minutes, and in most cases do not progress further. Not only that but also, the micro vascular diseases, which could lead to the so called vasculopathies or angiopathies, (e.g. diabetes and systemic hypertension) could cause also a third nerve paralysis [13]. Champollion's left eye lateral gaze is shown only in the last

painting, which was painted for him and his expedition in Egypt, when he was thirty-eight years old.

The main causes of morbidity and mortality along with third nerve paralysis are vascular anomalies such as a berry aneurysm of the posterior communicating artery of the Circle of Willis. Other diseases like MS, trauma, compression by a brain infiltrative disorder of the meninges and or due to degenerative diseases were already excluded. Lateral or bilateral middle cerebral artery stenosis, through the mechanism of hypo-perfusion [10,14-16] leads to almost the same clinical features as those of aneurysm rupture, except it is painless.

Hypothetically, one cause was behind the premature death of our Scholar, which was pursued in our article. Of course, there was no "infinite" number of etiologies, but there were several risk factors which had an adverse impact.

Berry aneurysm leaks, and, eventually, rupture was the only option which, in my opinion, is the most appropriate etiology for Champollion's early death. It is substantiated, In one hand, it is substantiated in the information accumulated up to date, and are supported by our analytical perspective, in the other hand. Even though it seems that Berry aneurysm had a small chance to be the cause of our Scholar's premature death, but indeed there is considerable chance of almost 20% of patients with Berry aneurysm to die after many years from the start of leakage.

Small-vessel disease (SVD) is a familiar, usually diffuse, aging phenomenon that is exacerbated by hypertension and diabetes mellitus. It is regarded as an important cause of lacunar (white) infarction and intracerebral hemorrhages (the red infarction), too [15,17].

Recent observations that some small deep brain lesions may be caused by increased permeability of arterioles. These diffusions obviously resemble the berry aneurysm leaks [17,18].

The separation between the pathophysiology of angiopathy and vasculopathy, of the Willis circus, and between that of the Berry aneurysm in the same circus, from the clinical aspects, is not easy at all. Thus it is too not correct to claim, overwhelmingly, that in all cases which we could not exclude the Berry aneurysm by clinical and / or laboratory means, vasculopathy is the case.

The impression exists among clinicians that there is a considerable overlap between the pathophysiology behind the formation of angiopathy or vasculopathy, which lead to SVD, and between that of berry aneurysm. The more it is in patients who suffer Diabetes Mellitus and arterial hypertension [19,20].

According to the "clinical" story of our privileged decipherer, the headache was recurrent, almost constant; something that is in favor of Aneurysm leakages than vasculopathy. His suffering from a permanent tinnitus favors cerebral vasculopathy, from the other side.

Observations in hypertensive humans invoke edema rather than ischemia in the formation of such lacunar lesions, the white infarcts [18,19]. Cerebral vasculopathy is of considerable importance, but may be implicated only in one third of all strokes [21].

Characteristic cerebral parenchymal changes in SVDs include small diffuse hemorrhages, and microbleeds, Lacunes, and Arteriopathic leukoencephalopathies [15]. The question, which

remains unresolved, is whether these diffuse lesions in his brain, could enable Professor Champollion to be an active lecturer, at a time when Dr. Young, his most eminent competitor in the task of deciphering the ancient Egyptian scripts, was sitting among his audience, together with other scholars?! or had he the ability to be an obsessive researcher and head of the delegation to Egypt, during remissions between one collapse and the another, while having a sound mind?

So, we conclude with enough certainty that Champollion died from a brain stroke due to a major aneurysm rupture of an artery of the Circle of Willis.

Was there a single or there were multiple aneurysms? Contrary to multiple Berry aneurysms which are usually genetic in origin, and always accompanied by congenital defects of kidneys and Aorta, hence they are inappropriate possibility-, the single aneurysms of the internal carotid and vertebra-basilar arteries branches constituting the Circle of Willis, could be induced by either diabetes or hypertensive atherosclerosis.

A cerebral aneurysm of the Circle of Willis may begin to 'leak' small amounts of blood. Nevertheless, when it completely ruptures, intracranial subarachnoid hemorrhage ensues, causing an unbearable "thunderclap" headache, the warning sign of rupture. This kind of headache knocked Champollion before one of his near death collapses. Together with confusion, lethargy, stupor, muscle weakness, difficulty moving any part of body, speech impairment and gaze deviation, all these features point towards completely rupture of one of these aneurysms leading to an intracranial subarachnoid hemorrhage that eventually caused his killing stroke.

## Interpretation

Certainly, Champollion died of a brain stroke. A major cerebral vascular event precisely. We investigated the real reason behind the final event, and concluded that it was a repeated 'mini'-leaks or ruptures of an aneurysm in any of the circle named Willis vessels, within months, to a maximum rupture.

Champollion expected his near death several months before it

knocked his door. Actually, his demise was the result of a serious disease or a combination of diseases that lasted for his entire life.

Theoretically, we assume that if there were previous publications on the subject—which we searched in vain in the medical literature- which might have come to the same conclusion, as ours, pertaining to what caused the death of Champollion, it will be then an intersection information that proves the willingness of our conclusions. If this assumption is not true, and another manuscript has been written before ours, about the same subject, then other researchers will have the opportunity to compare.

## Summary

Two years after his return to Paris from Egypt, with the unequivocal proof of the Hieroglyphic language system in his hands, death was „waiting for him indeed at 'Babel'”, as he had stated once. At the end of February 1832 he drifted, in and out of unconsciousness; when he suddenly became alert and regained his speech, and uttered the following tragic words: 'So soon- there still so many things inside here!' Sensing that his end was near, his brother called the priest to give him the last rites.

Towards 2 o'clock in the morning of 4 March 1832, surrounded by his family and his brother Professor Champollion-Figeac, Champollion condition took a marked turn for the worse, a handful of his closest friends were asked to leave.

At four o'clock in that morning, his physician Dr. Janine confirmed Champollion's death by holding a mirror to his mouth, and by illuminating his eyes with a light of a candle. Respecting Champollion family's will, his physician did not carry out an autopsy. His family halted any information about his disease, too. The exact and direct disease that caused his passing remained not discussed for more than a century, until this moment.

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## References

- 1 Kahn D (1967) *The Code breakers: The comprehensive History of Secret Communication from Ancient to the Internet*.
- 2 Adkins L, Adkins R (2000) *The keys of Egypt: the obsession to decipher Egyptian hieroglyphs*. HarperCollins.
- 3 Robinson A (2012) *Cracking the Egyptian code: the revolutionary life of Jean-François Champollion*. Oxford University Press.
- 4 Pirodda A, Brandolini C, Raimondi MC, Ferri GG, Borghi C, et al. (2009) Tinnitus as a warning for preventing vasovagal syncope. *Medical hypotheses* 73: 370-371.
- 5 Joshi P, Saxena A, Kaul U, Mansoor AH (2010) Catecholaminergic polymorphic ventricular tachycardia (CPVT) with associated sinus node dysfunction. *Indian Heart J* 62: 84-86.
- 6 Goto F, Tsutsumi T, Nakamura I, Ogawa K (2012) Neurally mediated syncope presenting with paroxysmal positional vertigo and tinnitus. *Epub* 39: 531-533.
- 7 Sheldon R, Hersi A, Ritchie D, Koshman ML, Rose S (2010) Syncope and structural heart disease: historical criteria for vasovagal syncope and ventricular tachycardia. *J Cardiovasc Electrophysiol* 21: 1358-1364.
- 8 Majumder B, Kumar A, Dutta G (2012) "A case of cardiac amyloidosis with syncope". *India Heart J* 62: 171-172.
- 9 CHADS2\_scorem (2012) Stroke risk assessment and antithrombotic therapy. <http://en.wikipedia.org/wiki/>
- 10 Gao S, Wong KS, Hansberg T, Lam WW, Droste DW, et al. (2004) Microembolic signal predicts recurrent cerebral ischemic events in acute stroke patients with middle cerebral artery stenosis. *Stroke* 35: 2832-2836.
- 11 Compston A (2005) Jean-Martin Charcot on „Sclérose en Plaques’ (MS). *ACNR* 5.
- 12 Janczak D, Skora J, Rucinski A, Szuba A (2012) Recurrent syncope caused by compression of internal carotid artery by an anomalous hyoid bone. *Vasa* 41: 221-224.
- 13 Bousser MG, Biouesse V (2004) Small vessel vasculopathies affecting the central nervous system. *J Neuroophthalmol* 24: 56-61.
- 14 Li H, Zhang Y, Li H, Sui Q (2011) Recurrent unconsciousness due to bilateral middle cerebral artery stenosis. *Stroke. Cerebrovasc Dis* 20: 266-268.
- 15 F GRAY (2014) *Small Vessel Diseases of the Brain: Pathological Definition & Classification*. AP-HP Hôpital Lariboisière-Université Paris VII.
- 16 Adams HP (2014) Cerebral vasculitis. *Handb Clin Neurol*. 119: 475-494.
- 17 Hajj-Ali RA, Calabrese LH (2014) Diagnosis and classification of central nervous system vasculitis. *J Autoimmun* 48-49: 149-52.
- 18 Lammie GA, Brannan F, Slattery J, Warlow C (1997) Nonhypertensive Cerebral Small-Vessel Disease. *An Autopsy Study*. *STROKE*.
- 19 De la Monte SM, Moore GW, Monk MA, Hutchins GM (1985) Risk factors for the development and rupture of intracranial berry aneurysms. *Am J Med* 78: 957-964.
- 20 Ashrafian H (2014) Deciphering the death of Jean-Francois Champollion (1790-1832), the man who decoded ancient Egyptian hieroglyphs. *Neurophysiologie Clinique, Clinical neurophysiology*.
- 21 Ogata J, Fujishima M, Tamaki K, Nakatomi Y, Ishitsuka T, Omae T (1981) Vascular lesions underlying cerebral lesions in stroke-prone spontaneously hypertensive rats. *Acta Neuropathologica* 54: 183-188.
- 22 Evans TW, Venning MC, Strang FA, Donnai D (1981) Dominant inheritance of intracranial berry aneurysm. *Br Med J (Clin Res Ed)* 283: 824-825.
- 23 Sharpe JA, Bondar RL, Fletcher WA (1985) Contra lateral gaze deviation after frontal lobe hemorrhage... *J Neurol Neurosurg Psychiatry* 48: 86-88.