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Edward Anson ✦ Michael Fronda ✦ David Hollander
Timothy Howe ✦ John Vanderspoel
Pat Wheatley ✦ Sabine Müller ✦ Alex McAuley
Catalina Balmaceda ✦ Charlotte Dunn



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Did Alexander the Great Die from Guillain-Barré Syndrome?

Katherine Hall

Abstract: *The most striking feature of Alexander the Great's death is that, despite being extremely unwell, he was reported to have remained compos mentis until just before his death. Combined with evidence that he developed a progressive, symmetrical, ascending paralysis, it is argued that he died from a sub-type of the autoimmune disorder, Guillain-Barré Syndrome (GBS), most likely induced from a Campylobacter pylori infection. GBS could also account for the reported lack of decay of his body, and his death may be the most famous case of pseudothanatos, or false diagnosis of death, ever recorded.*

Introduction

Previous medical explanations for the death of Alexander the Great have been varied and many, but none have been entirely satisfactory and all have had cogent counter-arguments put forward.¹ What has been held in common by almost all of these papers is an emphasis on either one, or both, of two very common symptoms which occurred in his illness; fever and abdominal pain. In comparison, the very unusual description of an extremely unwell man who remains *compos mentis* until moments before he is declared dead has received barely any attention. In addition, there is also the impression given from reading the accounts of his death that Alexander may have developed a progressive, symmetrical and ascending paralysis which first rendered him unable to walk, then unable to move his arms and finally took his ability to speak. This type of paralysis only rarely occurs and the most common reason for it today is a condition called Guillain-Barré Syndrome (GBS). Only Oldbach et. al.² have given this symptom any significance in their paper 'A Mysterious Death', but this was not explored fully. They were not able, due to when it was written, to take into account all the developments in our understanding of GBS over the past thirty years. I will argue that they were correct in drawing attention to this symptom. Further, I will argue that the type of acute, flaccid paralysis documented in Alexander's death is, in fact, GBS. Unlike them, I will conclude that the precipitant cause of his GBS was not typhoid, but much more likely to be a gastrointestinal cause, *Campylobacter jejuni*.³ This would also adequately account for his other symptoms, including a combination of fever and abdominal pain, thus avoiding having to give preference to one historiographic tradition over another.⁴ In addition, there has also been the mystery (largely dismissed as mythology or romantic legend) of the lack of decay in Alexander's corpse once he had died.

¹ Whilst there is no review paper evaluating all of these, Schep et. al. (2014) contains a reasonably up-to-date but not entirely complete overview; it is nevertheless useful.

² Oldbach et. al. (1998).

³ Oldbach et. al. dismissed this as a cause due to the lack of diarrhoea, but I will present evidence that diarrhoea can be absent in over 50% of cases of *Campylobacter jejuni* infection.

⁴ Readers coming to this paper via medicine may not be aware that the historiographic paradoxes of the varying accounts have been a source of much academic discussion in Classics. Examples of literature on this issue includes: Bosworth (1971), Bosworth (1988) especially pages 171-3, and Anson (2009).

Oldbach et. al. briefly mention this was due to a premature diagnosis of death: I will expand upon this extensively in support of this idea. I will discuss the difficulties of knowing when someone has actually died and why this could have been particularly problematic in Alexander's case. I will explain how this example (and possibly the most famous example) of *pseudothanatos*, or the false diagnosis of death, could occur making the diagnosis of GBS as the cause for his death both feasible and plausible.

There are four, major, historical accounts for Alexander's death: Arrian, Plutarch, Diodorus and Curtius.⁵ None of these were contemporary with his life and all of them have issues. I do not intend to enter this debate, as my argument can encompass the variations between these accounts. Medicine is very fond of applying Occam's Razor,⁶ and if all symptoms can be explained by a single diagnosis, doctors argue that this is the most likely conclusion. GBS does provide such a unifying 'diagnosis' to these varying stories.

Alexander's Death

Arrian⁷ and Plutarch⁸ record similar versions of Alexander's death; both authors state they base their accounts on the Royal Journal⁹ of Alexander's court. He became unwell following a drinking party and developed a fever, which was initially mild and intermittent. He continued his duties but increasingly needed to be carried rather than walk. By the fourth night the fever was constant. By the seventh night he was seriously ill and two days later he was extremely ill, but he remained alert and aware; 'When the officers came in he knew them, but said no more; he was speechless.'¹⁰ The following day the army filed past him, and being unable to speak, 'he greeted one and all, raising his head, though with difficulty and making a sign to them with his eyes.'¹¹ After this 'he died.'¹² Plutarch's account agrees essentially with this, making it clearer that Alexander died after eleven days of illness (from the 18th to the 28th of the month called Daesius).¹³ Diodorus' account of Alexander's death is the shortest; after a drinking party Alexander was stricken with a severe pain and died sometime thereafter.¹⁴ Curtius' account is fragmentary—some parts are missing—but he reiterates that Alexander acknowledged his troops as they filed past him with very little movement; 'he maintained the same posture ... until the last salute ... by now, even his voice had started to fail.'¹⁵ Curtius uniquely records that Alexander's body did not putrefy

⁵ To be specific this analysis is based on Arrian's *Anabasis*, Plutarch's *Life of Alexander*, Diodorus Siculus' *Library of History* and Quintus Curtius Rufus' *The History of Alexander*. The earliest account is that of Diodorus (c.80-20 BC) and the latest Arrian (c.86-172 AD). Alexander died in 323 BC.

⁶ For brief outline of Occam's Razor in medicine see Smith (2013).

⁷ Arr. *Anab.* 7.25-26.

⁸ Plut. *Alex.* 76-7.

⁹ Hammond (1988).

¹⁰ Arr. *Anab.* 7.25.6.

¹¹ Arr. *Anab.* 7.26.1.

¹² Arr. *Anab.* 7.26.3. The Greek used is the aorist infinitive ἀποθανεῖν.

¹³ Plut. *Alex.* 76.

¹⁴ Diod. 17.117.1-5. The Metz *Epitome* 99-100 also describes Alexander clutching his stomach, suggesting this pain was abdominal.

¹⁵ Curt. 10.5.3.

for six days despite the Mesopotamian heat.¹⁶ Other, minor accounts also exist, mainly supporting the idea that he was poisoned.¹⁷

Guillain-Barré Syndrome

GBS is the most common cause of acute (i.e. reaching the peak severity of symptoms within four weeks of onset), flaccid (the muscles are relaxed, not tight), ascending form of paralysis in the post-polio world.¹⁸ It is, however, quite rare with an overall incidence of 0.6 to 4 cases per 100,000 people per year but can still be fatal; even with modern treatment the mortality rate is between 4% and 15%.¹⁹ GBS is named after two of three authors—George Guillain and Jean-Alexandre Barré—who published a paper in 1916.²⁰ Originally in French, a translation is available in English.²¹ In this paper, they described two soldiers who developed a sudden onset of paralysis, abnormal sensation and loss of reflexes, but who recovered within a month. Until the 1980s, GBS was thought to be a single type of disease, but now it is known that there are three major variants of this syndrome.²² These variants are reactions to a precipitating cause but can vary significantly in terms of the likely aetiology, geographical distribution, mechanism of injury to the nerves and the symptoms;²³ this is very relevant to understanding the possible relationship between GBS and Alexander's death. All three variants are associated with damage to the peripheral nervous system giving rise to a symmetrical,²⁴ flaccid and ascending paralysis and loss of reflexes beginning with difficulty in walking. Sometimes the autonomic nervous system is involved which results in problems in the body's control of heart rate, blood pressure, and temperature. If the paralysis rises high enough, speech can be lost, pupils dilate, and respiratory arrest can occur.²⁵

GBS is an auto-immune disorder where the patient's own immune system has become confused in differentiating between an invading organism, such as a bacteria, virus, or (very rarely) vaccine products, and the patient's own body.²⁶ Approximately two-thirds of patients have clear-cut evidence for a preceding infection,²⁷ although this can be as high as

¹⁶ Curt. 10.10.9-13.

¹⁷ These include Ehippus (*FGrH* 126 F3 = Athen. 10.434a-b), Nicobule (*FGrH* 127 F1 & 2 = Athen 10.434c & 12.537d), Aelian (*Varia Historia* 3.23), Valerius Maximus (5.1, ext. 1b), Justin (12.13.6-16.1), Orosius (3.20.4), Pseudo-Callisthenes (3.31-33), and the unknown author(s) of the *Metz Epitome* (99-100).

¹⁸ Eldar and Chapman (2014).

¹⁹ Hughes and Cornblath (2005).

²⁰ Kusunoki (2016). The third author was André Strohl. Octave Landry had first described cases of ascending paralysis in 1869 (Wijdicks and Klein (2017), but failed to gain the eponym.

²¹ Guillain et. al. (1968).

²² There are several minor, very rare subtypes such as Miller Fisher Syndrome and Bickerstaff brainstem encephalitis but these are not relevant to the discussion here, as their symptoms differ from those experienced by Alexander.

²³ Willison et. al. (2016).

²⁴ Rarely, it can be asymmetrical.

²⁵ Hauser and Asbury (2005).

²⁶ Wakerley and Yuki (2013).

²⁷ Wakerley and Yuki (2013).

88% in some studies.²⁸ The three major variants are: acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), and acute and sensory axonal neuropathy (AMSAN). AIDP has a different mechanism of injury from the other two variants in that the nerves' protective covering, the myelin sheath, is affected leading to delayed or absent conduction along both motor (which control movement) and sensory nerves. With both AMAN and AMSAN, the nodes of Ranvier which sit at regular intervals along the nerves and concentrate and facilitate the conduction of electrical impulses, are affected; the difference between the two is that with AMAN only motor nerves are affected, and with AMSAN both motor and sensory nerves are affected.²⁹ Most of the medical literature is predicated upon the AIDP variant as this is the most common form seen in Europe and in America, with AMAN and AMSAN only contributing to 5% of cases in these regions.³⁰ Alexander's symptoms of ascending, symmetrical and flaccid paralysis fit best with AMAN, but with this being a rare cause of a rare disease, could this be a diagnosis too far?

Recently there has developed good cause to consider this may be so. AMAN was only first described in 1986.³¹ Until then, medical understanding of GBS had been entirely based on AIDP, which has a slower onset of four to six weeks after the precipitating illness and combined motor and sensory symptoms. In contrast, AMAN has a very rapid onset of within one to six days of contracting the precipitating illness.³² It usually only has motor symptoms such as paralysis.³³ sensory symptoms such as pins and needles, pain, or numbness do not occur or are rare and mild.³⁴ Whilst the AMAN variant is rare in Western Europe and America, it appears to be more common³⁵ the further east one travels towards Eastern Europe and the Near and Middle East; for example, 7% of GBS cases in the Western Balkans,³⁶ 13% in North-West Greece,³⁷ 9-24% in Iran,³⁸ 39% in Pakistan,³⁹ and 44% in India.⁴⁰ The cause of these variations in the relative frequency of GBS variants is unknown,⁴¹ but it is thought this relates to differing rates of local exposure to the organisms that cause the precipitating illness, rather than any hereditary susceptibility. In other words, Alexander's chances of contracting GBS would not be related to his Greek ancestry but to where he was at the time; i.e. Babylon in ancient Persia, or modern day Iraq.

In Iraq, where Alexander died in Babylon, the measured incidence of GBS (in a paediatric population) is 1.33 cases per 100,000 people aged less than fifteen years,⁴² rising

²⁸ Koga et. al. (2001).

²⁹ Hauser and Asbury (2005).

³⁰ Hughes and Cornblath (2005).

³¹ Feasby et. al. (1986).

³² McKhann et. al. (1991).

³³ Hughes and Cornblath (2005).

³⁴ Yadegari et. al. (2014); Ruts et. al. (2008).

³⁵ It is very difficult to get accurate figures for GBS in many countries; see Sejvar et. al. (2011).

³⁶ Peric (2014).

³⁷ Markoula et. al. (2007)

³⁸ Arami et. al. (2006); Yadegari et. al. (2014).

³⁹ Yadegari et. al. (2014).

⁴⁰ Shrivastava et. al. (2017).

⁴¹ Kanda (2016).

⁴² Jasem et. al. (2013).

to an extraordinarily high 22.9 in 100,000.⁴³ GBS accounts for 52.5% of cases of acute flaccid paralysis in Iraq.⁴⁴ Unlike Western studies, Iraqi GBS was often present with fever (55.4%) and in 97% of cases the time of onset to maximum paralysis was within four days.⁴⁵ This short time course is highly suggestive of a high incidence of AMAN. The percentage of GBS of AMAN and AMSAN combined is 18%.⁴⁶ Generally the group in the Iraqi and Iranian regions who contract AMAN are more likely to be male (approximately a 2:1 ratio to females),⁴⁷ are aged significantly younger than people getting AIDP in the West (one study showing an average age of 32 years), and are more likely to develop the syndrome in late spring or summer.⁴⁸ It is striking how well this profile matches that of Alexander, as he was male, 32 years old, and died at the start of summer.⁴⁹

Other conditions which can mimic GBS include: poliomyelitis, botulism, electrolyte disturbance, myasthenia gravis, acute myopathy, diphtheria, porphyria, tick paralysis and vasculitis.⁵⁰ Of these, poliomyelitis is the strongest possible alternative diagnosis. It is strongly associated with fever, but the paralysis that occurs usually does so within 24 hours of onset of symptoms and is most commonly asymmetrical. If extensive paralysis does occur, it is usually following an initial phase with headache, and a lowered level of consciousness or coma due to an associated meningitis.⁵¹ The time course of the illness does not fit with Alexander's illness, nor does the way he remained cognitively intact even at the end of his illness. Botulism does cause a symmetrical paralysis but this descends, not ascends, from the head to the extremities.⁵² Electrolyte disturbances, which can occur after excessive alcoholic intake (and Alexander had indulged just prior to his illness), are unlikely causes as they also result in a reduced level of consciousness and are not associated with fever.⁵³ Myasthenia gravis is strongly associated with fatigue of muscles with recurrent use, and a waxing and waning course over several years before diagnosis, all of which makes this to be an unlikely cause for Alexander's death.⁵⁴ Acute myopathy due to alcohol intoxication causes seizures and coma, neither of which was present in Alexander's case.⁵⁵ Diphtheria causes a severe sore throat,⁵⁶ which was absent in Alexander's case. Porphyria is an inherited disorder of the metabolism of heme, a by-product of haemoglobin

⁴³ Al-Dabbas (2016). This was also in a paediatric population, but is per 100,000 hospital admissions.

⁴⁴ Jasem et. al. (2014). In ancient times *poliomyelitis* is likely to have been a much more common cause.

⁴⁵ Jasem et. al. (2013). Western medical textbooks, based on the high proportion of AIDP in their countries, often discount fever as a symptom of GBS. For example, Hauser and Asbury (2005), page 2513, write 'Fever and constitutional symptoms are absent at the onset [of paralysis], and, if present, cast doubts on the diagnosis.' Hasan et. al. (2014) found the mean time from onset of paralysis to peak paralysis to be 5.4 days in a paediatric population in Iraq.

⁴⁶ Hasan et. al. (2014).

⁴⁷ Benamer and Bredan, (2014).

⁴⁸ Yadegari et. al., (2014); Abdul-Kareem et. al. (2004). Al-Dabbas (2016) found a bimodal distribution with peaks in winter and summer (including June, the time of Alexander's death).

⁴⁹ Alexander died on 11 June, 323 BC; for discussion of this date, see Depuydt (1997).

⁵⁰ Eldar and Chapman (2014); Hughes and Cornblath (2005).

⁵¹ Cohen (2005a).

⁵² Abrutyn (2005).

⁵³ Singer and Brenner (2005).

⁵⁴ Drachman (2005).

⁵⁵ Brown and Mendell (2005), 2539.

⁵⁶ Holmes (2005).

degradation; it can cause an acute crisis, especially after alcohol ingestion, but is rarely fatal and is always associated with a fever.⁵⁷ Alexander regularly drank and there was nothing to suggest previous porphyric crises in the past (porphyria has previously been suggested⁵⁸ and repudiated as a cause for Alexander's death⁵⁹). Tick paralysis is extremely rare, almost always occurs in children, and there is no fever.⁶⁰ Vasculitis is a usually long-term, complex disorder primarily affecting the connective tissues and causing symptoms of muscle and joint pain.⁶¹ The symptoms and time course of Alexander's illness make this diagnosis unlikely. If Alexander did suffer from acute ascending, symmetrical and flaccid paralysis, GBS is the most likely diagnosis.

Precipitant Causes of Guillain-Barré Syndrome

GBS can be associated with a number of infections preceding its onset. These include: *Campylobacter jejuni*,⁶² Cytomegalovirus,⁶³ *Salmonella typhi* (typhoid fever) and paratyphi,⁶⁴ Epstein-Barr (glandular fever) virus,⁶⁵ *Haemophilus influenzae*,⁶⁶ *Mycoplasma pneumoniae*,⁶⁷ Varicella zoster (chicken pox),⁶⁸ Influenza A and B and Parainfluenza 1 viruses,⁶⁹ Adenovirus,⁷⁰ Herpes simplex,⁷¹ Hepatitis E,⁷² Dengue fever,⁷³ Chikungunya virus,⁷⁴ Leprosy or Hansen's disease,⁷⁵ West Nile Virus,⁷⁶ and the Zika virus.⁷⁷ But association does not necessarily mean causation and some of these possible causes can be eliminated by looking at whether the chance of getting GBS is higher with these illnesses than in the general population: on this basis infections with *Haemophilus influenzae*, Influenza A, B and Parainfluenza 1 viruses, Adenovirus, Herpes simplex virus, and Varicella zoster virus, do not occur any more frequently in GBS patients than expected in the general population

⁵⁷ Desnick (2005).

⁵⁸ Schnorf (1998).

⁵⁹ Schep et. al. (2014).

⁶⁰ Edlow and McGillicuddy (2008).

⁶¹ Sneller et. al. (2005).

⁶² Wijdicks and Klein, (2017).

⁶³ Orlikowski et. al. (2011).

⁶⁴ Wakerley and Yuki (2013).

⁶⁵ Tam et. al. (2007).

⁶⁶ Yuki and Shahrizaila (2011).

⁶⁷ Eldar and Chapman (2014).

⁶⁸ Yadegari et. al. (2014).

⁶⁹ Kutlesa et. al. (2010), Tam et. al. (2007), Jacobs et. al. (1998).

⁷⁰ Jacobs et. al. (1998).

⁷¹ Jacobs et. al. (1998).

⁷² Geurtsvankessel et. al. (2013), Van Den Berg et. al. (2014).

⁷³ Solomon et. al (2000), Chen and Lee (2017).

⁷⁴ Agarwal et. al. (2017), Mehta et. al. (2018).

⁷⁵ Mittal et. al. (2017).

⁷⁶ Ahmed et. al. (2000), Marr and Calisher (2003).

⁷⁷ Musso et. al. (2015), Brasil et. al. (2016).

and are unlikely to be true causes.⁷⁸ On the same basis, *Campylobacter jejuni*, and Cytomegalovirus and Epstein-Barr viruses are highly likely to be a cause of GBS, and *Mycoplasma* moderately so.⁷⁹

Further possibilities may be eliminated by considering what symptoms and signs Alexander did not have, although here is where the difficulty of dealing with the historical sources becomes relevant: is a symptom absent from the record because it did not occur, or did it occur but was either not recognised or not recorded?⁸⁰ There was no report he suffered any respiratory symptoms, enlarged glands or rash. *Mycoplasma* causes a pneumonia-like disease. Cytomegalovirus is associated with sneezing and a sore throat, especially in the early days of the illness, and with a rash (although this is less common in adults).⁸¹ Epstein-Barr virus causes a sore throat in 75% of patients, widespread enlarged lymph nodes in 95%, and pharyngitis or tonsillitis in 82%.⁸² These are unlikely precipitants. The sources do not mention that Alexander became jaundiced, although this symptom was well described by ancient doctors,⁸³ and the lack of this symptom would make Hepatitis E unlikely.⁸⁴ Dengue fever is characterised by the sudden onset of a high fever (not the slow onset of an increasingly severe fever as Alexander suffered), together with headache, back pain, a rash and red eyes.⁸⁵ Alexander was not recorded as having any of these symptoms. Zika is unlikely to have been present in ancient times; it was only first identified in 1947, and is usually very mild and lasts two to five days, again with headache and red eyes.⁸⁶ West Nile Virus has previously been suggested as the cause for Alexander's death,⁸⁷ but again its symptomatology of confusion accompanying the paralysis and usually only being fatal in patients over 75 tells against it.⁸⁸ Chikungunya virus was first described in 1824, and starts with a sudden high fever, headache and severe arthritis.⁸⁹ Leprosy is an ancient disease, having been first described in 6th century BC Indian texts; it takes years to slowly develop, has marked, characteristic skin and limb changes, is not associated with a sudden fever, and is not fatal.⁹⁰ These, too, are unlikely precipitants.

This leaves the two most likely contenders: *Campylobacter* infection and *Salmonella* (typhi and paratyphi). *Campylobacter* is a world-wide bacteria which causes a gastroenteritis and was first described as being associated with GBS in 1982;⁹¹ it most

⁷⁸ Jacobs et. al. (1998).

⁷⁹ The other causes were not analysed because they were not known to be associated with GBS at the time of publication of this paper in 1998.

⁸⁰ These questions are unanswerable, but to make any meaningful progress in my argument I have made the assumption that the absence of a symptom or sign from the historical record means it was absent from Alexander's illness.

⁸¹ Jacobs et. al. (1998).

⁸² Cohen (2005b).

⁸³ Nutton (2013), 29.

⁸⁴ Dienstag and Isselbacher (2005), 1831.

⁸⁵ Peters (2005), 1164.

⁸⁶ Paixão et. al. (2016).

⁸⁷ Marr and Calisher (2003).

⁸⁸ Schep et. al. (2014)

⁸⁹ Kucharz and Cebula-Byrska (2012).

⁹⁰ Gelber (2005).

⁹¹ Rhodes and Tattersfield (1982).

frequently results in the AMAN variant.⁹² Identification of *Campylobacter* species in archaeological studies on ancient dental plaque show that this family of bacteria has been with us for millennia.⁹³ It is the most common cause for GBS worldwide,⁹⁴ and is the most common cause for the AMAN variant,⁹⁵ especially in the region where Iran borders Iraq—the very place where Alexander died.⁹⁶ Approximately one in 3000 cases of *Campylobacter* infection is associated with GBS.⁹⁷ GBS can begin as quickly as within one day of infection, although the mean duration between the precipitating infection and the onset of paralysis is ten days with this cause.⁹⁸ Although GBS is a rare complication of *Campylobacter* infection,⁹⁹ the associated risk of acquiring GBS can vary and appears related to the serotypes (subtypes) of the bacteria,¹⁰⁰ and also to the severity of the initial infection.¹⁰¹ *Campylobacter*-induced GBS is most commonly sporadic with only isolated cases occurring, although some epidemics have also been documented.¹⁰² It is a waterborne disease and can also be transmitted by animals, especially in milk from affected cows and chickens.¹⁰³ It most commonly causes a diarrhoeal illness with fever and abdominal pain, lasting from one to two weeks, but infections can occur without these symptoms; in one series only 24% had diarrhoea, 38% abdominal pain and 14% fever.¹⁰⁴ Thus, whether or not the sources record Alexander having or not having diarrhoea is irrelevant to making this diagnosis. Although his fever would be unusual, this could have been related to the severity of his infection.

Salmonella typhi, which causes typhoid fever, is still endemic in Iraq and an outbreak was reported there as recently as 2014.¹⁰⁵ It, and *Salmonella paratyphi*, cause similar illnesses although paratyphi infections tend to be milder; in the pre-antibiotic era, *S. typhi* had a mortality rate of 16.1% versus 1.5% for paratyphi for those aged 31 to 40 years.¹⁰⁶ Both organisms can cause typhoid fever, which begins with cough, headache, rigors and sore throat (none of which Alexander was reported having), progressing onto high, sustained fever in 75% and abdominal pain in 40%; 30% also develop a distinctive rash by the end of the first week of symptoms.¹⁰⁷ Later complications, usually in weeks three and four, include gastrointestinal perforation and peritonitis; only extremely rarely does this occur in week

⁹² Willison et. al. (2016).

⁹³ Warriner et. al. (2014).

⁹⁴ Moore et. al. (2005).

⁹⁵ Wakerley and Yuki (2013), Kusunoki (2016), Wijdicks et. al. (2017).

⁹⁶ Barzegar et. al. (2008). 52% of patients with AMAN or AMSAN had had a preceding *Campylobacter* infection.

⁹⁷ Moore et. al. (2005).

⁹⁸ Takahashi et. al. (2005).

⁹⁹ McCarthy et. al (1999), Tam et. al. (2006).

¹⁰⁰ Takahashi et. al. (2005).

¹⁰¹ Baker et. al. (2012).

¹⁰² McKhann et. al. (1991).

¹⁰³ Young et. al. (2007).

¹⁰⁴ Ho et. al. (1995). This shows Oldbach et. al. (1998) is incorrect in saying that the infection could not have been *Campylobacter* due to the lack of diarrhoea.

¹⁰⁵ Dworkin et. al. (2014).

¹⁰⁶ Gadeholt and Madsen (1963).

¹⁰⁷ Lesser and Miller (2005).

one.¹⁰⁸ Alexander's fever was not sustained but slowly worsened; in addition, if he had developed peritonitis and with it septic shock, he would have rapidly developed a 'muttering delirium'¹⁰⁹ typical of these cases. Typhoid fever also requires human-to-human transmission as there are no animal vectors and it is rare for isolated cases to occur. No epidemics were reported concurrently with Alexander's death (although the significance of his death could well have outshone those of the *hoi polloi*, no matter how many).

Typhoid fever has extremely rarely been associated with GBS. The first case was reported in 1969 in a 27-year-old male.¹¹⁰ Since then, it has also been reported in a 15-year-old female,¹¹¹ a 47-year-old woman,¹¹² a 53-year-old man,¹¹³ a 41-year-old man,¹¹⁴ a 43-year-old man,¹¹⁵ as well as two paediatric cases.¹¹⁶ Of the adult cases, four had pure motor loss indicative of AMAN, and two mixed motor and sensory losses. The paucity of cases must call into question whether typhoid fever is actually a cause, or merely an association.¹¹⁷ In addition, death is usually by gastrointestinal perforation in the third to fourth week of the illness.¹¹⁸ Data comparing outcomes in the pre-antibiotic era of 1,119 cases of typhoid fever and 1,528 cases of paratyphoid fever show that in the first week of the illness only 0.7% of typhoid patients developed this complication and no cases of this occur with paratyphoid illness. Those who died from typhoid did so 6 to 76 days after the start of their illness, and with paratyphoid this was between 11 and 66 days. Whilst GBS was not specifically mentioned in this paper, polyneuritis (inflammation of multiple nerves causing motor and/or sensory disturbance which could be cases of GBS) occurred in 0.4% of typhoid and 0.2% for paratyphoid cases and gives some idea of the frequency of GBS; far lower than *Campylobacter* which from this analysis remains the most likely cause for GBS in Alexander's case.

Proving Death in the Ancient Greek World.

It may seem counter-intuitive but death can be difficult to prove even in modern times.¹¹⁹ Celsus comments that Democritus had studied the diagnosis of death and had collected evidence of people coming back to life after having been declared dead, so much so that Democritus could 'not admit that there could be any sure signs of approaching death.'¹²⁰

¹⁰⁸ Gadeholt and Madsen (1963) report gastrointestinal perforation and peritonitis in the pre-antibiotic era occurring in only 0.7% of patients with typhoid fever in the first week.

¹⁰⁹ Lesser and Miller (2005), 899. Chanmugam and Waniganetti (1969) also describe a 'typhoid stupor'.

¹¹⁰ Chanmugam D and Waniganetti (1963).

¹¹¹ Samantray (1977).

¹¹² Aldrey et. al. (1999).

¹¹³ Berger et. al. (1986).

¹¹⁴ Donoso et. al. (1988).

¹¹⁵ Subrata (2012).

¹¹⁶ Mehndiratta et. al. (2012), Kapoor et. al. (2014).

¹¹⁷ Jacobs et. al. (1998) did not include typhoid fever in their statistical analysis, but if such analysis was applied it would be doubtful that it would reach statistical likelihood for causation.

¹¹⁸ Schep et. al. (2014).

¹¹⁹ Two anaesthetists called for further review of this as recently as 2013 (see <http://www.bbc.com/news/health-22730360> accessed 17 April, 2018).

¹²⁰ Cels. *Med.* 2.13-8.

Even up until the eighteenth century, the action of the heart and pulsation was still considered subordinate to respiration,¹²¹ probably because of the commonplace observation that when the breathing stopped, the heart usually then followed, whereas the reverse was harder to observe. Until the 19th century AD, it was generally ‘concluded that advanced putrefaction of the entire body was the only valid single test to exclude apparent death.’¹²² It is not surprising then that the embalmers were reluctant to commence working on Alexander’s body when it showed no signs of decomposition.¹²³

There is very little extant evidence as to how death was actually determined in the Ancient Greek world. While the Hippocratic corpus, particularly in *Prognosis*, does discuss what was termed a ‘sign of death’ (τὸ σημεῖον Φανατῶδεις),¹²⁴ this actually refers to a sign of *impending* death, not diagnosing actual death.¹²⁵ It has been argued that in the ancient Greek world (not specifically but including the time of Alexander), the presence or absence of a heartbeat was considered the *sine qua non* for determining death.¹²⁶ But at the time of Alexander’s death this was almost certainly not the case. He died at a time when pulsation or ‘throbbing’ was considered ‘an unnatural and mostly random phenomenon, a sign of disease simply by virtue of its existence ... as opposed to natural phenomena such as respiration, urine and sleep.’¹²⁷ Certainly, the Ancient Egyptians had identified the heart as the central organ of the cardiovascular system,¹²⁸ and some believe that the Egyptians also linked the heart with the pulse.¹²⁹ Aristotle noted that the heart was the first organ to form and the last to stop.¹³⁰ But Hippocrates did not advocate taking the pulse from the wrist (although he writes of observing temporal artery pulsating in fever),¹³¹ and the link between peripheral pulses and heartbeat was poorly understood by Greeks, including Aristotle.¹³² He did connect the heart with respiration and pulsation, but believed the peripheral vascular system had its own active, intrinsic pulsation rather than passively distending in response to cardiac pulsation.¹³³ After the time of Alexander’s death Praxagoras identified that only arteries pulsated,¹³⁴ but five centuries would then pass before Rufus of Ephesus correctly linked peripheral pulsation to the heart, not to the

¹²¹ Cooper et. al. (2006).

¹²² Powner et. al. (1996), 1220. Pernick’s chapter (1988) also gives an informative overview of the history of how death was diagnosed (or not) over the ages.

¹²³ Curt. 10.10.13.

¹²⁴ Hp. *Prog.* 2.22. Also see Hp. *Aph.* 7. end fragment (page 219 of Loeb edition), which lists such signs as a retracted right testicle, blackened nails, cold toes, livid fingertips, dark, swollen lips, and swollen, puffed up bowels as signs of death.

¹²⁵ Bondeson (2001), 18.

¹²⁶ Bondeson (2001), Powner et. al. (1996), Pernick (1988). The first two sources appear to make Pernick the basis for their claim. Pernick cites Wiener PP (ed.), *Dictionary of the History of Ideas*, 1968, New York, Scribner, not the primary Ancient Greek sources.

¹²⁷ Lewis (2015), 346.

¹²⁸ Loukas et. al. (2016).

¹²⁹ Loukas et. al. (2007).

¹³⁰ Harris (1973), 173.

¹³¹ Cheng (2001).

¹³² Harris (1973), 124-135.

¹³³ Shoja et. al. (2008).

¹³⁴ Ghasemzadeh and Zafari (2011).

vessels themselves.¹³⁵ The relevancy and importance of the pulse in deciding if life was extinct was not truly appreciated until Galen's seminal works on the subject.¹³⁶ At the time of Alexander's death, therefore, the absence of pulsation was unlikely to be considered a sign of death.

Given this relative silence of medical texts on the determinants of diagnosing death, non-medical literature was also reviewed.¹³⁷ Here, too, the evidence remains generally scanty. Vermeule's analysis of death and its relationship to early Greek Art and Poetry fails to consider the aspect of determining when one is dead,¹³⁸ as does Christiane Sourvinou-Inwood in her book *Reading Greek Death*.¹³⁹ Religious piety and convention generally restricted the visual act of killing onstage in tragedies (although the process of dying could be presented),¹⁴⁰ and prior to either Sophocles or Euripides there were few depictions of death-bed scenes.¹⁴¹ Two rare examples are Eurydice's suicide which mentions changes in the eyes; 'By the altar, with a sharp-whetted sword, she struck until her eyes went slack and dark,'¹⁴² and the death of Sophocles' Ajax in the eponymous play which is less subtle. Having fallen on his sword, he subsequently 'spurt[s] the darkened gore of his self-inflicted slaughter up his nostrils and out of the bloody gash.'¹⁴³ Death from acute trauma is usually self-evident and non-problematic, which may be why, despite describing 240 cases of death in *The Iliad*, Homer did not specifically mention any means by which the diagnosis of death was confirmed.¹⁴⁴ In contrast, one Hippocratic aphorism states 'in the case of acute diseases to predict either death or recovery is not quite safe.'¹⁴⁵ The clearest example in literature for the determination of death is that of Socrates in *Phaedo*: 'the attendant uncovered him [Socrates]; his eyes were fixed. And when Crito saw it, closed his mouth and eyes. Such was the end.'¹⁴⁶

Other references for signs of death included the extinguishing of *thymos*, which was most commonly thought to reside in the chest, and a loosening of the limbs.¹⁴⁷ In Hippocrates' *Aphorisms* it states that 'the boundary of death is passed when the heat of the soul has risen above the navel to the part above the diaphragm.'¹⁴⁸ Another more self-

¹³⁵ Bedford (1951).

¹³⁶ Horine (1941). See especially pages 220-221.

¹³⁷ This presents a synopsis of what is relatively easily found, not a full and complete account. This would be fascinating but beyond the scope of the current article and possibly frustrating; Garland (1985),16, states that such descriptions of the moments of death itself are 'in both art and literature extremely rare.'

¹³⁸ Vermeule (1979), 5.

¹³⁹ Sourvinou-Inwood (1995).

¹⁴⁰ Sommerstein (2010), chapter 2.

¹⁴¹ Vermeule (1979), 10.

¹⁴² *Soph. Ant.* 1301. More strictly this refers to eyelids, rather than eyes, as in the original it reads: 'ή δ' ὀξυθήκτω βωμία περι ξίφει λύει κελαινὰ βλέφαρα,...'

¹⁴³ *Soph. Aj.* 918-9.

¹⁴⁴ Garland (1981).

¹⁴⁵ *Hp. Aph.* 2.19. The Greek reads: 'Τῶν ὀξέων νοσημάτων οὐ πάμπαν ἀσφαλέες αἱ προαγορεύσεις, οὔτε τοῦ θανάτου, οὔτε τῆς ὑγιείης.'

¹⁴⁶ *Plat. Phaedo* 118a. reads; 'ὁ ἄνθρωπος ἐξεκάλυψεν αὐτόν, καὶ ὃς τὰ ὄμματα ἔστησεν· ἰδὼν δὲ ὁ Κρίτων συνέλαβε τὸ στόμα καὶ τοὺς ὀφθαλμούς. "Ἦδε ἡ τελευτή.'

¹⁴⁷ Garland (1981).

¹⁴⁸ *Hp. Aph.* 7.(end fragment).26-9. The Greek reads: 'ὄρος δὲ θανάτου· ἐπειδὴν τὸ τῆς ψυχῆς θερμὸν ἐπανάλθη ὑπὲρ τοῦ ὀμφαλοῦ ἐς τὸ ἄνω τῶν φρενῶν.'

evident sign of death than the cessation of cardiac activity is the cessation of respiratory effort. As Alcestis lies dying on behalf of her husband, it is commented that ‘she has scarcely any breath within her.’¹⁴⁹ Homer refers to the *psyche* being released or leaving the body at death, most commonly via the mouth or the wound.¹⁵⁰ Despite his remarks on the primacy of the heart (see first paragraph this section), Aristotle also stated that breath ‘evidently has control over life and death, for it results synchronously that when respiring animals are unable to breathe they perish.’¹⁵¹ The large philosophical and medical literature concerning *pneuma* underscores its conceptual importance to both these fields, with a full analysis being beyond the scope of this article. But given *pneuma*’s pre-eminence in the thinking in both these fields, it could be surmised that this would have been the early sign of death.

Unlike in modern times, the determination of being dead was probably not done by doctors, but more likely performed by family, relatives or non-medical attendants. There is no advice in the Hippocratic corpus that a doctor needed or should be involved in declaring death.¹⁵² No medical certificate was required, although one was in Roman Egypt where bureaucratic processes required such a certificate to be produced to avoid posthumous taxation.¹⁵³ Whilst death from trauma would often be very obvious, death from an internal cause with an outwardly intact body could be far more problematic. For Alexander, only Diodorus mentions physicians being present early in his illness¹⁵⁴ and neither he, Arrian, Plutarch, Diodorus or Curtius mention any at his death. The priests of Serapis, who may also have had a doctoring role,¹⁵⁵ perhaps wisely stayed away in their temple.

With proving death being problematic, and often left to the layperson, perhaps it is not surprising that stories of people reviving after having been declared dead have been recorded in classical literature.¹⁵⁶ The stories of the *Ephesian Tale*, *Metamorphoses* of Apuleius and the anonymously authored *Apollonius, Prince of Tyre* all employ a character who is mistaken for being dead.¹⁵⁷ Then there is the tale of Er in Plato’s *Republic*, an Armenian soldier who wakes up on the funeral pyre ten days after being found and thought dead on the battle-field, which clearly describes a case of non-putrefaction of a ‘corpse’.¹⁵⁸ Pliny the Elder, in his *Natural History*, had a whole section in Book Seven entitled ‘Persons Who Have Come To Life Again After Being Laid Out For Burial.’¹⁵⁹ He described several instances of people being placed on funeral pyres only to waken, including Acilius Aviola, Lucius Aelius Lamia and Gaius Aelius Tubero. Although the date for the death of the consul Acilius

¹⁴⁹ Eur. *Alc.* 205. The Greek reads: ‘καίπερ σμικρὸν ἐμπνέουσ’ ἔτι.’

¹⁵⁰ Garland (1981).

¹⁵¹ Aristot. *Resp.* 11.472b.

¹⁵² Bondeson (2001), 18, argues that within the Hippocratic corpus that there are specific instructions for a doctor to withdraw when a patient is considered terminally ill, but there is no citation and it is not clear to which section is being referred. In Hippocrates’ *The Art* (ΠΕΡΙ ΤΕΧΝΗΣ) 8, the author discusses whether doctors should ‘undertake desperate cases’ but rather than being abandoned, the author argues that attempts should not be started in the first place.

¹⁵³ Garland (1985), 17.

¹⁵⁴ Diod. 17.117.2-3.

¹⁵⁵ Geller (2010), see chapter two: ‘Who Did What to Whom’.

¹⁵⁶ Amundsen (1974).

¹⁵⁷ Amundsen (1974).

¹⁵⁸ Plat. *Rep.* 10.614-21.

¹⁵⁹ Plin. *Nat.* 7.53

Aviola is not absolutely certain,¹⁶⁰ he probably died in Pliny's childhood, and the story of this would certainly have had a strong impression on a growing lad. Lucius Lamia was accorded 'a censorian funeral',¹⁶¹ so that if Pliny was correct, Lamia's subsequent reanimation and final death (the pyre was burning too fiercely for him to be rescued) would have been a public event, witnessed by thousands, possibly including a young Pliny. These, in addition to several other more plebeian cases recorded by him, led Pliny to rather gloomily conclude: 'we cannot be sure of anything, no, not even that a person is dead.' These stories have parallels with the *disthanees*, the mortals who visit Hades and yet still return.¹⁶² A much later case of misdiagnosed death was also reported from mediaeval Persia.¹⁶³

Guillain-Barre Syndrome as a Cause for *Pseudothanatos*

Thus in the Ancient World, determining when a patient was dead could be very problematic, especially if the cause was not related to trauma, and especially since often it was lay people who made this judgement. Whilst there is no evidence in the ancient literature for GBS being a cause for misdiagnosed death or *pseudothanatos*, since 1979 there have been a number of cases in the modern medical literature which show GBS can cause particular difficulties in the diagnosis of death, even with modern medical technology. A small number of cases of GBS¹⁶⁴ have been reported developing a deep coma, unresponsive to sight, sound or painful stimuli, with no voluntary respiratory effort. Pupils become dilated and unresponsive to light and no reflexes can be elicited. Formal brain-stem death testing shows that death—as defined by a non-functioning brain-stem—has occurred. Fortunately for these people, one of the essential criteria for brain-death before removing ventilator and intensive care support is to be certain of the cause for the brain-stem non-function, and that reversible causes are eliminated. Due to uncertainty about the cause and suspicions of GBS being present, intensive care support was not withdrawn, and further diagnostic tests were done which confirmed GBS was indeed present. Of the 15 cases so far described, 12 (80%) have been male with an average age of 47 years. What is perhaps more extraordinary is that despite being thus afflicted, the cerebral effects proved to be fully reversible with 13 out of 15 cases who survived regaining their brain function intact, albeit with no memories of the time they were so unwell.¹⁶⁵ Of course, these cases involve patients who are in an intensive care unit, and who have their respiration, circulation and temperature closely monitored and treated continuously. How could someone like Alexander, completely or near-completely paralysed, possibly survive any length of time?

¹⁶⁰ Gallivan (1978).

¹⁶¹ Tac. Ann. 6.27.

¹⁶² Vermeule (1979), 132.

¹⁶³ Agutter et. al. (2013).

¹⁶⁴ The following details are a summary of Carroll and Mastaglia (1979), Coad and Byrne (1990), Hassan and Mumford (1991), Vargas et. al. (2000), Friedman et. al. (2003), and Joshi et. al. (2008).

¹⁶⁵ This is not to be confused with the 'Lazarus Syndrome' where patients are declared dead after having a cardiac arrest and failed cardiopulmonary resuscitation only to be found to be alive some time later. These GBS patients had uninterrupted cardiac function. For further details on the Lazarus Syndrome see Braun, Herff, and Paal (2011) and Sahni (2016).

To grasp Alexander's condition it is necessary to understand two determinants of survival: the oxygen needs of the body, and the ability of the body to absorb oxygen without any muscular effort. The oxygen needs of the body are determined by the energy needs. These comprise three broad categories: the basal metabolic rate (BMR) or resting energy expenditure (REE) which is the energy required for involuntary, basic cellular survival associated with functions like respiration and circulation; the energy associated with voluntary physical activity; and the energy that is required to absorb and digest one's food.¹⁶⁶ Normally the BMR/REE accounts for 70% of all energy needs with physical activity and digestion accounting for 20% and 10% respectively. When paralysed and not being fed, as would be the case for Alexander, his energy expenditure would automatically be reduced by 30%. In addition, there would be a further reduction in his BMR/REE due to the paralysis of all his muscles, which would be in a flaccid, relaxed state and rapidly wasting from not being innervated. There have not been any studies in the BMR/REE of patients with GBS, but in studies with patients who have had a high spinal cord lesion resulting in paralysis of all four limbs (tetraplegia) there is a marked drop in the BMR.¹⁶⁷ Assuming a pre-illness weight of 75kg, at age 32 his BMR would have been about 1734+/-167 kcal/day before his illness.¹⁶⁸ When paralysed in all four limbs his BMR would have dropped to 1324 kcal/day¹⁶⁹; it may have been even lower as no studies have ever been done in the acute phase of the first month of spinal cord injury. Given a premorbid total daily energy expenditure of 2476 kcal/day,¹⁷⁰ having his energy requirements reduced to 1324 kcal/day represents a reduction of 54% of his energy requirements. Furthermore, apart from paralysis, there is a second mechanism by which Alexander's metabolism could have been reduced further: hypothermia. Normally one regulates one's temperature within fairly tight parameters. GBS can lead to destabilisation of this by affecting the autonomic nervous system. Despite having a fever earlier in the illness, once Alexander had reached the stage of being extensively paralysed his GBS could have caused his temperature to subsequently fall. Abnormal sweating¹⁷¹ and poor regulation of body temperature can occur with GBS and is more likely to occur if the symptoms are severe.¹⁷² Hypothermia does have its advantages; it causes more blood to be shunted to the vital organs of heart, brain and lungs away from the limbs and it also protects the brain by slowing metabolism.¹⁷³ Put simply, he would need less oxygen to exist.¹⁷⁴

The second factor to consider concerning Alexander's miraculous non-putrefaction is how oxygen is absorbed into the body.¹⁷⁵ Although mechanical effort in the form of respiratory muscles contracting is needed to shift large volumes of air in and out of the lungs, oxygen is absorbed into the body by diffusing down a concentration gradient; it

¹⁶⁶ Prentice (2014).

¹⁶⁷ Cox et. al. (1985) and Nevin et. al. (2016).

¹⁶⁸ See Table 6.5 in Prentice (2014).

¹⁶⁹ This BMR estimation comes from Cox et. al. (1985), only one of two studies investigating BMR/REE in patients with spinal cord lesions within six months of injury.

¹⁷⁰ Calculated from a premorbid BMR/REE of 1734 kcal/day representing 70% of his energy needs.

¹⁷¹ Eldar and Chapman (2014).

¹⁷² Asahina et. al. (2002).

¹⁷³ Gauci et. al. (2015).

¹⁷⁴ Mirzakhani and Nozari (2011), see especially page 152.

¹⁷⁵ Arroyo and Schweickert (2015). For a physiology textbook it is remarkable for its accessibility and ease of reading, even for a layperson.

flows from an area of high concentration (the air) across the lung membrane to an area of lower concentration (the blood). This movement is entirely passive and driven by the degree of difference in the two concentrations. It is not related to actual chest wall movements at all.¹⁷⁶ Under normal circumstances, the oxygen concentration in the lungs needs to be maintained and refreshed by breathing to ensure an adequate concentration gradient and flow of oxygen into the blood. Even when fully paralysed, this oxygen concentration gradient remains from mouth to alveoli and continues to move passively (but in a far diminished amount) into the circulation. If this situation is combined with the condition of low oxygen demand due to reduced metabolic need, theoretically prolonged survival may be possible with very reduced respiratory effort.¹⁷⁷

As the paralysis progressively ascended his body, Alexander's respiratory muscles would have become progressively paralysed. This would have reduced the size of his breaths (tidal volume) and the visible use of his muscles. Eventually he would have lost the function of his chest wall muscles and become dependent on his diaphragm and neck muscles to shift air into his lungs. This type of breathing can be very difficult to see, especially if the breaths are small and infrequent. With a reduced metabolism, he would make less carbon dioxide which is the primary driver for breathing, and this would reduce his respiratory rate further. His body temperature autoregulation may have failed and, despite the heat of a Babylonian summer, his body temperature may have dropped, reducing his metabolism yet again.

As Alexander lay critically ill his staff, more used to dealing with death due to battle carnage, trauma, and exsanguination, would not be well trained to diagnose the moment of his passing. His GBS could have resulted in a situation where he was only breathing infrequently and with very small tidal volumes, unable to be seen by the naked eye easily. They would be unlikely to have checked for a pulse. His body could have cooled somewhat, again suggesting death, and his pupils could have become fixed and dilated again mimicking these signs of death. His miraculous bodily preservation, rather than being due to his innate divinity, could well have been because of the far more prosaic reason that he was not dead (yet). The embalmers were correct in hesitating, although it is very likely Alexander was in a deep coma by this stage and would have had no awareness when they began their task.

Conclusion

By concentrating on the very unusual combination of bilateral, flaccid, ascending paralysis with the absence of coma, confusion or unconsciousness, it is both feasible and plausible that GBS was the cause of Alexander's demise. It is most likely that if this were the case, that he had the AMAN variant, which produced a paralysis without associated sensory symptoms. The AMAN variant fits best with Alexander's recorded symptoms and is supported by modern day epidemiological evidence. The most likely cause for his GBS

¹⁷⁶ This is why patients can be kept well oxygenated during brain-stem death testing when the ventilator is stopped to see if they can breathe on their own, simply by passing a catheter into the trachea (windpipe) and entraining 6 litres of oxygen per minute.

¹⁷⁷ I am extrapolating from the data for survival after drowning. Not surprisingly, it is difficult to find volunteers for the necessary experiments to prove this and ethics committee approval would probably be problematic too.

would be *Campylobacter jejuni* infection given its ubiquitous world-wide distribution, evidence of existence contemporaneously with Alexander's time, and being the most frequent cause for GBS world-wide. His death was further complicated by the difficulties in diagnosing death in ancient times, a problem which can still occur in modern times. This, along with the nature of his illness and lowered oxygen demands which would result there from, explain his otherwise miraculous preservation of his body. The elegance of this diagnosis for the cause of his death is that it explains so many of otherwise diverse elements and renders them into a coherent whole.

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