Søren Kierkegaard (1813–55): a bicentennial pathographical review

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Abstract
Researchers in the fields of psychology, psychiatry, medicine and theology have made exhaustive efforts to shed light on the elusive biography/pathography of the great Danish philosopher Søren Kierkegaard (1813–55). This ‘bicentennial’ article reviews his main pathographical diagnoses of, respectively, possible manic-depressive [bipolar] disease, epilepsy, complex partial seizure disorder, Landry-Guillain-Barré’s acute ascending paralysis, acute intermittent porphyria with possible psychiatric manifestations, and syphilidophobia.

Keywords
Complex partial seizure disorder, Landry-Guillain-Barré’s disease, manic-depressive disease, pathography, porphyria, Søren Kierkegaard, syphilidophobia

In this bicentennial year of the birth of the great Danish philosopher, Søren Aabye Kierkegaard, born in Copenhagen on 5 May 1813, it is timely to present a review of his pathography. To paraphrase Henning Fenger (1980), detectives in the fields of psychology, psychiatry, medicine and theology have turned over every scrap of paper in order to shed light on his life. Therefore, the question is whether any ‘new’ sources or evidence have been retrieved or established with which to enlighten us further with respect to his physical and mental health.

Kierkegaard’s physical and mental illnesses in retrospect
Fenger (1980) wrote that Kierkegaard’s ‘Papers’ (Thulstrup, 1968) contain many references to indispositions, headaches, insomnia, nervous tension and nervous pain, heliophobia connected...
with conjunctivitis, and that around 1851 he had certainly suffered from intestinal colic. To this list Joakim Garff (2007) added that Kierkegaard suffered from cramps, urinary difficulties and recurrent constipation. As noted by Fenger (1980), Troels-Lund (1924) postulated that Kierkegaard had suffered from tuberculosis of the lungs, this assumption being based on a dairy entry from August 1835 (I A 75 in Thulstrup, 1968). Kierkegaard’s nephew, H.F. Lund, wrote to his brother on 2 April 1841 (Fenger, 1980): ‘The one who is rather ill is Uncle Søren […] His chest has been affected and he has begun to spit blood again.’

The author E. Rasmussen (1905: 74–81) postulated that Kierkegaard had manifested signs of epilepsy. However, this was rejected by the psychiatrist F. Hallager (1906: 138–40); the psychiatrist Hjalmar Helweg, probably the most prominent Kierkegaard pathographer, concurred with him. Helweg (1933: 79–80, 115) refuted that the following sources could support this assumption:

1. Kierkegaard’s journal entry 9 Feb. 1838 (II A 702 [in Thulstrup, 1968]): ‘When at times there is such a commotion in my head that the skull seems to have [lifted] up. It is as if goblins had hoisted up a mountain a bit and are now having a hilarious ball in there. God forbid!’

2. The often quoted statement [from T.E. Spang, 1846] (see Garff, 2007: 460) that Kierkegaard ‘often when at Gjødvad’s had strong attacks of his suffering so that he would fall to the floor, but he fought the pain with clenched fists and tensed muscles, then took up the broken thread of the conversation again, and often said: ‘Don’t tell anyone; what use is it for people to know what I must bear?’

3. Kierkegaard’s secretary Israel Levin reported [Kierkegaard, 1978: 449; see Garff, 2007: 460] that ‘last night at Gjødvad’s [Kierkegaard] was sitting on the sofa and had been so gay, amusing, and charming, and then he slid off the sofa, and we helped him up, but [soon] exhausted, he [stammered]: ‘Oh, [le-le-lea] leave it – let the maid sweep it up in the morning’, he stammered, but fainted shortly thereafter [‘afmægtig’]. According to P.A. Heiberg (Helweg, 1933: 80), this incident had taken place at least one year before Kierkegaard’s death in 1855.

The psychiatrist Heidi Hansen and her theologian husband, L.B. Hansen (1988; see also Hansen, 1994) argued that, not dissimilar to Dostoyevsky, Kierkegaard had experienced symptoms of temporal lobe epilepsy – in current terminology, Complex Partial Seizure Disorder, manifested as hypergraphia, aura, transcendental experiences and dissociative phenomena. Corroborative of their interpretation, the Hansens placed great emphasis on Kierkegaard’s intense interest in and study of his contemporary countrymen, Magister A.P. Adler and Justinus Kerner (1786–1862).

Garff (2007) took up the issue of whether ‘more specifically’ a diagnosis of temporal lobe epilepsy could be made. In support of this he gave an account of epilepsy as described by Kierkegaard’s family doctor, the eminent Professor Oluf Lundt Bang (1788–1877), in his ‘Handbook of Therapy’
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(1852: 42–7): aura epileptica, followed by convulsions and loss of consciousness, at times involuntary urination, disturbed speech or screaming, lasting from hours to minutes only. The attacks would recur, but ‘very rarely regularly’, ‘most frequently every fourteenth day, or every month, lunatic disease, at times every third or fourth day, rarely daily; now and then several times per day, even one hundred times’. Among other common causes of epilepsy, Bang mentioned: from catching cold, bathing, stimulating drinks, tight-fitting clothing, mental stress, and debauchery, and ‘especially masturbation, narcotic remedies etc.’ ‘The main sign’ was the state of unconsciousness, ‘the absence’, ‘which recurs periodically, even though, what happens often [is] that it lasts for seconds and without being accompanied by the convolution’. Thus, Bang described features of both grand mal and petit mal epileptic seizures (temporal lobe epilepsy as an illness entity was not described until the 1860s by Hughlings Jackson). Among anti-epileptic remedies, Bang recommended: ‘Rad[ix] Valerianæ in large doses, up to five ounces i-iii daily in powder or infusion’.

Garff (2007) found corroboration that Kierkegaard suffered from epilepsy in the fact, as he put it, that on his admission to hospital in 1855 he had ‘a particular medication in his blood’, namely ‘Rad. valerianæ’. Garff (2007: 461) gives further possible evidence of temporal lobe epilepsy from Kierkegaard’s journal entry in 1848 (IX A 128 in Thulstrup, 1968): ‘Reply: Alas, to be transported up into the third Heaven only once in an entire lifetime – and in remembrance of it to retain a thorn that brings it to mind perhaps many times every day!’ (original emphasis).

Gemmer and Messer (1925: 31) questioned whether Kierkegaard might have contracted a sexually transmitted disease. Vetter (1928: 40) pursued this vexed issue further, suggesting that he might have died from the same disease as Nietzsche, who is generally assumed to have died from tertiary syphilis – dementia paralytica. Gemmer and Messer further queried whether Kierkegaard had broken off his engagement to Regine Olsen in 1841 because he feared he had a venereal disease. Helweg (1933: 315) rejected outright that Kierkegaard might have suffered from dementia paralytica. However, the physician Carl Saggau (1958; see Fenger, 1980) reintroduced the syphilis theory, raising the question whether Kierkegaard’s father might have contracted syphilis in his youth and perhaps later infected his family members. Further, he conjectured that Kierkegaard, around 1840, may have been absorbed by medical theories on syphilis – inherited from his father – and its consequences for him.

Over the years several medics have argued for and against the presence of mental pathology per se in Kierkegaard. Heiberg (1895: 34–5) could not decide whether he suffered from states of pathological melancholy. However, he found some support for this in Kierkegaard’s own verdict in 1849 (A 519 in Thulstrup 1968, X(1): 333): ‘the unhappy melancholy [‘Tungsind’], which surely at one point has been a kind of partial insanity’. Viggo Christiansen (1906) rejected any suggestion that Kierkegaard was a pathological individual. As he put it: his ‘peculiarity’ and his melancholy fell within normal limits, whereas H.I. Schou (1924: 42–3) espoused the opinion that Kierkegaard was of manic-depressive constitution.

Helweg in his classic Kierkegaard pathography (1933, see also Brand, 1934; Fenger, 1972, 1980) – but tacitly relegated into oblivion by Garff (2007) – gave a detailed account of Kierkegaard’s family history in support of Schou’s opinion and went on to conclude that:

the nature of the more acute attacks of illness as well as its course and the hereditary predisposition strongly suggest that in Søren Kierkegaard’s case it was a manic-depressive illness which manifested itself as a mixed [manic-depressive] state with a prolonged but somewhat atypical course but otherwise typical regarding its symptoms. (original italics)
Helweg expanded on this to the effect that at times Kierkegaard’s thoughts could border on the paranoid, but not beyond what very often can be observed in manic-depressive illness. Further, he saw some ‘schizoid elements’ in his constitution. However, Ib Ostenfeld (1957, 1978), Helweg’s colleague psychiatrist, saw it as his task to exonerate this ‘outstanding’ man, Kierkegaard, from the assertions by psychiatrists that he was a ‘sick man’. In Ostenfeld’s opinion a normal psychologically understandable thread ran through the whole of Kierkegaard’s life, but he thought that it was Helweg’s lasting achievement to have drawn attention to Kierkegaards’ ingrained manic-depressive predisposition. In a peculiar twist, Ostenfeld finally concluded that Kierkegaard himself was spared this. In other words, as he put it, that there was not even ‘a weak trace’ of this predisposition to be found in him.

**Admission to hospital and death in 1855**

In 1855 Kierkegaard was admitted acutely ill to Frederiks Hospital in Copenhagen on 2 October and he died on 11 November, aged 42. His medical file has been published in part or full and/or analysed by several authors (e.g. Geismar, 1926–28; Heiberg, 1895; Helmig, 1971: 25–8; Helweg, 1933; Jacobsen, 1955; Jørgensen, 1964, 1: 150; Kierkegaard, 1978; Norregaard, 1913; Staubrand, 1989, 2009; Søgaard, 1991, 2007; Thulstrup, 1953–54). Here is a summary of Kierkegaard’s medical record (Kierkegaard, 1978: 28–32):

*Admission entry 2 Oct. 1855: He had suffered from the usual childhood diseases, otherwise been in good health; for a considerable length of time he had suffered from constipation though; ‘lately movements have been daily and natural’. He could not provide any specific reason for his current illness, however ‘he does associate it with drinking cold Seltzer water last summer, with a dark dwelling, together with the exhausting intellectual work that he believes is too taxing for his frail physique’. ‘He considers the sickness fatal’. – ‘For some time he has had a cough, which at the start brought up a creamlike expectorate, later a serous one, clear with yellow clots’. About two weeks before admission, he had slid off a sofa to the floor and had difficulties getting up again; the next day he fell again; no accompanying dizziness, cramps, or loss of consciousness, but ‘a feeling of utter weakness’, ‘he could not raise himself, his legs failed him for a while, yet he did manage to get up again’. The symptoms persisted for some days; ‘at times he would collapse’, and his gait became unsteady followed by ‘formication’ and ‘numbness’, ‘at times shooting pains from the loin down through the legs’. ‘Sensation remained unimpaired’; ‘urination was either obstructed or involuntary’; ‘he became constipated’. ‘The upper extremities unimpaired in every respect; good appetite; mental abilities completely unimpaired’.

4 Oct.: For the last couple of days ‘completely unable to stand on his legs or to get up’; ‘if supported, he can move his legs forwards, but not place his feet properly on the ground; they fall, heels first’. ‘For a short period he can hold himself upright, but not steadily’. ‘If he sits ups, he can neither twist nor turn’, but ‘slumps’ to the left; when lying down he can pull his legs up slightly but not lift them; ‘sensation still unimpaired’, ‘formication’; pain and urination more frequent; no fever. Examination of his chest, vertebral column or ‘in any particular place’ detected nothing abnormal. He ‘has used Inf. valerian’. ‘Paresis’.

5 Oct.: Urine contains a copious amount of ‘phosphates, is clear, cloudy’. Little sleep; ‘cough is fairly frequent’. ‘The expectorate as described’; frequent urination.

6 Oct.: ‘He is even less able than before to support himself on his legs’.

10 Oct.: His strength continues to decrease; ‘cannot stand on his legs or help himself’, but he ‘can sit up’, ‘speaks fairly well’; constipated.

12 Oct.: Urination ‘quite involuntary when coughing or moving’; ‘completely unable to move his left leg and feels weaker’. ‘He continues to insist that his death is near’.

13 Oct.: Coughing continues, expectorates ‘a clear serous liquid with loose yellow clots’; involuntary urination.
14 Oct.: No major change; constipated for 3 days.
15 Oct.: Unable to ‘support himself at all with his arms, because he cannot contract’ his back muscles, but ‘able to move his arms freely’; ‘keeps his left leg in a bent position at hip and knee and tilted over the right leg’, no particular contraction of the muscles.
16 Oct.: Involuntary and very frequent urination.
18 Oct.: No major change; constipated for 3 days.
19 Oct.: Fairly considerable contraction of biceps, semitendinosus, and membranous [flexor] muscles.
21 Oct.: Little change. Constipated.
22 Oct.: Continued involuntary urination. ‘The patient himself thinks that his strength is decreasing more and more’. ‘He seems to collapse more’. Pulse 100.
23 Oct.: ‘Today he is lying with his legs stretched out, slightly turned inwards’. Troubling cough. Expectorate with ‘purulent clots, a few of which are closely mixed with light red blood’.
25 Oct.: ‘Today he is lying with his legs stretched out, slightly turned inwards’. Troubling cough. Expectorate with ‘purulent clots, a few of which are closely mixed with light red blood’.
29 Oct.: Strength ‘seems to decrease more and more’; ‘sits up during the day, but is very slumped’. ‘Mental abilities are still unimpaired’. ‘V oiding must always be brought about by clyisma’; continued involuntary urination.
1 Nov.: Condition largely unchanged; for a couple of days legs been treated with electricity but with little effect.
4 Nov.: Bed sores; considerable effect of electricity on the leg muscles.
6 Nov.: Strength continues to decrease; unable to bring up expectorate; appetite ‘quite good’; electric treatment of his leg muscles are ‘now working well’.
9 Nov.: Strength ‘visibly decreased’ in recent days; ‘he is lying quietly dozing (stuporous), says nothing, and ingests nothing’; ‘his face seems a little distorted, for the left corner of his mouth appears to be pulled slightly upwards’. The pulse was ‘close to 100 the day before yesterday, steady, weak’; ‘urine continues to be passed involuntarily, fairly clear’; ‘faeces are also passed involuntarily’; pulse ‘130 today, less steady, weak’.
10 Nov.: He ‘continues to lie in the same stuporous condition’; pulse ‘130, still fairly steady’ and ‘irregular’. ‘If one raises his arms, they fall back again heavily and are somewhat stiff in the elbows’. ‘He can raise both eyelids’, ‘the distorsion in his face is not markedly pronounced’. ‘His habitus is very collapsed, he breathes rapidly, noiselessly’.
11 Nov.: He is ‘in the same condition’, pulse is ‘slower, his breathing heavy and short’.
11/12 Nov. ‘He continued in the same condition, died last night at 9pm’.

An eyewitness account by the physician H.S. Lund, Kierkegaard’s nephew who had visited him in hospital, should be inserted here; it was in Lund’s letter to Kierkegaard’s close friend, E. Boesen, dated 11 November 1855 (Kirmmse, 1996: 129): ‘[Kierkegaard] therefore became weaker and weaker, and subsequently became less and less aware of things going on around him. He recognized no one, made no replies, and at last fell into a comatose state, in which sad condition he remained the last three days, without regaining consciousness’.

Kierkegaard’s medical record carries the final diagnosis: ‘Paralysis – (tubercul?)/ – Hemiplegia’.
An autopsy was not performed.

Retrospective diagnoses of SK’s last illness – porphyria?

In Heiberg’s (1895) opinion, a medical diagnosis could hardly go any further than to surmise that Kierkegaard had suffered from chronic myelitis of traumatic origin and insidious onset. Helweg (1933) was uncertain whether Kierkegaard had suffered from a compression of the spinal cord as the result of pathology of the vertebral column or myelitis. He commented that the doctors at the hospital had queried whether the condition was a tubercular spondylitis, an assumption that Helweg
found could be supported by some blood in the patient’s expectorate. J.O. Jacobsen (1955), a physician, found the most likely diagnosis to be myelitis, which had started in Kierkegaard’s youth. However, he dismissed a diagnosis of spinal tuberculosis. The neurologist C. Jørgensen (1964) argued that Kierkegaard had succumbed to an abscess of the lung, and saw this as the cause of his prostration and death. He also held the opinion that Kierkegaard had suffered from a chronic tuberculosis of the lungs. O. Helming (1971), a physician, concluded that the diagnosis was spondylitis tuberculosa. The philosopher J. Staubbrand (1989), having sought medical opinions from O.E. Hansen, T.O. Christensen, H. Hertz and K. Weismann, concluded that the illness can be assumed to have been ‘pulmonary tuberculosis which spread to the spinal canal; in other words spondylitis tuberculosa’, causing compression of the spinal cord, and thus ‘fit[ting] the picture of Kierkegaard’s illness’, namely ‘paralysis of the legs and problems with urination’. The co-author of the present paper, a neurosurgeon, argued that Kierkegaard did not suffer from tubercular spondylitis with compression of the spinal cord by a destroyed vertebra (Søgaard, 1991, 2007). He gave credence to the assumption that the paralysis of the legs and intermittent urination problems could well be explained by compression of the spinal cord, but not total compression, for in that case Kierkegaard would not have had any tactile sensation. It was noted that his tactile sensation was intact. It must also be borne in mind, he emphasized, that a compression of the spinal cord caused by direct compression from a destroyed vertebra would have led to a stationary paralysis. This is the salient point in the argumentation, for the record entries clearly indicate that the paralysis took a progressive, ascending course.

In 1859 the French physician Octave Landry (1859) described Acute Ascending Paralysis, naming it ‘Paralysie ascendante aigüe’. Ten years later Carl Lange (1869) published a comprehensive description of this illness.

61-year-old man admitted to Frederiks Hospital in February 1866. For several months before he had sensed weakness in his legs, especially when mounting and descending stairs, and of very rapid onset. Later, the same month, he collapsed from exhaustion in the street. Soon after, he was unable to walk. Initially the weakness was most pronounced in the right leg, but soon this difference evened out. On transfer to Almindeligt Hospital in Copenhagen, in late March 1866, he was still able to walk a little but only with support, but soon after he became paralytic in both legs. – On admission to Frederiks Hospital no paralysis of his arms was being manifested, but such emerged over the next four weeks. There was difficulty speaking and coughing up mucus, of which large quantities collected in his throat. His intellect, higher and tactile senses were still completely unimpaired. His breathing, especially exhaling, became increasingly difficult, with consequent accumulation of large quantities of mucus in bronchia. He died of suffocation (strangulation) on 30 May 1866.

In addition to his own report, Lange (1869) mentioned 12 similar cases in the literature, including Landry’s. Søgaard (2007) argued that the case described above resembles Kierkegaard’s in an important way.

In 1916 the French neurologists Guillain, Barré and Strohl (1916) described two similar cases, and the illness picture became known as Landry, Guillain, Barré’s Disease, or Guillain-Barré’s Disease. Given the course of Kierkegaard’s final illness and seemingly similar cases described in the literature, Søgaard (2007) concluded that Kierkegaard might have suffered from Guillain-Barré’s disease.

This illness is now generally known to be of auto-immune pathology, arising after an acute infectious disease, viral or bacterial. However, given Søgaard’s interpretation, Schioldann (2013) has drawn attention to the fact that the ascending polyneuropathy which can occur in acute intermittent porphyria can mimic or simply be mistaken for Landry-Guillain-Barré’s disease (Courville
and Mason, 1931). Therefore, the question is whether Kierkegaard might have suffered from this rare, hereditary, recurrent, potentially fatal *metabolic* disease, rather than tuberculosis or other bacterial or viral infection.

An overview of porphyria was given in this journal in connection with the analysis of King Christian VII’s insanity (Schioldann, 2013: 232–3) and is quoted here with some minor modifications.

Acute Intermittent Porphyria, including the variegate type with dermatological symptoms, are the most common forms, inherited as autosomal dominant metabolic diseases. Severe abdominal colic, nausea, vomiting, constipation and tachycardia occur very frequently. Porphyrin becomes excreted in excessive amounts in the urine, often resulting in burgundy red or black urine. Motor weakness (pareses or paralyses) is frequent, at times as an ascending spinal paralysis, the so-called Landry’s (or Landry, Guillain, Barré’s) Disease. The disease can ascend to the brain stem, resulting in dysphagia, dysarthria, dysphonia, and when bulbar paralysis ensues, respiratory failure and asphyxia. Epileptic seizures are frequent. Temporary or protracted blindness can occur. A variety of neuropsychiatric disorders can be prominent as vague subjective complaints, resembling functional conversion symptoms or anxiety attacks, insomnia, or erratic behaviour, or metabolic-toxic psychoses with delirium, hallucinations and delusions. Some patients, however, develop psychoses which cross-sectionally are indistinguishable from schizophrenia and manic-depressive disorder. Intriguingly, these illnesses can occur as the only clinical manifestation of Acute Intermittent Porphyria.

Kierkegaard’s anamnesis, physical and mental, does not allow a confident retrospective diagnosis of porphyria to be made, in the absence of occurrence of burgundy red or black urine. It must be emphasized, however, that porphyria can manifest itself without such pathology, as well as without some of the other signs and symptoms of this disease, which has rightly been dubbed *la petite simulatrice*. We argue that the possibility of Kierkegaard having suffered from Acute Ascending Paralysis in its own right could be suggestive of porphyria. We have not found any literature to the effect that valerian can trigger Acute Intermittent Porphyria – if he was actually taking this remedy during the time leading up to his admission to hospital. As mentioned, his medical case record simply states that he ‘has used’ valerian but not when last taken.

This also raises the question whether Kierkegaard was using any other medicament, which might have induced an attack or attacks of Acute Intermittent Porphyria. Certain agents, e.g. alcohol, lead, arsenic, sulphonial, barbiturates and camphor, can induce attacks of this disease. Kierkegaard wrote in his diary in 1836 a laconic, undated entry (Thulstrup, 1968, 1: 133): ‘pulvis temperans campheratus’. It can therefore be assumed that he had been prescribed this remedy. Some corroboration that Kierkegaard actually took camphor is possibly found in his ‘metaphorical’ rejoinder to the author Johan Ludvig Heiberg in 1836, and first published by Garff (2007: 67):

Indeed, I suffer from a ‘cloudburst of ideas’ [Heiberg’s expression] a great deal as long as they remain inside me. If I did not expel them every now and then with a sweat bath [‘Svedekuur’] – this is how I metaphorically describe my activity as a writer – they would undoubtedly attack the nobler inner parts.

Garff saw this ‘Freudian jab below the belt’ as explaining Kierkegaard’s literary activity during these spring months ‘as a sublimation of inner – implicitly sexual – energy which ought to have found a more direct biological discharge’.

One must query therefore whether around this time Kierkegaard had experienced a more direct biological discharge. Heiberg (1912) even suggested that during a period of debauchery in
1836, possibly inebriated, he had been carried away by his emotions and visited a whore. Garff (2007: 104) went into more detail, quoting a journal entry from June 1836: ‘Strange anxiety – every time I woke up in the morning after having had too much to drink, it was finally fulfilled’, and an entry 5 months later, 11 November (IA 271–272 in Thulstrup, 1968): ‘My God, my God’, followed by: ‘The bestial sniggering’ [‘Den dyriske Fnisen’]. Rohde (1959: 36) also cited the latter, expressing the opinion that on the day before Kierkegaard had ‘probably’ visited a brothel and now, in Rohde’s words, panic-stricken he relived the whore’s sniggering, and that this event ‘probably’ was Kierkegaard’s one and only sexual encounter. However, Garff appeared dismissive of the suggestion that Kierkegaard had visited a brothel and possibly contracted syphilis there, or fathered a child.  

Camphor has been in medical use since antiquity. In 1772 Auenbrugger introduced it in the treatment of ‘mania’ (Pearce, 2008). His rationale was to provoke epilepsy, which was believed to have an antimanic effect. It was also used as an aphrodisiac, in small doses, and as an anaphrodisiac, in large doses. In Danish medicine, Møller (1823: 376) listed *pulvis temperans campheratus* as a remedy in sweat cures (3 units [lod] of cooling powder mixed with 40 Gran of pulverized camphor; a teaspoonful to be taken).  

Camphor, together with absinthe and turpentine, is grouped with the terpenes. If Dr Bang did not already know, he would have read about its potentially severe, at times lethal, side-effects in two short reports in the Danish medical journal, *Bibliotek for Læger* (Anon., 1851; Nutzhorn, 1850): states of delirium, nausea, vomiting, convulsions, epilepsy, intermittent loss of consciousness, involuntary frequent bowel movements and frequent urination, rapid pulse and respiration. Also strong abdominal pain and shock-like shooting back pain and similar manifestations in the arms were described. To this profile of camphor’s side-effects must be added its potential to induce attacks of porphyria. However, cases of paralysis in humans have not been found in the literature.  

Even if a retrospective diagnosis of camphor-induced porphyria cannot be established for Kierkegaard, camphor may provide an important piece of the controversial jigsaw puzzle regarding his state of health which has always perplexed Kierkegaard scholars.  

Given the fact that Kierkegaard had taken valerian, Garff (2007) interpreted this to mean that Bang was possibly treating him for epilepsy, based on Bang’s description of epilepsy and its treatment with valerian in his ‘Handbook of Therapy’ (1852).  

However, adding camphor to his treatment regime, Kierkegaard would have been treated with this plus valerian and Seltzer water, although not necessarily concomitantly. Perusing Bang’s manual, it can established that he actually prescribed these drugs in the treatment of spermatorrhoea, also cited in part by Garff. Bang (1852: 409–14) described this condition as seminal emission with erection: ‘spermatorrhoea erotica’ or without emission: ‘spermatorrhoea torpida’, ‘with or without lustful feelings or dreams – nocturnal or diurnal, spontaneously or on exertion [...]’, the touching of women’. Persisting spermatorrhoea would result in ‘mental and physical weakening, more or less spreading to all organs, and finally no longer intermittently, but constantly increasing’, manifested in the form of headache, fatigue, pain in the extremities and hypochondriaca ‘immediately after the emission’. ‘The cause is onanism and too frequent sexual intercourse, especially the former though, and indulged in since earliest childhood’ (emphasis added). Among long-term sequels were: ‘convulsions and paralysis, especially of the legs – tabes dorsalis, early blunting of the mental faculties, memory, and perceptual function, [...] the vision’, ‘epilepsy’ and ‘various insanities’, namely ‘idiotism in adults’. Risk of impotence was also mentioned. Bang did not hesitate to emphasize that some of these individuals due to their ‘sins’ and through reading books about it could become melancholic or commit suicide.  

In ‘erethic’ [i.e. erotic] spermatorrhoea, Bang prescribed antiphlogistic remedies, venesection, *pulvis refrigorens* in the evening and Seltzer water ‘the whole day’, *Infus. Valerianae*, and in the

Bang’s doom-and-gloom description of the final stages of spermatorrhoea – paralysis, tabes dorsalis, insanity, idiotism in adults – reads like a description of tertiary syphilis, and it could only have instilled panic and fear in any individual, including Søren Kierkegaard, and as Bang stated, melancholy or even risk of suicide.

**The syphilis theory revisited: the ‘thorn in the flesh’**

The possibility of Kierkegaard having indulged in masturbation in childhood has been debated by several authors (e.g. Garff, 2007; Geismar, 1926–28; Helweg, 1933). If he was given to such indulgence, it is most likely to have caused him pangs of guilt and sin, and in his adult years also fear of dire consequences. However, weight must also be given to possible illicit sexual activity and, with this, real or imagined contraction of syphilis, all of which was clearly spelt out by Bang (1852), although he did not use this term. It begs the question, however, why this eminent doctor and professor, well-acquainted with the currents in European medicine, did not describe syphilis as such in his ‘Handbook of Therapy’, nor for the matter in any other publication by him, given the fact that a synopsis of some of Ricord’s work on syphilis had been published in the Danish medical literature in 1840 (Anon., 1840).

It is an intriguing co-incidence that Kierkegaard became engaged to Regine Olsen on 10 September 1840 and that the account of Ricord’s works was published in *Ugeskrift for Læger* on 26 September and 3 October 1840 (Anon., 1840). Without any reason given, Kierkegaard returned the engagement ring to her on 11 August 1841, and the final break-up happened 2 months later, on 11 October. The synopsis in the Danish journal was based on Ricord’s ‘Leçons sur les maladies vénériennes’, which had been published in *Gazette des Hôpitaux*, March-August 1840. The editor of *Ugeskrift for Læger* emphasized that it would be of interest to readers as Ricord’s previous work on syphilis had not been described in any detail in Danish medical literature (Schioldann, unpublished manuscript).

Ricord wrote that the principal class of syphilis, possibly caused by ‘small animals’ or a virus, was made up of the venereal diseases, contracted during sexual intercourse. One group was blenorragias. The other was chancres and their sequel, syphilis. Among the latter he distinguished between the primitive cases, which were transmittable either by contagion or inoculation; the secondary cases, which were non-inoculable but transmitted via inheritance, the so-called diathesis syphilitica; and the tertiary cases, which were manifested ‘a long time after the primitive’ cases and not transmittable by inoculation or inheritance. In these cases the complications cited were: osteomyelitis, tissue tubercles and necrosis, and syphilitic tubercles in the brain.

Via Bang, Kierkegaard might have gained knowledge of Ricord’s views. This might also explain why he thought that his last illness would become fatal. Further, it can be argued that he took the apparently drastic step of breaking up with Regine in order to protect her and their possible offspring from being affected with syphilis. However, his medical record does not provide retrospective evidence that he succumbed to dementia paralytica, as his mental faculties remained intact, nor does his ascending paralysis resemble tabes dorsalis.

As mentioned above, Garff (2007: 461) used Kierkegaard’s Corinthian citation (‘Alas, to be transported up into the third Heaven only once in an entire lifetime – and in remembrance of it to retain a thorn that brings it to mind perhaps many times every day!’; original italics) as suggestive of an experience caused by temporal lobe epilepsy. However, it can be argued that Kierkegaard is describing a one and only orgasmic experience with a whore, as Rohde (1959: 36) – without reference to this journal entry – suggested, and consequently falling victim to tormenting feelings of sin.
and guilt, and fear of having inherited or contracted syphilis, and this perhaps becoming his ‘painful [qvalfuld] thorn in the flesh’ (1849 A 3221 in Thulstrup, 1968, 10(1): 214; see also Fenger, 1980; Gemmer and Messer, 1925: 31; Hansen, 1994; Heiberg, 1895; Helweg, 1933; Saggau, 1958).

Postscript
An article in the September 2013 issue of Bibliotek for Læger (205: 314–326) is entitled: ‘Søren Kierkegaard’s illness and death. Did he suffer from Potts paraplegia?’. The authors, Jens Staubrand and Kaare Weismann, argue that Kierkegaard’s symptoms were restricted to paralysed legs. In our opinion, as in previous publications on this vexed subject, the ascending features of Kierkegaard’s disease have been overlooked, his medical record clearly showing an illness progressively involving his abdominal muscles, arms and lower cranial nerves, and that he died in a state of respiratory failure. We cannot confirm that a tuberculous spondylitis would explain his final illness.

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Note
1. It has been argued that his ‘thorn in the flesh’ is a metaphorical expression for (his) melancholy states, but in the diary entry quoted here Kierkegaard wrote: ‘… both an original melancholy and a painful thorn in the flesh [saavel ved et oprindeligt Tungsind og en qvalfuld Æl i i Kiœdet] …’.

References
(All publications with translated titles are in Danish.)
Christiansen V (1906) Kliniske Forelæsninger og Foredrag over Sindssygdom [Clinical Lectures and Talks on Insanity]. Copenhagen: Gyldendal.
Schioldann and Søgaard


