Vitamin A Deficiency in Nineteenth Century Naval Medicine

Ironically, technology, more than medical science, brought about a decline in the nineteenth century in the recorded incidence of night blindness at sea. The faster a ship could accomplish its transoceanic mission – that is, in weeks rather than months – the less uninterrupted time its sailors had to live under shipboard conditions, including on inadequate rations. Progress in marine propulsion thus translated into shortened periods of insufficient vitamin A in sailors’ diets.

Technological advances revolutionized transoceanic travel in the late-1700s and the 1800s. With the introduction of the coal-fired steam engine for marine propulsion, the result of efforts by English, French, and American engineers, motor-propelled ships plied the oceans alongside vessels still reliant on venerable means, wind and sail. The main difference between the two was speed. A steam-powered ship with a paddlewheel – the first motorized means of marine propulsion – could complete a long journey in a fraction of the time that a sailing vessel required. In spring 1838, the British Great Western set a record by crossing from Bristol to New York in fifteen days. To do so required an average cruising speed of 8.2 knots (the equivalent of 9.4 miles per hour) [1]. This was nearly double the speed of that of an average sailing ship.

Commercial shippers were the first to seize the advantages of steam-powered propulsion, and the paddlewheel became the mainstay of civilian fleets by 1850. Navies, meanwhile, had to await further technological progress. Though faster and nimbler than sailing ships, and far less vulnerable to foul weather and turbulent waters, steam-driven paddlewheels had overwhelming drawbacks.

The wheels themselves, being mounted on a ship’s sides, made easy targets for enemy fire. Moreover, the wheels, the boilers, and the coal fuel all occupied a significant portion of a craft’s internal space, encroaching seriously on the room needed for artillery [2]. A steam-propelled paddlewheel armed with only a dozen guns always faced the prospect of confronting a wind-powered sailing ship’s one-hundred and twenty guns. The technological advance that finally put steam power ahead of wind and sails for naval navigation was the screw propeller. Because the optimal placement of the screw propeller was the stern, it freed up the broadsides for artillery. And being mounted below the water line, it made a difficult target for enemy fire.
But until the screw became the primary means of propelling military vessels, navy sailors continued to have to withstand months-long periods at sea and to bear the attendant health hazards. Naval records, not the logs of merchant ships, therefore dominate the history of illnesses that beset sailors on very long voyages. Navy crewmen, far more than merchant mariners, suffered the diseases caused by inadequate, ill-balanced diets. And it was the men on naval ships who challenged the physicians on board, who tried to understand and cure what ailed the sailors. Much of what is known about the nutrition-related diseases that affected sailors in the nineteenth century comes from the journals, diaries, and official records of those navy doctors.

**Night Blindness at Sea**

In late October 1860, the French warship *La Cornélie* set sail from the port of Toulon. East of Marseilles on the Mediterranean coast, Toulon was one of France’s key departure points for building and defending the empire. *La Cornélie*, a sleek, three-masted corvette, was making her maiden voyage on that autumn day. The sun glinted off her fresh paint and polished brass, contrasting with the dull iron of her twenty-two cannons. *La Cornélie* was bound for the South Pacific.

A suitably seasoned crew would guide *La Cornélie*, with topmen Jacques Plée and Louis-Marie Stéphan to tend the sails and rigging, and Jean Denon in charge of the guns. Even seaman Elie Morin, though only twenty-four, had sailed the South Pacific. The health of these four, plus another two hundred and fifty-three officers, supervisors, servants, and seamen, was under the care of Marie-Louis-Eugène Chaussonnet, a physician in the employ of the French Navy.

With a brisk wind filling her sails, *La Cornélie* headed south into deep Mediterranean waters, then west past Gibraltar into the Atlantic Ocean. Sailing southwest, she reached South America, rounded Cape Horn, crossed the Pacific, and arrived within range of New Caledonia, Australia, and New Zealand – all within four untroubled months. Chaussonnet, who had been on the alert for complaints of loose teeth and spontaneous bleeding, noted in his journal with satisfaction that the crew was healthy, with ‘not a single case of scurvy’ on board [3].

By the end of 1862, *La Cornélie* had again traversed the Pacific and arrived at the coast of Chile, from which she sailed north toward Mexico. Suddenly, however, the tone of Chaussonnet’s journal changed. Topman Plée came to the doctor complaining that he could no longer work at night: he could see perfectly well in daylight, but at night he was having difficulty seeing the rigging. And the problem was getting worse each night.

Identifying Plée’s problem as acute night blindness, Chaussonnet followed a course that had been advocated by an American colleague and utilized by many other doctors in that era. He gave the topman five milligrams of strychnine to take by mouth each morning [4]. After four days the patient had no more symptoms.
But a month later Plée returned, this time with worse complaints: now, after twilight, he could see virtually nothing. Again Chaussonnet administered strychnine, and, in addition, he fumigated Plée’s eyes mornings and evenings with ammonia water. One course to which Chaussonnet did not resort, although it had its advocates, was to induce vomiting with emetics. Nor did he use purgatives to cause intestinal evacuation [5]. After a week of the strychnine-and-ammonia regimen, Plée announced that his vision problem was cured and resumed his nighttime duties.

But other sailors began to appear with the same complaint, including Denon, Stéphan, and Morin. Soon the doctor was busy giving strychnine, fumigating the eyes, and applying medicines to the skin. Applied around the eye or to the nape of the neck, vesicatories—such as Spanish fly [cantharis]—caused skin blistering but were deemed beneficial because they caused irritation that would supposedly counter the disease [6]. Some of the afflicted seamen got better but then relapsed. Others simply got worse. Clearly, an epidemic was making its way through the crew of La Cornélie.

Chaussonnet had no previous experience with the disease he confronted, although its symptoms fit perfectly with the night blindness (hemeralopia, see textbox 1–1) described in an 1856 treatise by Jean-Baptiste Fonssagrives, a professor of medicine at Brest [7]:

The nocturnal blindness is at first partial, the patient is enabled to see objects a short time after sunset, and perhaps will be able to see a little by clear moonlight. At this period of the complaint he is capable of seeing distinctly by bright candle-light. The nocturnal sight, however, becomes daily more impaired and imperfect, and after a few days the patient is unable to discriminate the largest objects after sunset or by moonlight; he gropes his way like a blind man, stumbles against any person or thing placed in his footsteps, and finally, after a longer lapse of time, he cannot perceive any object distinctly, by the brightest candle-light.

Plainly, the treatments with strychnine, fumigations, and vesicatories were not working. Many men returned after a few days with relapses or complaints of no relief at all. Chaussonnet therefore decided to change his approach and resort to a radical treatment advocated by a colleague in the army. This course entailed shutting a patient for at least a few days in a cabinet ténébreux, a dark closet [8]. From March to August 1863, forty of La Cornélie’s sailors spent time in the cabinet ténébreux before their vision returned—some, nearly two weeks in total darkness and one, a full month.

Textbox 1–1. Differentiating night blindness and naming it

Night blindness has been recognized in the West since antiquity and identified by many different terms. Aulus Cornelius Celsus, a first-century Roman scholar, called it inbecillitas oculorum (weakness of the eyes) [9]. The seventh-century Byzantine Greek physician Paulus Aegineta referred to it as nyctalopia (night blindness) [10]. In early modern history, the French surgeon Ambroise Paré too called it nyctalopia, while his follower Jacques Guilleaumeau wrote of vespertina caecitudo (evening blindness) [11]. The Dutch clinician Hermann Boerhaave referred to it as visus diurnus (sight by day), while in France, François Boissier de la Croix
This course of action, although ultimately effective, nonetheless frustrated Chaussonnet. It was slow to take effect, and, moreover, he did not know why it worked. Nor could he tell why only certain members of La Cornélie's crew suffered from the disease. He looked for revealing patterns, pondering the common and divergent characteristics of the men who were and were not affected by night blindness. He arrived at a keen observation: none of the ship's fifteen officers, eight supervisors, eleven servants, was affected. What was making these three groups immune, while the general seamen were susceptible? Did an explanation lie in the conditions under which they performed their duties? The topmen, working the sails, spent their time high up in the rigging – that is, in open air and bright sunlight. The gunners, in contrast, worked mostly below deck in the half-light of crowded galleys. The ordinary seamen worked at various duties both above and below deck. Exposure to bright light versus darkness, and to wind versus shelter, seemed to Chaussonnet not to be causal factors. Could the seamen's susceptibility have anything to do with age or where they came from? Most

de Sauvages called it *amblyopia crepuscularis* (lazy eye of the dawn) [12]. In the Arabic-speaking world, meanwhile, medical scholars also applied diverse terms, including *shebkeret*.

By the mid-nineteenth century, nighttime vision problems were recognized as falling into two categories. The first, now termed *retinitis pigmentosa*, is congenital but rare [13]. A hereditary disease, it occurs mostly in families. It begins with moderate symptoms, mainly poor vision in low light and loss of peripheral vision, and worsens over time. Examination of the retina with an ophthalmoscope usually finds changes of pigmentation and narrowing of the blood vessels. No effective treatment for *retinitis pigmentosa* has yet been found.

The other night blindness can be severe in the early stages but is rarely permanent. In the nineteenth century it was often, but not universally, referred to as *hemeralopia* – an irony, since the Greek *hemera* means light. Until the cause was identified, it occurred in epidemics such as the one La Cornélie experienced. Usually acute and often transient, it afflicted several or many subjects living under the same, extreme conditions, such as on shipboard, in an army battalion, or in a prison.

The terminology in use today remains something of a muddle. Both *hemeralopia*, literally meaning difficulty seeing in bright light, and *nyctalopia*, referring to vision problems in low light, are commonly used interchangeably for night blindness. The conditions defined by the two terms are in fact each other's opposites. The mixup goes back to the writings of ancient Greek and Roman scientists and other physicians, and it has never been definitively straightened out. The term more widely used throughout the eighteenth and nineteenth centuries, however, was *hemeralopia*, though nyctalopia can still be heard to mean the same condition [14].
of the affected seamen were between twenty and twenty-five years old and came from the west and northwest of France. As with the men's duties, Chaussonnet again found no link between age and birthplace, and susceptibility. He noted his frustration in his journal: enclosure in complete darkness ‘...is the only treatment to cure night blindness for most of the cases, and without it the disease cannot be cured, but it takes a lot of time: six, eight, ten, fifteen, and twenty days’ [15].

Chaussonnet's discouragement echoed that of an army colleague, who spoke for the medical profession:

Night blindness is a 'strange' disease! There is not a single author who, in addressing this subject, does not make this remark, a sort of formulaic and obliged reference which has become today a stereotype of medical language, repeated incessantly by one or another. Nevertheless, what does it all mean? 'Strange', according to our dictionaries, means bizarre, fantasimagoric, capricious, extravagant; but can nature, in its manifestations, be considered extravagant, capricious, fantasimagoric, or even bizarre? Isn't night blindness a disease and, as such, a manifestation of nature, therefore included in the domain of science, as any other natural phenomenon? How can we say of any disease that it is 'strange'? [16].

Some physicians voiced the skepticism that certain seamen pronounced cured in fact were not. After days or weeks with no work to do and confinement in total obscurity to endure, some of the men merely feigned improved night vision so they could be released from the cabinet ténébreux. In any case, no resolution to the night blindness quandary was found while the epidemic aboard La Cornélie ran its course. The ship returned to Toulon in spring 1864, with her crew in much the same condition as it had been eighteen months before.

Chaussonnet’s observations of the distinctions between men aboard La Cornélie who did and did not report night blindness seem not to have led anywhere, at least not immediately. The puzzle persisted, and other explanations abounded. One that was common among sailors was that sleeping on deck exposed to moonlight and humidity caused night blindness [17]. One physician's counter to this notion was that, were it correct, nearly all seamen would have night blindness because ‘...everyone who has sailed on state vessels knows that it would be almost impossible to prevent the sailors from falling asleep on deck during night duty, given the fact that the necessary labor requires the active participation of only a few of the them, and given that the peaceful state of the sea, the beauty of the sky, and the gentle rocking of the waves are such a strong invitation to sleep’ [18].

Others argued that night blindness was a tropical malady caused by warm weather [19]. As boats left the tropics and entered temperate climates, however, the number of sailors with night blindness did not diminish [20].

Homesickness was another hazard of long voyages held responsible, and hemeralopie nostalgique (homesickness night blindness) was implicated because sailors who were affected sometimes were depressed to the point of wasting away [21].

In the French navy, sailors aboard warships stood watch on deck for six hours – la grande bordée – during which they could be exposed to continuous, direct light from
the sun and bright reflections off the sea, the deck, the sails, plus the metal fittings and objects that were ‘much too highly polished and shining these days’ [22]. Some naval physicians therefore subscribed to the belief that so long an exposure brilliant light was causing night blindness [23]. Too much light, the theory held, led to ‘an exhausted condition of the retina’ [24].

To some physicians – and probably clergymen – night blindness was less a medical than a moral condition, specifically, an affliction of onanists. In 1841, a Dublin physician identified the ‘sinful’ and ‘long continued indulgence of the most morbid sexual propensities, such as the constant usage of artificial means of arousing exhausted passions’ as the cause of night blindness in six patients. Stopping ‘the improper use of the genitals,’ he asserted, could prevent the condition: applying a silver nitrate solution to the glans penis could be a ‘very effectual’ means to avert further cases of night blindness [25]. Ascribing night blindness to masturbation may have seemed reasonable at the time, in light of the belief that healthy young men who, as phrased in Genesis, ‘spilled their seed,’ were poisoning their bodies, impairing their digestion, and generally wasting away [26]. After all, a noted Swiss physician and authority on masturbation had argued that the loss of one ounce of seminal fluid was equivalent to the loss of forty ounces of blood [27]. One doctor’s cure for masturbation-caused night blindness was ‘the discontinuance of the lamentable vice’ combined with a ‘generous diet’ [28].

Whatever its cause, an epidemic of night blindness among crew members could disable a ship and cause a mission to be aborted. The French frigate, L’Andromède, setting out to sail the entire Pacific rim, was forced to halt its mission at the coast of South America because so many crew members reported sick with night blindness. The ship physician’s journal reported:

... three quarters of the crew were afflicted, and I could exempt only the most seriously affected crew members from night duty, thus one would often find on deck men who had trouble getting around on dark nights and others who, when steering, could only see the illuminated compass but could not see the sails. These sailors had so many relapses that I no longer counted on anything but the rain and their return to Brittany to effect a complete cure [29].

Night Blindness Linked to Other Diseases of Malnutrition

In the early nineteenth century, physicians reported that night blindness was associated with increased mortality. In 1819, the British naval surgeon Andrew Simpson described two cases of young sailors with night blindness who died from infections. The first died nineteen days after a bout of severe diarrhea; the second, with respiratory problems [30]. Simpson warned that medical practitioners should pay attention to night blindness, because other diseases combined with the night blindness to produce ‘a fatal termination’.

The association of night blindness and diarrheal disease was also well recognized [31]. The Italian ophthalmologist Antonio Scarpa, for example, considered intestinal
problems to be commonly associated with night blindness [32]. The chief surgeon of the French naval station in the Antilles, M. Barat, noted that the worse cases of night blindness were found in sailors who were suffering with diarrheal disease [33]. The outbreak of night blindness on the Prussian ship Arcona in 1861 was associated with dysentery and chronic diarrhea among the sailors, and many sailors died. Eitner, a naval physician, described xerosis of the cornea, an advanced eye lesion of vitamin A deficiency, in one sailor who died of chronic dysentery [34]. Something missing from the food – vitamin A – likely contributed to the great number of deaths among common seamen from diarrheal disease and other infectious diseases.

Vitamin A deficiency probably played a part, too, in the high death rate on British ships conveying convicted prisoners to Australia – a voyage that could take a sailing ship two hundred days in the eighteenth and early-nineteenth centuries. Of course, many of the involuntary passengers began the journey with their health severely compromised: prison diets usually provided little or no vitamin A. For example, in England and Wales in the mid-nineteenth century, the regulation prison diet consisted of oatmeal gruel, bread, cooked meat, potatoes, soup, molasses, and cocoa [35]. As one naval physician noted:

In the convict ships proceeding to Australia, both scurvy and night blindness have frequently made their appearance, but the latter often escapes notice in consequence of the prisoners being sent down into prison either at or a little after sunset. Aboard the Marquis of Hastings, which conveyed prisoners to Hobart Town in 1841, many cases of scurvy occurred, and there were ten of night-blindness, which presented no other symptoms of scorbutic disease [36].

Vitamin A deficiency probably also contributed to increased shipboard injuries and deaths through trauma resulting from accidents and falls [37].

Records of the vigorous transoceanic maritime activity of the nineteenth century – stimulated by the building and defending of empires, civilian travel, and trading in slaves as well as legitimate merchandise – attest to widespread occurrence of hemeralopia. That, in turn, drew extensive medical attention to the phenomenon. More than one hundred reports of night blindness on ocean voyages were published in the nineteenth century alone. A British naval surgeon in the West Indies station, Sir John Forbes (1787–1861), for example noted that night blindness was common. ‘I have known it to exist in a proportion greater than one in twenty, and have been informed by surgeons and other officers on that station that they have seen it prevailing in ships to a much greater extent’ [38]. As was the case with La Cornélie, the condition was most common in men who put to sea for many consecutive months [39].

For the physical well-being of the crewmen, the vigorous slave trading conducted under several nations’ flags was the most pernicious of all maritime undertakings. Portugal and Spain, for example, had doubled their trading in slaves from West Africa to the New World to 135,000 a year by 1840. Great Britain, before eventually abolishing slavery along with most other European nations, took the high moral ground, and based a small fleet at the West African squadron to intercept illegal slave trading. British ships patrolled the area from Cape Verde in the north to Cape Negro south of
the Equator. Alexander Bryson, a physician and British naval officer in charge of statistical reporting, declared this fleet’s work to be ‘the most disagreeable, arduous, and unhealthy service that falls to the lot of British officers and seamen’ [40].

Bryson described slavers’ methods: ‘To avoid observation slaves were seldom embarked till the dusk of the evening, and this, which seldom occupied more than an hour or two, according to the nature of the bar or surf, having been effected, the vessel immediately made all sail, and endeavored to gain an offing beyond the cruiser, if possible, before daybreak’ [41]. He also described how the men aboard the cruisers flying the Union Jack conducted the job of surveillance. Six men – two at the bow, two amidships – were assigned ‘. . .to scan the horizon with a night glass’, – that is, until night blindness struck.

Bryson noted the effects aboard the British brigantine Griffon in 1851: ‘. . .out of about fifty white men twenty-two were affected, and immediately after the sun went down, they had to be led about on the upper deck, in a helpless state of blindness. There was now just cause for alarm, as the vessel with so many men unfit for night duty, was hardly a match for any of the well-armed slavers so common on the coast at that period’ [42].

The crews of West Africa squadron ships faced yellow fever, diarrhea and dysentery, malaria, scurvy, and more. Bryson noted the squadron’s exceptional annual mortality between 1825 and 1845 from infectious diseases alone: 54.4 deaths per 1,000 men of the mean force employed. This mortality rate was much higher than that found in the other squadrons – South America, 7.7 per 1,000; the Mediterranean, 9.3 per 1,000; Home (England), 9.8 per 1,000; the East Indies, 15.1 per 1,000; and the West Indies, 18.1 per 1,000 [43].

The lurid conditions in the British naval hospital on the island of Fernando Pó off Cameroon acquired a widespread reputation. Sailors made a joke of the situation: the standing orders were, ‘Gang No. 1 to be employed digging graves as usual. Gang No. 2 making coffins until further orders’ [44].

Night blindness sometimes occurred on long voyages during outbreaks of scurvy, leading some physicians to conclude as early as the eighteenth century that, besides the bleeding gums, red blotches on the skin, and loss of teeth, it too was a characteristic of scurvy [45]. In his 1785 treatise, Observations on the diseases incident to seamen, the British naval physician Sir Gilbert Blane cited night blindness as a symptom of scurvy [46]. On the French frigate La Belle-Poule off Madagascar, night blindness affected one hundred and eighty seamen, of whom all but four also had signs of scurvy [47]. Scurvy and night blindness were also closely associated in large outbreaks, for example, among seventeen men on Le Colbert in the Gulf of Mexico in 1864, and among thirty-three men on L’Embuscade and seventy-five men on L’Alceste in the Pacific [48]. The night blindness that attacked many crew members of the British squadron during the Siege of Gibraltar was attributed to scurvy. Many physicians therefore surmised that night blindness was just one point in the constellation of signs and symptoms of scurvy.
Detailed logs from the 1857–1859 global circumnavigation of the Austro-Hungarian frigate *Novara* – a voyage of extraordinarily long duration – shed valuable scientific light on the different causes of night blindness and scurvy [49]. A distinguishing characteristic, observable most clearly over so long a period at sea, is the length of time each condition takes to develop. In brief, scurvy can appear within a span as short as four months at sea. Night blindness, in contrast, may not develop in sailors on the same mission for as long as a full year. The explanation lies in the foods available to the sailors and how their bodies could and could handle the vitamins in those foods (Textbox 1–2).

**Textbox 1–2. Scurvy versus night blindness: a lesson from the Novara**

The voyage of the *Novara* provides a dramatic contrast of the time it takes to develop vitamin A deficiency versus vitamin C deficiency. Vitamin C (ascorbic acid) is essential to many important biological functions (e.g. the formation of the connective tissues’ collagen and the synthesis of neurotransmitters; it is also an antioxidant). The body has no organ that can store vitamin C, however, so with no dietary intake of vitamin C, an adult can develop scurvy within just three or four months.

Vitamin A, in contrast, has a storage organ: the liver. When the dietary intake of vitamin A stops, a healthy adult with a recent history of adequate intake may have as much as a year’s worth of vitamin A in reserve. Experiments conducted in the 1940s with volunteer subjects recruited from among Great Britain’s conscientious objectors showed that concentrations of vitamin A in the blood start to drop in some subjects after about eight months. Moreover, most vitamin A-deprived subjects showed some abnormalities in night vision after several months [50].

In the case of *Novara*’s circumnavigation, cases of scurvy generally appeared during long legs of the voyage and not between the ship’s many stops at ports where sources of vitamin C such as citrus fruits and potatoes were abundantly available. According to the *Novara*’s log, the ship put in at Madeira, Rio de Janeiro, Cape Town, Ceylon, Madras, Singapore, Shanghai, Sydney, Tahiti, Valparaiso, and Gibraltar [51].

Night blindness, however, appeared in the *Novara*’s towards the end of the circumnavigation, when the seamen’s vitamin A levels were presumably at their lowest.

Night blindness and the other diet-related disorders with which it was associated were by no means limited to long ocean voyages. Pellagra, for instance, with its characteristic skin lesions, diarrhea, wasting, as well as neurological and psychiatric disturbances (medical students often refer to pellagra’s ‘3-DS’ – dermatitis, diarrhea, and dementia), was sometimes associated also with night blindness. Seen far more often on land than at sea, pellagra was once common in rural France, Italy, Spain, and the
southern United States. Its usual victims were peasants and sharecroppers whose diet was poor in dairy products and meat. As early as the 1780s, Italian physician Gaetano Strambio observed several peasants with both pellagra and night blindness [52]. Louis Billod, a French psychiatrist, attributed night blindness to the general debility and sun exposure found in patients with pellagra, although he also noted that night blindness could occur by itself [53]. In the 1860s, France’s Théophile Roussel, a physician, legislator, and leading authority on pellagra, also noted an association between that disease and night blindness [54].

Malaria, too, was sometime linked to night blindness. The British naval surgeon Andrew Simpson described a sailor who developed night blindness of nearly two weeks’ duration after recovering from a malaria attack [55]. French and Italian physicians, too, considered their malaria patients to be predisposed to night blindness [56]. Similarly, a Spanish physician observed night blindness to be present in regions where the malaria rate was high [57].

Hookworm infection (helminthiasis), too, was occasionally associated with night blindness [58]. The Italian ophthalmologist Antonio Scarpa considered treatments to expel worms also useful in treating patients with night blindness [59].

**Diagnosis and the Search for a Cause**

Until the development of the ophthalmoscope in 1850, a diagnosis of night blindness was usually made on the basis of a patient’s complaint and on the external appearance of his eyes – specifically of the pupils. Night-blinded eyes have a distinctive feature: the openness of the pupils is inappropriate to the light level of the environment. In healthy eyes, the pupils react to bright light by constricting and to dark or dim conditions by dilating. Eyes affected by hemeralopia lack this responsiveness.

The pupils’ reaction to light is under the control of the retina. A diseased retina can impede the normal action of the pupils, causing the pupils to respond to changes in light levels either sluggishly or not at all. Pupil size, therefore, was a clue but not the basis of a definitive diagnosis in the nineteenth century. Indeed, sailors were sometimes suspected of using this recognized clue as the basis for trickery. The suspicion arose that a would-be patient, seeking relief from his duties, might have instilled belladonna in his eyes, thus emulating one key sign that was associated with night blindness. The name of this drug, which would have been on board for the treatment of intestinal disorders, means beautiful woman for the very fact that it causes enlarged pupils that enhance the eyes’ loveliness.

Whether or not sailors actually resorted to the belladonna ploy, it was made impossible by the widespread use of the ophthalmoscope. This revolutionary instrument enabled a physician to look through the pupil directly into the interior of the eye and at its back – that is, at the retina. By examining a retina with an ophthalmoscope, a
physician can distinguish a case of retinitis pigmentosa from a case of hemeralopia (textbox 1–1) [60].

Before the widespread use of the ophthalmoscope, different methods were developed for diagnosing and grading the severity of night blindness. Great Britain’s Andrew Simpson described a vision test in 1819 in which affected patients were asked at twilight to discern different-sized dots on white paper [61]. In 1865, French naval physician Piriou developed a six-level scale based on ability to see light sources in the sky and the sea (table 1.1) [62]. Jean Boudet disparaged both these methods and, in 1871, developed his own method based on the diameter of the pupil [63]. He pasted eleven black circles on white cardboard, with each circle larger in diameter by one millimeter than the previous; the smallest circle was two millimeters across, and the largest, twelve millimeters. The physician could place this pupillary scale next to the patient’s eyes to gauge the degree of night blindness.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria</th>
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<tbody>
<tr>
<td>1</td>
<td>Impossible to distinguish clearly the moon</td>
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<tr>
<td>2</td>
<td>Possible to distinguish the moon</td>
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<tr>
<td>3</td>
<td>Possible to distinguish the planets and Sirius</td>
</tr>
<tr>
<td>4</td>
<td>Possible to distinguish stars of primary magnitude, lunar light, and the phosphorescent glow of the sea</td>
</tr>
<tr>
<td>5</td>
<td>Possible to distinguish stars of secondary magnitude</td>
</tr>
<tr>
<td>6</td>
<td>Possible to distinguish stars of third magnitude and light of a clear atmosphere</td>
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Textbox 1–3. A clever test for diagnosing a true case of night blindness

In the mid-19th century, the German ophthalmologist Alfred Graefe noted that, in a normal subject, if a glass prism were placed in front of one eye to displace the image, the patient would move both eyes to overcome the prism, fuse the image, and avoid double vision. If a subject with night blindness were situated in some degree of darkness, when a prism was placed in front of one eye, the patient’s eye would not move to fuse the image, presumably because of his inability to see in the dim light [64]. Thus, Graefe advocated the use of this prism test to diagnose true cases of night blindness.

To prevent night blindness, of course, a cause had to be found. With the rate of night blindness so high among sailors, excessive exposure to bright light seemed an obvious culprit. Carl Stellwag von Carion, a Viennese ophthalmologist, declared definitively in 1867, ‘The immediate cause of hemeralopia is over-dazzling of the eyes’ [65]. Another observer noted in 1888, ‘...in the middle of the sea, in the tropics where the sky is always
clear and the night dazzling, [night blindness] strikes caulkers, topmen, and all those whose duty renders them defenseless against its influence’ (see textbox 1–4) [66].

Accordingly, one naval surgeon proposed the use of broad-brimmed straw hats for seamen [67]. Another advised the use of dark blue eyeglasses [68]. By the 1870s, when steam-powered vessels shared the seas with sailing ships, different precautions were advocated: on sailing ships, sailors could find refuge in the sails’ shadow; on steamships, shade could be provided with an awning or tent on deck [69]. One physician proposed that blue curtains be suspended from the awnings when the ships were in the bright sun [70].

A far more economical method of prevention that was tried and declared highly successful involved patching the sailors’ eyes, one at a time. Reporting in 1868 on a hellish entanglement at sea involving a British naval ship, a Spanish privateer, and an intruding pirate vessel, Nottidge MacNamara, Professor at Calcutta Medical College, wrote with considerable medical knowledge as well as practical sense:

In a few days, at least half the crew were [sic] affected with nyctalopia. . . . This circumstance put me on devising some means of curing the people affected with night blindness, and I could think of none better than excluding the rays of the sun from one eye during the day, by placing a handkerchief over it; and I was pleased to find, on the succeeding night, that it completely answered the desired purpose, and that the patients could see perfectly well with the eye which had been covered during the day; so that, in future, each person so affected had one eye for day, and the other for night. It was amusing enough to see [a sailor] guarding, with tender care, his night eye from any of the slightest communication with the sun’s rays, and occasionally changing the bandage, that each eye in turn might take a spell of night duty, it being found that guarding the eye for one day was sufficient to restore the tone of the optic nerve, a torpor of which and of the retina is supposed to be the proximate cause of the disease. I must question whether any purely medical treatment would have had so complete, and above all, so immediate an effect [71].

In the search for a cause, the question posed by La Cornélie’s physician Chaussonnet – Why do only certain groups of men on shipboard suffer from night blindness? – did receive some serious consideration. Pondering on why night blindness tends to attack common seamen and to spare officers, servants, and cabin boys, one physician felt that the lowly sailors’ eyes were naturally predisposed to blinding when these men came topside into the sunshine from their dark quarters below: ‘When eyes accustomed to this half-darkness are struck by the bright daylight on the upper deck, the cleaned metal objects shining in the sunlight, even by the sailors’ white clothing, a blinding sensation occurs’ [72].

Communal living, bad hygiene, and poor ventilation were also implicated as predisposing factors for night blindness among the common seamen [73].

**Something Missing from the Food**

In 1787, when the British First Fleet began transporting convicts to Australia, the weekly government-provided victuals for each crewmember that consisted of salt beef or salt
pork, dried peas, oatmeal, biscuits, cheese (12 ounces), butter (6 ounces), and vinegar [74]. Olive oil, which contains no vitamin A, was substituted for butter because the latter went rancid on long voyages. The cheese was an inexpensive ‘flet’ cheese, made from skim milk and containing virtually no vitamin A. The rations were of course even worse for the convicts: males were allowed one third less than the seamen, and females were allowed two-thirds of what the male convicts’ ration, or about half that of the crew.

Thomas Trotter, another physician of the Royal Navy, wrote in his 1799 *Medicinal nautica: an essay on the diseases of seamen*, that the seamen’s diet should be a ‘branch of medicine of the first importance’. He asserted that, ‘...if one half of the money expended on chests of medicine were laid out in the comforts of diet, much real advantage would be gained’ [75]. Despite Trotter’s insistence, the navy’s regulation diet stayed the same for nearly a century (table 1.2) [76].

Finally, in 1825, the daily meat and beer allowances were increased, but cheese and butter, which were difficult to preserve, were removed [77]. With butter and cheese off the list throughout most, the diet of Britain’s seamen was virtually devoid of vitamin A. Later modifications added chocolate, raisins, and preserved potatoes, but still no butter or cheese.

Great Britain’s Alexander Bryson was in the vanguard of physicians to point to bad food as a cause of night blindness. He attributed hemeralopia, like scurvy, to the lack of fresh meat and vegetables, asserting that night blindness was ‘entirely dependent on an improper or erroneous diet’ [78]. France, at least, approached the matter with some sensitivity to the pleasure that food can give, and innovations in the diet of the French navy included the baking of fresh bread on board ship and the introduction of coffee [79].

In the 1840s, after the development of a technique for desiccating vegetables, a factory was established in Paris to produce dried vegetables. The French navy sought

<table>
<thead>
<tr>
<th>Table 1.2. Rations in the British Navy, 1740–1825¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food</td>
</tr>
<tr>
<td>---------------</td>
</tr>
<tr>
<td>Biscuits, lbs</td>
</tr>
<tr>
<td>Beer, gallons</td>
</tr>
<tr>
<td>Beef, lbs</td>
</tr>
<tr>
<td>Pork, lbs</td>
</tr>
<tr>
<td>Peas, pints</td>
</tr>
<tr>
<td>Oatmeal, pints</td>
</tr>
<tr>
<td>Butter, oz²</td>
</tr>
<tr>
<td>Cheese, oz³</td>
</tr>
</tbody>
</table>

¹ Both butter and cheese were removed from the rations in 1825.
² Olive oil substituted for butter on long voyages.
³ Cheese was ‘flet cheese’, a low-quality skim-milk cheese from Suffolk.
to determine whether the vegetables could be used to increase the variety in the diet. The dried vegetables were tested on a voyage of *Le Caïman* to the Red Sea in 1853 and 1854, and met with some acceptance by the crew [80]. In 1856, discussion began about introducing dried vegetables in the regular rations of the French navy, and dried vegetables were introduced to some extent after 1861 [81].

Nevertheless, the French navy diet of 1877, like its British counterpart, remained grossly deficient in vitamin A (table 1.3) [82], and Spanish sailors fared little better (table 1.4) [83]. The French sailors’ rations consisted mostly of salt-preserved meat and pork, bread or biscuits, dried peas, and dried vegetables. For vegetables, there were desiccated potatoes, broad beans, and cabbage (the last containing trace amounts of vitamin A). Olive oil, rather than butter, served as the basis for cooking. Cheese was offered primarily when the ships were in port.

In contrast to the navy, the English East India Company attempted to provide its ships with foods that were the ‘very best in their kind, and with respect to the quantity allowed much exceed that in any service’ [84]. The company’s shipboard provisions

<table>
<thead>
<tr>
<th>Table 1.3. Rations in the French Navy, 1877</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type of food or drink</strong></td>
</tr>
<tr>
<td>Fresh bread (750 g) or Biscuit (550 g)</td>
</tr>
<tr>
<td>beef conserve (200 g) with</td>
</tr>
<tr>
<td>beans (60 g) or peas (60 g) or</td>
</tr>
<tr>
<td>mixed dried vegetables (potatoes,</td>
</tr>
<tr>
<td>broadbeans, cabbage) (18 g) or bees</td>
</tr>
<tr>
<td>or bacon (225 g) with mixed dried</td>
</tr>
<tr>
<td>vegetables (potatoes, broadbeans,</td>
</tr>
<tr>
<td>cabbage) (18 g) or fresh beef (300 g)</td>
</tr>
<tr>
<td>with green beans</td>
</tr>
<tr>
<td>On Fridays, in lieu of the above: beans</td>
</tr>
<tr>
<td>or sardines in oil (70 g) with beans (60 g)</td>
</tr>
<tr>
<td>When in port: cheese (80 g)</td>
</tr>
<tr>
<td>Dried beans (230 g) or peas (230 g)</td>
</tr>
<tr>
<td>Bacon (80 g)</td>
</tr>
<tr>
<td>Rice (80 g)</td>
</tr>
<tr>
<td>Eau-de-vie (60 ml) or rum (60 ml) or</td>
</tr>
<tr>
<td>tafia [West Indian rum] (60 ml)</td>
</tr>
<tr>
<td>Wine (460 ml)</td>
</tr>
<tr>
<td>Coffee (20 g)</td>
</tr>
<tr>
<td>Brown sugar (25 g)</td>
</tr>
<tr>
<td>Seasonings (chutney, olive oil, fats,</td>
</tr>
<tr>
<td>mustard, pepper, salt, vinegar)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of food or drink</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tomato (60 g) or mixed dried vegetables</td>
</tr>
<tr>
<td>(potatoes, broadbeans, cabbage) (18 g)</td>
</tr>
<tr>
<td>or fresh beef (300 g) with green beans</td>
</tr>
<tr>
<td>On Fridays, in lieu of the above: beans</td>
</tr>
<tr>
<td>or sardines in oil (70 g) with beans (60 g)</td>
</tr>
<tr>
<td>When in port: cheese (80 g)</td>
</tr>
<tr>
<td>Dried beans (230 g) or peas (230 g)</td>
</tr>
<tr>
<td>Bacon (80 g)</td>
</tr>
<tr>
<td>Rice (80 g)</td>
</tr>
<tr>
<td>Eau-de-vie (60 ml) or rum (60 ml) or</td>
</tr>
<tr>
<td>tafia [West Indian rum] (60 ml)</td>
</tr>
<tr>
<td>Wine (460 ml)</td>
</tr>
<tr>
<td>Coffee (20 g)</td>
</tr>
<tr>
<td>Brown sugar (25 g)</td>
</tr>
<tr>
<td>Seasonings (chutney, olive oil, fats,</td>
</tr>
<tr>
<td>mustard, pepper, salt, vinegar)</td>
</tr>
</tbody>
</table>
consisted of ample amounts of salt beef and pork, stockfish, chickpeas, flour, peas, yams, brandy, or arrack – but, again, no substantial sources of vitamin A. Compared with seamen from Europe, the East India Company’s lascars – seamen for hire born on the Indian subcontinent – were reputedly more susceptible to night blindness [85].

Table 1.4. Rations in the Spanish Navy, 1805

<table>
<thead>
<tr>
<th></th>
<th>Sunday</th>
<th>Monday</th>
<th>Tuesday</th>
<th>Wednesday</th>
<th>Thursday</th>
<th>Friday</th>
<th>Saturday</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biscuit, oz</td>
<td>18</td>
<td>18</td>
<td>21</td>
<td>18</td>
<td>18</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Beef, oz</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pork, oz</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>4</td>
<td>4</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Menestras, oz</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>5</td>
<td>5</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Cheese, oz</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Vinegar, quartillo</td>
<td>0</td>
<td>0</td>
<td>0.25</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Oil, oz</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Garlic (head)</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Wine, quartillo</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Salt, quartillo</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Textbox 1–4. Light, the eye, and vitamin A

The pigment rhodopsin, also sometimes called visual purple, is the essential substance for the eye’s adaptations to light and dark conditions. Rhodopsin resides in the retina, specifically in the rod photoreceptors; it is not present in the retina’s other photoreceptors, the cones. The cones and rods have complementary functions: to enable visual perception in light and dark conditions, respectively. Vision relies on the cones during the day. As the day gives way to night, responsibility for seeing passes from the cones to the rods.

The transition – that is, adaptation to darkness – is gradual and can take as long as a half hour to become complete [86]. During the dark adaptation process, the eyes transfer their reliance from the cones to the rods, both of which must be in good working order to enable both day and night vision.

Whereas darkness – partial or total – cannot damage or impede the function of the retina, too much light can, because excessive exposure to bright light bleaches the rhodopsin in the rods. In healthy eyes, however, the bleaching is not permanent. A person going from bright conditions to dark undergoes a period of partial or total blindness, which, ideally, lasts only until the rods have recovered their rhodopsin. Eyes lacking sufficient rhodopsin, however, make this recovery poorly.

The adequacy of rhodopsin is a function of food – specifically, of the amount of vitamin A in a person’s regular diet. The less vitamin A a person ingests, the likelier he is to suffer from night blindness. Butter and cheese are two of the foods
that boost a person's supply of vitamin A. When ships were at sea for long periods in the nineteenth century, the sailors who manned them had little or no butter or cheese in their diets for months on end.

While Great Britain's lowly seamen made do with inadequate and unappetizing rations, the officers enjoyed a substantially different diet. The captain and officers usually had their own cook and stocks of food and wine. In addition, many ships had livestock on board to provide fresh eggs, milk, and cheese. Commodore Anson had his own French cook on board the *Centurion* during his celebrated circumnavigation in the mid-eighteenth century. Aboard the *Prince George*, as she made her way from Spithead to New York, Admiral Robert Digby dined on roast duck, butter, potatoes, carrots and greens, and plum pudding [87]. But no one seems to have drawn any conclusions about who on board was and was not reporting sick with night blindness. The mysterious epidemics of night blindness, disease, and deaths slowly disappeared from naval records towards the end of the nineteenth century. As to their cause, further clues were come from investigations conducted far from the sea.

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