



Rembrandt – Aging and Sickness: A Combined Look by Plastic Surgeons, an Art Researcher and an Internal Medicine Specialist

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Abstract

Rembrandt van Rijn (1606–1669) left behind the largest collection of self-portraits in the history of art. These portraits were painted over a period of 41 years, using a realistic technique. To evaluate Rembrandt's aging process we studied 25 uncontested Rembrandt oil self-portraits by means of objective and descriptive techniques. By measuring brow position changes through the years, we demonstrated that brow descent started in the second half of the third decade and began to level out in the fourth decade. Based on Rembrandt's aging physiognomy, from age 22 to 63, we believe that Rembrandt did not suffer from temporal arteritis, hypothyroidism, rosacea, or rhynophima and that no other facial signs of systemic diseases are evident, contrary to the opinions expressed by other medical professionals. We suggest that Rembrandt suffered from melancholia or mild depression, and propose the possibility of chronic lead poisoning as a theoretical illness that he might have had.

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The renowned Dutch painter Rembrandt van Rijn (1606–1669) left behind the largest collection of self-portraits in the history of art. Rembrandt's heritage was 80 self-portraits, including 40 oil portraits, 30 etchings and 10 sketches, in which he is portrayed from the age of 22 (1628) until shortly before his death in 1669 at the age of 63 [1]. There is agreement regarding the authenticity of only 25 of the 40 oil portraits [1]. This plethora of material and Rembrandt's realistic style allow observers to follow the artist as he aged in a "pictorial autobiography." Rembrandt's self-portrait documentation over a period of 41 years provides a rare and unique look into the aging process of human as well as Rembrandt's self-acceptance of this process, in an era in which the average life expectancy was 44; Rembrandt himself reached the advanced age of 63 [2]. It appears possible to gather clues from his portraits to assess his overall mental and physical condition. Most portraits present his face in the same manner, with each bristle and wrinkle documented; and despite the richness of

his costumes and hats, the old familiar face continuously stares out at us from the center of each drawing. Since little is known about the true medical history of the great artist, certain medical investigators have taken the liberty of diagnosing a myriad of illnesses that are supposedly reflected in these portraits, based on a number of minor physical aberrations seen in them. In addition, other investigators have attempted to explain that some of the stylistic changes seen in Rembrandt's works are related to various physical ailments.

We present here various medical conditions cited in the literature, analyze Rembrandt's self-portraits, and perform a medical and artistic assessment in an attempt to verify these diagnoses. We also investigate the aging process of Rembrandt, based on the widely accepted oil self-portraits. Our study is based on the widely accepted premise that Rembrandt painted a realistic rendering of his subject, so that features seen in one picture could be compared to another. In order to descriptively evaluate the facial aging of Rembrandt, we studied 25 uncontested self-portraits from age 22 to 63. For precise measurements of Rembrandt's physiognomy, we analyzed only eight self-portraits, after excluding all paintings in which the facial features were indistinct, the facial position was unusual, or there was an expressional distortion. The self-portraits were analyzed using the following methods:

- **Objective measurements:** The portraits studied were Nuremberg 1629, Glasgow 1632, Louvre with Bart 1633, London 1640, Karlsruhe 1645, Vienna 1652, Edinburgh 1659, and London 1669. Using graphic software (MB ruler) we measured (in pixels) the interpupillary distance in each of the portraits, and then divided the upper most position of the right brow by the IPD, creating an individual brow index that characterizes each portrait [Figure 1A]. The brow indexes were plotted graphically, demonstrating the natural descent of the brow with aging. We chose to use the IPD, a distance that does not change over the years, as a yardstick to determine positional changes of the brow with aging. This overcomes any problems in slight positional changes of the head, or variations in picture size.

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IPD = interpupillary distance

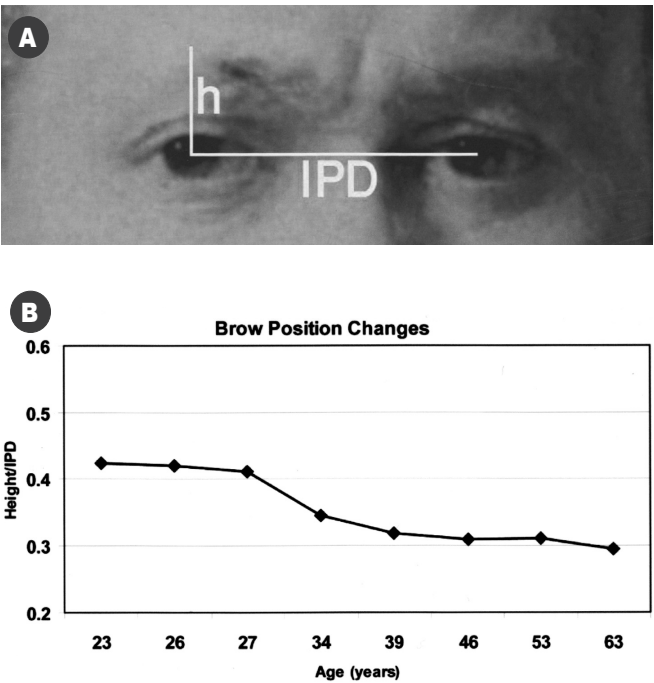


Figure 1. The interpupillary distance divided by the brow height creating an individual Brow Index, which characterizes each portrait [A] and brow indexes plotted graphically, demonstrating the natural descent of the brow with aging [B].

- *Descriptive techniques:* These included a) grading the 25 uncontested portraits of Rembrandt with regard to signs of facial aging: progression of forehead and glabellar wrinkles, dermatochalasis (skin redundancy of the upper eyelids), nasolabial folds, marionette lines (wrinkles lateral to oral commissures), submalar hollows, jowl formation and accumulation of neck fat; and b) reevaluating and discussing the health problems cited in the literature on Rembrandt and proposing our own suggestions, based on a meticulous medical, stylistic and historical evaluation of Rembrandt's self-portraits.

Rembrandt's aging

We present here both an objective and subjective description of the artist's aging physiognomy. The brow position was the only facial structure we could reliably measure. We were able to demonstrate a process of brow descent that started in the second half of his third decade and began to level out in his fourth decade [Table 1] [Figure 1B]. We also noticed in a few consecutive portraits executed when Rembrandt was 52–56 years old that his brows seem very highly positioned, together with exaggerated horizontal forehead wrinkles, or compensatory brow ptosis. These are: New York 1658, Black Hat 1660, Paris Musée du Louvre 1660 [Figure 2], New York, The Metropolitan Museum of Art 1660, Amsterdam, Rijksmuseum 1661, and Cologne, Wallraf-Richartz-Museum 1662. This chronic frontalis muscle contraction elevates the low-positioned brow. This is seen in persons with severe ptotic upper eyelid skin, which

Table 1. Brow descent during aging

Rembrandt's age (yrs)	Brow index
23	0.425
26	0.42
27	0.4117
34	0.345
39	0.318
46	0.31
53	0.312
63	0.295



Figure 2. An example of compensated brow ptosis: Paris, Musée du Louvre, 1660



Figure 3. Nuremberg, Germanisches Nationalmuseum, 1629

causes visual field disturbances. The subjective descriptions are provided below.

- **Nuremberg – Germanisches National Museum 1629, age 23 [Figure 3]**
Upper face: The hair is thick and bulky, the eyebrows are a little high for a male, and the lateral third of the left eyebrow is missing. There is a vertical wrinkle in the right glabellar area, resulting from corrugator muscle activity. The lower lids are smooth with no wrinkles. The cheek is youthful, with a round malar fullness.
Lower face: Smooth skin with no wrinkles. A soft fold is seen in the left commissural area that resembles a marionette line. The bulbous tip of the nose is round and full, with no color alterations. A double chin is slightly prominent.
- **Paris – Musée Du Louvre 1633, age 27**
Upper face: The lateral third of the eyebrow is missing. The eyebrows seem lower and the asymmetric glabellar furrow is more apparent. Extra skin appears on the upper eyelids for the first time (dermatochalasis), which is more obvious on the left. The hypertrophic orbicularis is noticeable in both lower lids.
Lower face: The cheek is no longer a smooth surface, with a single concavity around the zygomatic area, but is portrayed with a light lateral fullness in the nasolabial fold area. Even a small



Figure 4. Karlsruhe, Staatliche Kunsthalle, c.1645



Figure 5. Edinburgh, National Gallery, 1659



Figure 6. Hague, Royal Cabinet of Paintings, 1669



Figure 7. Washington, National Gallery of Art, 1659

jowl now exists lateral to the chin, so that the jaw line is no longer consistent. He has a thin new beard and a mustache. More fat is gathering under the chin.

• **Karlsruhe – Staatliche Kunsthalle 1645, age 39 [Figure 4]**

At the end of his fourth decade, many changes emerge in all parts of Rembrandt's physiognomy. He has gained weight and has no beard or mustache.

Upper face: The hairline is shifted backwards. There is an accentuation of the glabella and forehead wrinkles, and the appearance of a new, horizontal wrinkle in the glabella due to procerus muscle activity. Only the medial part of the eyebrow is still visible, and even that is very sparse. Dermatochalsis is more substantial on the left. Some new wrinkles traverse the lower lid. The youthful fullness in the malar area has disappeared and the cheek is empty (more pronounced on the left cheek due to shadow gradation).

Lower face: The nasolabial fold and the marionette line are clearly seen, accentuated by the inferior-medial malar fat pad ptosis. The jowls are larger and much more obvious. Similarly, the double chin is now very apparent.

• **Vienna – Kunsthistorisches Museum 1652, age 46**

Upper face: The forehead is mostly shadowed, but we can see deep wrinkles, especially in the glabellar area. A central fat bulge is detected on the lower eyelid over the bony orbital margin with emphasis of the shadowed nasojugal groove.

Lower face: The cheek mass continues to sag, emphasizing the nasolabial fold and the submalar hollowing.

• **Edinburgh – National Gallery of Scotland 1659, age 53 [Figure 5]**

Upper face: It seems that the painter has concentrated on the detailed description of every wrinkle. Asymmetric dermatochalsis has continued, more severe on the left. Vessels are observed in the right temple. Lower eyelid skin is wrinkled, an orbital fat pocket seems to bulge, and the nasojugal groove is evident and continues laterally as the palpebromalar groove. Laterally, a new fullness is noticed, which seems like a malar mound under which an infrazygomatic concavity is present.

• **Hague – Royal Cabinet of Painting 1669, age 63 [Figure 6]**

This portrait is accepted as Rembrandt's last portrait. In his last years, Rembrandt mostly documented himself in his simple gray painter's robe. The lonely, defeated and penniless Rembrandt is heavier, white-haired and his skin is pale and gray. His look depicts deep sadness.

Lower face: Looks heavy, possibly edematous.

Rembrandt's medical condition

Temporal arteritis

Espinel [3] raised the possibility of temporal arteritis, a painful inflammation and swelling of the temporal artery at the temporal region, based on the Washington self-portrait [3] [Figure 7]. This interesting finding appears only in two portraits (Washington, Edinburgh) done in 1659. However, Rembrandt did not demonstrate any of the clinical signs or complaints related to this disease [4]. Therefore, we support the assumption of some authors [5,6] that it is highly unlikely that the artist suffered from temporal arteritis and much more probable that he simply illustrated a distended sentinel vein in the temporal region [7].

Rosacea, rhynophima, arcus senilis, xanthelasma, and pinguecula

Espinal [3], analyzing the Washington self-portrait (1659), claimed that the cream-colored lines in his lower eyelid probably represented the existence of xanthelasma. In addition, the left nasal conjunctiva seemed elevated to him, compatible with a pinguecula, and a white arc in the left iris seemed to be arcus senilis [3]. He interpreted the red patches on Rembrandt's cheeks as telangiectasies, pointing to the possibility of existing rosacea, and the bulbous nose as rhynophima. We disagree with these assumptions for several reasons. First, from a young age, Rembrandt's nose tip was bulbous and although it widened over the years, this seems to be his normal aging physiognomy. Moreover, his use of red colors in the Washington portrait does not recur in chronologically adjacent portraits. In particular, it is not present in Edinburgh [Figure 5], which was executed in the same year. Since all the lesions mentioned develop slowly, we would expect to see some evidence in successive portraits.

However, after scrutinizing the subsequent self-portraits, we were unable to substantiate these findings. We believe that there is no evidence to prove any of Espinel's suggestions.

Hypothyroidism

From an early age, the artist consistently depicts himself with short eyebrows. This finding could be of no significance, but short eyebrows are one of the signs of hypothyroidism and several authors have raised the possibility that the painter did indeed suffer from this malady [8]. Marcus and Clarfield [6], however, felt that the artist probably did not have hypothyroidism. Replacement therapy did not exist then, so untreated hypothyroidism would have had serious implications for Rembrandt's health and it is highly unlikely that the artist would have lived as long as he did. Apart from the last year of his life, there are certainly no other obvious facial signs of the disease in his portraits such as puffiness around the eyes, marked loss of hair, or tongue enlargement. However, there is swelling in the lower anterior neck in a number of works that suggests a thyroid goiter, but this is an inconsistent finding (Madrid 1642–43, Vienna 1652, Vienna 1655, Florence 1655).

Clinical depression

Rembrandt's weight shifts and the sadness flowing from his eyes brought Espinel [3] to conclude that Rembrandt probably suffered from clinical depression in his final years. His isolation from society supports this assertion. Espinel uses it to explain the use of darker colors, a claim dismissed by many who are familiar with his work during his career years, in which he consistently used dark and monochromatic colors. This is an important issue because Rembrandt actually experienced many losses throughout his adult life: the loss of his family, his fortune and his professional status. It is well known that the loss of his family did aggravate his seclusion from society in a period when he was poor and had difficulty supporting and sustaining himself. Several studies have raised the possibility of Rembrandt's depression in his final years and claim that the paucity of portraits from that period provides evidence of his increasing inability to function. Although it is true that he painted less at the end of his career, we find no reason to believe that this decrease resulted from a mental condition, since another possible reason could be the decrease in demand for his paintings [1]. We must also remember that Rembrandt was active to his last day. There is a report from Rembrandt's friend, Van Evedungen, who visited him regularly, that he planned to embark on a new series of copper plates before his death [9].

Chronic lead poisoning

It is known that lead powder was used as the basis for all white colors at that time. It was extracted from lead cylinders covered in cattle excrement (lead carbonate and lead acetate). When the flakes had been separated from the remaining metal, they were ground with water between millstones, and the pigment was then moulded into small pieces and exposed to the sun to dry [10]. Artists constituted a group at risk for lead poisoning and there are reports that

some artists apparently completely ignored the already known danger of lead white. For example, there are written recommendations from the 17th century with regard to color handling in situations when the color did not lie smooth on the canvas: "You may lick it all off with your tongue or wipe it off with a moist sponge" [10]. In this way, painters were exposed to lead by inhaling the lead powder, through direct cutaneous penetration, or via the gastrointestinal system. Chronic exposure to lead is potentially damaging to various systems such as the gastrointestinal, blood, renal or central nervous systems [11]. Early symptoms of lead poisoning are usually chronic fatigue, apathy, nervousness, gastrointestinal symptoms, arthralgia, myalgia and neurologic deficiencies such as confusion, memory loss, inability to concentrate and even peripheral neuropathies in the form of distal motor neuropathy. Lead poisoning can cause high blood pressure with or without kidney abnormalities. Anemia can appear prior to all other symptoms due to suppression of bone marrow erythropoiesis. In his final portraits, Rembrandt's skin was pale and probably edematous. It is possible that he suffered from chronic anemia and chronic renal failure, which could explain his weight gain.

Discussion

In this study, we analyzed the aging process of Rembrandt's physiognomy, using objective measurements to demonstrate the brow aging and descriptive aging analysis to track general changes in his facial features in consecutive self-portraits from age 22 to 63. Our objective measurements were based on the interpupillary distance, which is constant in any healthy adult and does not change with age, as a yardstick for brow position comparison. We were able to demonstrate a progression of brow descent in the second half of the third decade, with an apparent stabilization from the age of 40 onwards. We also recognized a few self-portraits done in subsequent years (1658–1662) in which his brow was in a very high position, as if demonstrating a process of compensating brow ptosis. We recognized the temporary nature of this brow elevation, which would be permanent if he truly had a disturbed visual field. The probable explanations we can offer are subsequent visual loss (blindness) of one or both of his eyes, as a result of which there was no further need to eliminate the visual field disturbance. The other possibility is that the elevation was purely voluntary, an active brow movement chosen for expressional reasons. Based on Rembrandt's ability to paint his figure from different angles, as demonstrated in subsequent paintings from later dates, and knowledge of new requests for paintings that Rembrandt received in his last year of life, we suggest that he probably did not lose his eyesight with advancing age. Moreover, he probably had a proper visual field without raising the brows at all, as evident in his last self-portraits (the period 1665–1669).

We have described the aging physiognomy of Rembrandt including the progression of facial wrinkles and soft tissue ptosis, evident from his third decade on. Espinel [3] and Marcus and Clarfield [6] have suggested the possibility of premature aging. It should be emphasized that the quality of life in the 17th century was very different compared with today. Moreover,

Rembrandt lived until the age of 63 at a time when the average life expectancy was 44. Accordingly, although this assumption is interesting, we think that any comparison between the "normal" and "pathological" rates of facial tissue aging is problematic and inaccurate. Additionally, when comparing the detailed pathophysiology of aging known today, one can only be overwhelmed by Rembrandt's spectacular insight into his own aging process.

We have also discussed the possible signs of illnesses in Rembrandt cited in the literature. It should be emphasized that Rembrandt lived until an old age, was productive during the last year of his life, and there was no specific report about any symptom or illness from which he might have suffered [1,12]. We must keep in mind the extensive documentation of Rembrandt's life in the form of letters of complaint he wrote to the city of Amsterdam, and legal and professional correspondence, while there was not a single document regarding any medical problem. Moreover, his friends and students never mentioned any medical problems, physical disabilities or illnesses in their writings about him. Rembrandt probably died at home. In a document confirming his death, the cause of death was "old age," with no designation of any disease.

We have attempted to discuss Rembrandt's medical condition based on historical data, his self-portraits and relevant medical literature. In our opinion, there is no concrete knowledge supporting any significant systemic problem, but we do suggest the possibility of melancholia or mild depression and chronic lead poisoning as open and relevant questions.

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It's a poor sort of memory that only works backwards

Lewis Carroll (1932-1998), British writer and mathematician, most famous for his children's classic *Alice in Wonderland*. It, and the sequel *Through the Looking-Glass* – sophisticated books that combine elements of fantasy, logic and nonsense and have had a lasting appeal for adults as well as children.

Capsule

Folate absorbing tale

Folate is a water-soluble vitamin that plays a critical role in metabolism. Because humans cannot synthesize it biochemically, they must obtain it by ingestion from folate-rich dietary sources. Maternal folate deficiency has been associated with an elevated risk of neural tube defects in the developing embryo, which can lead to malformations of the spine (such as spina bifida), skull and brain. Because of these public health issues, there is considerable interest in understanding the specific molecular mechanisms that the body uses to absorb folate from food. Through a combination of database mining, cell biology and human genetic analysis, Qiu et al. have identified

a transporter protein that appears to be responsible for the intestinal absorption of folate. Previously isolated as heme carrier protein HCP1, the proton-coupled folate transporter (PCFT) was expressed in the small intestine, bound folate with high affinity, and transported folate efficiently into cultured cells at the low pH that characterizes the intestinal milieu. An inactivating mutation in the corresponding gene was identified as the molecular culprit in a family with hereditary folate malabsorption.

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