Authentication of Rembrandt’s Self-Portraits through the Use of Facial Aging Analysis

Tal Friedman MD MHA1, Doron J. Lurie PhD2 and Avshalom Shalom MD MHA1

1Department of Plastic Surgery, Assaf Harofeh Medical Center, Zerifin, affiliated with Sackler Faculty of Medicine, Tel Aviv University, Ramat Aviv, Israel
2Tel Aviv Museum of Art, Tel Aviv, Israel
Dr. Lurie is Curator of 16th–19th Century Art and Chief Conservator at the Tel Aviv Museum of Art

ABSTRACT: The Dutch painter Rembrandt van Rijn (1606–1669) left behind the largest collection of self-portraits in the history of art. Although about 40 of his oil paintings could be considered “self-portraits,” controversy still exists regarding 14 of them. We undertook to determine the identity of the painter or the subject. Our work was based on the generally accepted premise that these portraits represent a “realistic” rendering of the subject. Self-portraits on which there is consensus regarding the authenticity were chosen as the basis for our measurements. Using a computerized technique we measured the brow ptosis. We also subjectively analyzed Rembrandt’s facial aging and the unique asymmetrical elements in his face. We could not add any useful information on 6 of the 14 portraits and suggest that 8 should be considered authentic. Facial aging analysis and the unique surface anatomy allowed us to confirm Rembrandt as the painter in four of six self-portraits. We confirmed Rembrandt as the subject and painter in three more paintings. Of the two paintings in which the subject’s identity was controversial, we determined Rembrandt as the subject in one. We were able to date Rembrandt’s age in two other works and considered another portrait to be a copy. Our methodology may serve as an additional tool for the authentication of self-portraits.

KEY WORDS: Rembrandt, facial aging, authentic self-portraits, analysis, computerized technique

Many painters documented themselves in at least one self-portrait, yet few painted more than ten. Nineteenth and early 20th century art historians were astonished at the vast number of such paintings by Rembrandt. Until the early 20th century, almost 100 oil paintings were considered to be Rembrandt’s self-portraits. In the 20th century, researchers – using pigment analysis, X-rays, examination of the canvas and wood, brush techniques and many more tests – demonstrated that some of these self-portraits could not have come from Rembrandt’s studio. Today only 39 paintings are considered self-portraits [1,2]. Some of the rejected portraits were painted by Rembrandt’s students, some were copies of authentic self-portraits, some had no connection to the master at all, and others were deliberate forgeries.

There is still controversy regarding 14 of the 39 known self-portraits since there is no accurate objective way to prove whether or not a certain painting is truly a self-portrait executed by Rembrandt and that he is the subject portrayed. In our research we used objective and subjective measurements of the pathophysiology of the aging face as another method to determine the authenticity of Rembrandt’s controversial self-portraits. With these methods we helped determine the identity of the painter or the subject as well as date particular paintings.

METHODS

Our work was based on the widely accepted premise that Rembrandt painted a realistic rendering of his subject, especially in the self-portraits, so that proportions seen in one picture are applicable to another [3,4]. To evaluate the facial aging of Rembrandt, we studied 25 uncontested self-portraits from the age of 22 to 63. We eliminated all pictures in which the facial features were indistinct, the facial position was unusual or there was distortion of the features due to smiling or compensating brow ptosis. This left us with eight portraits as the basis for the study.

The portraits were scanned at maximal resolution and the faces enlarged to computer screen size. We looked for a constant variable of the facial physiognomy that might be used as a yardstick to allow us to compare different sizes of pictures. This was the interpupillary distance (the distance between the centers of the pupils).

We encountered various difficulties measuring other facial parts. The first is the illusion of a third dimension due to the painter’s use of light and shadow, which is interchangeable based on the painter’s choice and hence prone to different interpretations. Secondly, anatomic borders between the facial parts such as the hairline, nose tip and lip border change with age and cannot serve as static points of reference. In contrast, the distance between the pupils is a static variable that does not change with age, given no history of orbital diseases.
Facial analysis • Brow index: This was calculated by dividing the distance of the highest point of the right brow from the interpupillary line by the interpupillary distance [Figure 1, left]. Subjective analysis of Rembrandt’s facial aging: analysis of the forehead and glabellar wrinkles, dermatochalasis, nasolabial fold development, submalar hollows, jowl formation and development of upper neck fat [Figure 1, right].

- Unique features of Rembrandt’s face: These include short brows, the asymmetric glabellar furrow going off to the right, the increased dermatochalasis of his left upper eyelid, and the prominent ears [Figure 1, right]. It should be emphasized that the asymmetric wrinkles appear consistently on the same side in the oil paintings, and on the opposite side in the etchings, being a mirror image of the original plate etched by the artist. The vertical glabellar crease, which is tilted to the right in the oil paintings, is tilted to the left in the etchings, a fact that strengthens the credibility of this finding. The oil paintings were based on the mirror image the painter saw in the mirror, which he faced, and the etchings were an opposite print of a plate based on the mirror image. The resulting image, therefore, had facial features that were positioned on the same side as those of the true subject in the etchings and on the opposite side of those in the oil paintings.

Computerized technique
The measurements were computerized using the graphic software MB ruler, which allows for unlimited enlargement of each part of a photo for optimized testing. The values were measured in pixels.

Results
Rembrandt’s Brow Descent [Figure 2]
The authentic self-portraits for measuring facial traits were: Nuremberg 1629 (age 23), Glasgow 1632 (age 26), Louvre (with Bart) 1633 (age 27), London (N.G) 1640 (age 34), Karlsruhe 1645 (age 39), Big Vienna 1655 (age 46), Edinburgh 1659 (age 53), and London (N.G) 1669 (age 63) [5]. By plotting the brow index in these eight authentic self-portraits, we noticed the natural descent of the brow with aging. This began in Rembrandt’s twenties and leveled out in his forties. The controversial portraits were divided into groups, according to the point in question:

The sitter was Rembrandt, but the artist is controversial
Pasadena, Norton Simon Art Foundation c.1638–41 [Figure 3]
The authenticity of this work was recently questioned. This work came from Rembrandt’s studio but is thought to possibly be the work of his student Karel Fabritius and not of the master [6]. Here we looked at Rembrandt’s facial asymmetry. If Rembrandt was the painter, we would see the vertical glabellar fold tilted to the right. If a student painted his master, it would be reversed. Thus we think this might be an authentic self-portrait.

Controversy Regarding Both the Artist and the Subject
London, the Wallace Collection, 1637 [Figure 4]
Although the identity of both the artist and the model are controversial, it has been proved that the work was done in Rembrandt’s
studio, as the panel comes from the same tree as that of the Berlin Self-Portrait, 1634 [7]. We searched for the familiar pathology of Rembrandt’s face and identified the short brows, the vertical glabellar wrinkle tilted to the right, the bulbous tip, and the double chin. The brow index measured also correlated with Rembrandt at the age of 31. The proper asymmetry of Rembrandt’s face, including more severe dermatochalasis of the left upper eyelid and right-sided tilted vertical glabellar wrinkle, might indicate that this is probably a Rembrandt self-portrait.

**DATES ARE CONTROVERSIAL**

Pasadena, Norton Simon Art Foundation, 1641

It is thought that this painting was executed between the years 1638 and 1641 (age 32–35). Our analysis of the brow index and facial physiognomy, and comparison of the facial aging features with chronologically different uncontested portraits, staged it closer to the age of 36 (c. 1642).

**DISCUSSION**

Until the beginning of the 20th century more than 100 oil paintings were thought to be Rembrandt’s self-portraits. Modern research reduced this number by half, categorizing the rejected paintings to four origins [1]

- Rembrandt’s portraits painted by others (mainly his students) in his studio
- 17th century copies of Rembrandt’s self-portraits
- Paintings not painted by Rembrandt in which Rembrandt was also not the sitter
- Deliberate forgeries.

Even today, not all researchers are convinced that all the paintings presumed to be Rembrandt’s self-portraits are in fact his self-portraits. There are several methods (pigment analysis, dendrochronology of the panel, X-rays, infrared, etc.) that can be used to prove that a specific painting is from 17th century Holland and even from Rembrandt’s studio. However, beyond these methods, tests are much more subjective than objective, with parameters like quality, expression, brushstrokes, anatomic understanding, the use of chiaroscuro, etc. In an age where the monetary value of these paintings has reached astronomical sums, it is surprising that almost no one has utilized the analysis of Rembrandt’s facial aging as a criterion to help authenticate these works. Clearly, the application of aging analysis parameters based on anatomic physiopathologic knowledge has been relatively neglected in the discussion of Rembrandt in particular or art works in general.

It should be emphasized that Rembrandt, in contrast to other painters, used his face as a model for different purposes: historical paintings, “trony” portraits (a painting of a character type, rather than strictly a self-portrait), and self-portraits. In doing so, he gained tremendous experience in depicting his own features. When he planned to paint a self-portrait he was obligated to accuracy, and this is the basis for our research.

We understand that, as accurate as this method may be, one must consider the margin of error that arises in the attempt to portray facial features and contour. Nonetheless, our basic assumption maintains that the facial contours in certain paintings are measurable and comparable, mostly due to Rembrandt’s ability to portray details brilliantly, the precise method he used, and his painting technique that was based on timely repetitive work on one portrait, which allowed further judgment and corrections.

The present study was developed by plastic surgeons together with an art historian. By studying Rembrandt’s physiognomy and the aging process in eight agreed-upon self-portraits, we were able to use objective and subjective facial measurements as reference values for Rembrandt’s aging process and facial physiognomy.

Our study of the 14 controversial self-portraits supports the authenticity of 8 of them. Facial aging analysis and the unique surface anatomy allowed us to confirm Rembrandt as the painter in four of six self-portraits. We support the possibility that Rembrandt was both the subject and painter in three more paintings. Of two paintings in which the identity of the subject is controversial, we think that Rembrandt was the subject in one. We were able to determine Rembrandt’s age in two works. We were unable to reach a definitive decision concerning three more paintings, where the controversy related to the dating in one and the painter’s identity in two.

Obviously, our methodology is not the final word but an additional parameter that can be used to authenticate the self-portraits of this truly grand master.

**STUDY LIMITATIONS**

Although the study is based on realistic self-portraits, the portraits – irrespective of their accuracy – are not as precise as pictures. Moreover, our method is limited somewhat due to the light and shadow of the portraits, which might make the surface anatomy difficult to evaluate. Another limitation is the position of the subject in the portraits, which renders some of the portraits unsuitable for evaluation. Nevertheless, our new tools can serve as an important addition to all other known methods.

**Corresponding author:**

Dr. T. Friedman  
Dept. of Plastic Surgery, Assaf Harofeh Medical Center, Zerifin 70300, Israel  
Phone: (972-8) 977-8416  
Fax: (972-8) 977-0427  
Email: dr.tali@gmail.com
References

Capsule

NLRP6 negatively regulates innate immunity and host defense against bacterial pathogens

Members of the intracellular nucleotide-binding and oligomerization domain (NOD)-like receptor (NLR) family contribute to immune responses through activation of nuclear factor-κB (NF-κB), type I interferon and inflammasome signaling. Mice lacking the NLR family member NLRP6 were recently shown to be susceptible to colitis and colorectal tumorigenesis, but the role of NLRP6 in microbial infections and the nature of the inflammatory signaling pathways regulated by NLRP6 remain unclear. Anand et al. show that Nlrp6-deficient mice are highly resistant to infection with the bacterial pathogens *Listeria monocytogenes*, *Salmonella typhimurium* and *Escherichia coli*. Infected Nlrp6-deficient mice had increased numbers of monocytes and neutrophils in circulation, and NLRP6 signaling in both hematopoietic and radioresistant cells contributed to increased susceptibility. Nlrp6 deficiency enhanced activation of mitogen-activated protein kinase (MAPK) and the canonical NF-κB pathway after Toll-like receptor ligation, but not cytosolic NOD1/2 ligation, in vitro. Consequently, infected Nlrp6-deficient cells produced increased levels of NF-κB- and MAPK-dependent cytokines and chemokines. These results reveal NLRP6 as a negative regulator of inflammatory signaling and demonstrate a role for this NLR in impeding clearance of both Gram-positive and negative bacterial pathogens.

*Nature* 2012; 488: 389
Eitan Israeli

Capsule

Endogenous antigen tunes the responsiveness of naive B cells but not T cells

In humans, up to 75% of newly generated B cells and about 30% of mature B cells show some degree of autoreactivity. Yet, how B cells establish and maintain tolerance in the face of autoantigen exposure during and after development is not certain. Studies of model B cell antigen receptor (BCR) transgenic systems have highlighted the critical role of functional unresponsiveness or ‘anergy’. Unlike T cells, evidence suggests that receptor editing and anergy, rather than deletion, account for much of B cell tolerance. However, it remains unclear whether the mature diverse B cell repertoire of mice contains anergic autoreactive B cells, and if so, whether antigen was encountered during or after their development. By taking advantage of a reporter mouse in which BCR signaling rapidly and robustly induces green fluorescent protein expression under the control of the Nur77 regulatory region, antigen-dependent and antigen-independent BCR signaling events in vivo during B cell maturation were visualized. Zikherman et al. show that B cells encounter antigen during development in the spleen, and that this antigen exposure, in turn, tunes the responsiveness of BCR signaling in B cells at least partly by down-modulating expression of surface IgM but not IgD BCRs, and by modifying basal calcium levels. By contrast, no analogous process occurs in naive mature T cells. These data demonstrate not only that autoreactive B cells persist in the mature repertoire, but that functional unresponsiveness or anergy exists in the mature B cell repertoire along a continuum, a fact that has long been suspected, but never yet shown. These results have important implications for understanding how tolerance in T and B cells is differently imposed, and how these processes might go awry in disease.

*Nature* 2012; 488: 160
Eitan Israeli