

Henry VIII's obesity following traumatic brain injury

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To the Editor,

Henry VIII (1491–1547) is considered as one of the most prominent kings of England. Famous for his marriage to six wives and the renunciation of Roman Catholicism to establish the Anglican Church of England, Henry also bestowed patronage to several foundation medical institutes within the United Kingdom, which survive to this day. His modern image is frequently marred by a reputation for cruelty, pomposity and self-indulgence that were most notably manifest in his later years.

Although Henry VIII was celebrated for his health in youth, demonstrating an enthusiasm for equestrianism, hunting and combative sports, his physical condition declined such that he became morbidly obese, suffered from depression and was noted to have bilateral leg ulcers (“sorre legge”) that were possibly due to severe venous hypertension as a result of deep vein thromboses [1].

His health status is identified in several reports that offer evidence of the progressive change in his body habitus (Fig. 1). Although he successfully recovered from smallpox when aged 24 and malaria when aged 30, his body mass and waist size changed dramatically with increased age (Table 1).

Five months before his 45th birthday, Henry suffered from a jousting accident that rendered him unconscious for 2 h. From this incident onward, he is reported to suffer a decline of general health status, increase in body weight and the deterioration of his mood associated with depression. He died approximately 10 years later with a body

habitus of super-obesity (BMI >50 kg/m²) and symptoms of severe immobility and cardiac failure.

Although Henry's dramatic increase in body weight have traditionally been explained by over-eating and gluttony, contemporary sources consistently relate his increased body size with a decline in health and mood to follow his jousting accident. If this timeline of events are genuine, then Henry's health deterioration may be associated with the likely traumatic brain injury (TBI) that resulted in his prolonged episode of unconsciousness.

Traumatic brain injuries can cause several neuroendocrine effects [3] of which growth hormone deficiency (GHD) is amongst the most common hormonal changes [3, 4]. The symptoms of GHD in adults include changes in body composition reflected by increased fat mass with visceral obesity and reduced lean and bone mass, muscle weakness with increased morbidity and mortality and reduced quality of life. Patients with GHD also suffer from a higher prevalence of cardiovascular disease and a deterioration of cardiac function [5, 6].

Henry VIII's final 10 years consisted of an unsuccessful war with France and the death of one wife (Jane Seymour) with the subsequent marriage to three others (Anne of Cleves, Catherine Howard and Catherine Parr), amongst whom one was subsequently beheaded. He increasingly relied on hoists and mechanical aids for mobility as a result of his body mass and decreased muscle strength. His overall decline in quality of life and increased obesity are consistent with the possibility of GHD following TBI in association with the complex socio-behavioural environment of leadership and kingdom.

Increased obesity may have increased the severity of Henry's venous hypertension and leg ulceration. Furthermore, the possibility of Henry's GHD may have limited the healing response to his leg ulcers, as it has recently been

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Fig. 1 Contemporary images of Henry VIII **a** on horseback (1540–1570) and **b** in portrait (1544), demonstrating a progressive deterioration of body habitus. © Trustees of the British Museum



Table 1 Henry VIII body dimensions with increasing age

Data collected from Keynes [2], Chalmers and Chaloner [1]. Figures calculated with a height between 6 ft 2 inches–6 ft 4 inches

Age (years)	Early 20's	23	37	45	50	56
Waist size (in.)	32	35	N/A	37	54	N/A
Chest size (in.)	39	42	45	N/A	57	N/A
Body weight (stone)	15	N/A	N/A	N/A	N/A	28
Body mass index (kg/m ²)	25.6–27.0	N/A	N/A	N/A	N/A	47.7–50.3

postulated that this condition may limit the growth hormone promoting effect on dermal connective tissue which can be recovered by growth hormone replacement therapy [7, 8].

The sequelae of GHD are increasingly recognised as a complication of traumatic brain injury. This pathology may explain the behaviour and actions of Henry VIII following a substantial sporting accident. It may also offer an increased understanding of one of England's most famous monarchs whose performance and deeds were likely influenced by an underlying neuroendocrine condition in his final decade.

References

1. C.R. Chalmers, E.J. Chaloner, 500 years later: Henry VIII, leg ulcers and the course of history. *J. R. Soc. Med.* **102**, 514–517 (2009)
2. M. Keynes, The personality and health of King Henry VIII (1491–1547). *J. Med. Biogr.* **13**, 174–183 (2005)
3. H.J. Schneider, I. Kreitschmann-Andermahr, E. Ghigo, G.K. Stalla, A. Agha, Hypothalamic-pituitary dysfunction following traumatic brain injury and aneurysmal subarachnoid hemorrhage: a systematic review. *JAMA* **298**, 1429–1438 (2007)
4. V. Popovic, GH deficiency as the most common pituitary defect after TBI: clinical implications. *Pituitary* **8**, 239–243 (2005)
5. F.F. Casanueva, A.I. Castro, D. Micic, F. Kelestimir, C. Dieguez, New guidelines for the diagnosis of growth hormone deficiency in adults. *Horm. Res.* **71**(1), 112–115 (2009)
6. E. Ghigo, G. Aimaretti, G. Corneli, Diagnosis of adult GH deficiency. *Growth Horm. IGF Res.* **18**, 1–16 (2008)
7. R. Cimaz, R. Rusconi, E. Fossali, P. Careddu, Unexpected healing of cutaneous ulcers in a short child. *Lancet* **358**, 211–212 (2001)
8. V. Monafò, G.L. Marseglia, M. Maghnie, K.M. Dyne, G. Cetta, Transient beneficial effect of GH replacement therapy and topical GH application on skin ulcers in a boy with prolidase deficiency. *Pediatr. Dermatol.* **17**, 227–230 (2000)