The glands of Owen

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J R Soc Med 2004;**97**:494–495

In May 1834 the Zoological Society of London purchased its first Great Indian Rhinoceros (*Rhinoceros unicornis*). When the animal died on the evening of 19 November 1849, its carcass was offered to Sir Richard Owen (1804–1892), Hunterian Professor and Conservator of the Museum in the Royal College of Surgeons of England (Figure 1). Owen welcomed the 'rare opportunity', and the dissection took place in the winter months of 1849 to 1850 at the Conservator's resident quarters. The animal weighed about 2 tons and by the time Owen's work was completed it was in an 'offensive state of decomposition'. His account of the dissection, published in 1862, includes the rhinoceros's last days as recorded in the Head-Keeper's minute book:[1] it had vomited 'slimy mucus with blood' for a week before succumbing. Owen found that a fractured rib had punctured the animal's lung on the left side and concluded that this was the likely cause of death.

In his detailed description of the anatomy, Owen refers to 'a small compact yellow glandular body attached to the thyroid at the point where the vein emerged'—a structure we now know as the parathyroid gland. The original preparation in which Owen made the observation is still to be seen in the Hunterian Museum at the College. It measures 30 × 14 × 8 cm and consists of part of the larynx and trachea of the rhinoceros, showing the lateral lobe of the thyroid with a parathyroid attached to its upper extremity and partly embedded in its substance (Figure 2).

Although Owen's description is the earliest reference to the existence of the parathyroid glands, it is the Swedish-born Ivar Viktor Sandström (1852–1889) who is credited with discovering and naming the glands. He was a medical student employed as a recorder in the department of anatomy in the University of Uppsala when he came across structures in a dog's neck that were of interest to him. In 1880, he stated:

> 'I encountered on the thyroid of a dog a small hardly hemp-seed sized structure which was included in the same capsule as the thyroid but distinguished itself from it by a brighter colour. A superficial examination revealed an organ of a structure entirely different from that of the thyroid and particularly amply vascularised, because of which I considered it probable that here a vascular gland had been encountered, analogous to the carotid glands'.[2,3]

Figure 1 **Sir Richard Owen, KCB, FRS (1804–1892)** (courtesy of Royal College of Surgeons of England, London)

Having then identified the organ in other animals including cat, rabbit, ox and horse, Sandström proceeded to human anatomy and, to his astonishment, in the first individual examined, 'found on both sides of the inferior border of the thyroid an organ of the size of a small pea which judging from its exterior, did not appear to be a lymph gland, or an accessory thyroid gland and which upon histological examination showed a rather peculiar structure'. He named the new structures *glandulae parathyroidae*.

Sandström was not aware of Owen's description, published in a journal with a limited circulation at the time. His report was not well received and the work remained barely noticed for several years. He suffered from

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Figure 2 **Specimen L333.1, Hunterian Museum, Royal College of Surgeons, London.** This specimen, showing 'parts of the larynx and trachea of a rhinoceros (*R. indicus*), was added to the museum before 1858.' Courtesy of Royal College of Surgeons of England, London

a hereditary mental disorder and took his own life at age 37 years.[4]

The significance of Sandström's discovery remained unappreciated until the experiments of Eugene Gley (1857–1930) helped bring the endocrine function of the parathyroid glands to the fore. Gley, a physiologist and successor to Brown-Séquard at the Collège de France, observed that the tetany and death caused by experimental thyroidectomy in dogs occurred only if the excised material included the glands described by Sandström. This was confirmed by several workers during the first decade of the 20th century, who went on to show that the neuromuscular abnormalities that develop in thyroidectomized animals can be prevented by transplantation of these glands. That the parathyroid glands were central in calcium metabolism, however, was not yet appreciated.[5,6]

In 1891 Friedrich Daniel von Recklinghausen (1833–1910) reported on a patient who had experienced recurrent fractures of several bones with negligible trauma and had subsequently shown 'bending' of the long bones with extensive fibrosis, cysts and brown tumours. This group of findings was subsequently termed 'osteitis fibrosa cystica of von Recklinghausen'.[7] Although an important clinical observation had been made, von Recklinghausen did not correlate the bone disease with any parathyroid abnormality. Most authorities at this time believed that the enlargement of the parathyroid gland noted in patients with bone disease was a compensatory event. It was not until 1915 that Friedrich Schlagenhaufer (1866–1930), professor of pathology in Vienna, suggested that an enlarged parathyroid might be the cause of bone disease and not the result of it. He presented two patients with osteomalacia each of whom was found to have a single parathyroid tumour at necropsy.[8]

Despite this finding it took 10 years before the first attempt at removal of a parathyroid tumour. The surgeon was Felix Mandl (1892–1957), of the University Surgical Clinic in Vienna,[9] and the patient had severe von Recklinghausen's disease. Mandl had at first thought the patient to be hypoparathyroid. When a trial of animal parathyroid yielded little benefit he transplanted four parathyroid glands from a dead accident victim. It was as a last resort that, on 30 July 1925, under local anaesthesia, Mandl removed a 'yellowish-brown almond shaped tumour' measuring $25 \times 15 \times 12$ mm from the left inferior area behind the thyroid gland. There was immediate improvement. Within a few days the blood and urine calcium content was considerably lower and the patient was soon able to walk. He was well for the next six years before developing recurrent hypercalcaemia and a renal calculus. Mandl operated once again but this time could not find any abnormal parathyroid tissue. There was no postoperative improvement and the patient died in February 1936. The recurrence was probably due to aberrant parathyroid tissue, but none was found at necropsy.[10]

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