Original paper

Wax hearts: seeking the antiquity of cardiac pathology

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Wax models of normal and diseased organs were formerly essential medical teaching tools. The ceroplastic heart models from two 19th century pathology museums at the Universities of Florence (n = 8) and Coimbra (n = 10) were analysed. The Florentine collection comprised congenital malformations as well as infectious and inflammatory disorders. The Coimbra waxworks included congenital defects, cardiac hypertrophy and dilation, valvular pathology and cardiac adiposity. This study focuses on heart diseases and teaching resources in European university hospitals during the 19th century. It also highlights the importance of wax models in medical education both then and today, in an era of informatics and digital photography.

Key words: heart, wax models, pathology museums, medical teaching.

Introduction

Wax, from the old English *wæx*, *weax*, of Germanic origin [1], consists of a long-chain fatty acid linked through an ester oxygen to a long-chain alcohol. It is insoluble in water but soluble in organic, non-polar solvents. Natural waxes, such as beeswax and the wax coating on the outer surface of the plant cuticle, are generally mixtures and melt more readily than the pure components [2].

Wax modelling is a procedure that has been used since the dawn of ancient civilizations. Wax could be easily modelled by hand or melted and cast employing the moulage technique [3, 4, 5, 6]. By the end of the 17th century, through the joint work of the Sicilian wax sculptor Gaetano Giulio Zumbo (1656-1701) and the French surgeon Guillaume Desnoues (1650-1735), the first models for medical teaching had been produced. Subsequently, until the beginning of the 20th century, wax models held a prominent position as teaching tools for medical students and professionals [3, 4, 5, 6]. Due to the complex configuration of the heart, three-dimensional (3D) models are of the utmost importance to understand both the morphological and pathophysiological features of this organ [7].

Herein, we present a joint study carried out on the 19th century heart wax models held in two antique and internationally renowned Pathology Museums at the Universities of Florence, Italy, and Coimbra, Portugal. This work aims to draw attention to the cultural treasure epitomised by unique cardio-pathological collections from a historical perspective, potentially catalysing a renovated interest in these models as ancillary didactic devices in medical education.

Material and methods

The Pathology Museum of the University of Florence, established in 1824, is currently housed at Careggi University Hospital, and among its specimens are eight heart wax models (Fig. 1). The Pathology Museum of the University of Coimbra, founded in 1822, is now located at the Anatomical and Molecular Pathology Institute of Coimbra's Faculty of Medicine, and holds ten heart wax models (Fig. 2). All models were morphologically examined and photographed. The morphological lesions were then contextualized according to current medical practice.

Results

A comprehensive inventory of the cardiac waxes from both collections is provided in Tables I and II. Scrutiny yielded a total of 18 cases, of which 5 were congenital cardiopathies, 3 metabolic disorders or degeneration of the heart, 5 infectious diseases of the heart or their complications (Fig. 3A, B), 2 degenerative valve diseases, and 3 hypertrophic or dilated heart conditions (Fig. 4).

Table I. Florence Heart Wax Models

	Origin, modeller	Current nomenclature
1	Florence, Egisto Tortori	Congenital interventricular communication (VSD)
2	Florence, Egisto Tortori	Congenital univentricular heart
3	Florence, Egisto Tortori	Pulmonary valve endocarditis
4	Florence, Giuseppe Ricci	Mitral valve endocarditis
5	Florence, Giuseppe Ricci	Mitral valve perforation (Figs. 3A and 3B)
6	Florence, Giuseppe Ricci	Myocarditis with abscesses (purulent collections)
7	Florence, Egisto Tortori	Tuberculous pericarditis
8	Florence, Egisto Tortori	Fibrinous pericarditis

Fig. 1. The main hall of the Pathology Museum of Florence University

Fig. 2. A view of the Pathology Museum of Coimbra University

Table II. Coimbra Heart Wax Models

	Origin, modeller	Current nomenclature
1	Paris, Vasseur-Tramond	Congenital interventricular communication (VSD)
2	Paris, Vasseur-Tramond	Congenital interventricular communication (VSD)
3	Paris, Vasseur-Tramond	Congenital interventricular communication (VSD)
4	Paris, Vasseur-Tramond	Mitral valve stenosis
5	Paris, Vasseur-Tramond	Mitral valve stenosis
6	Paris, Vasseur-Tramond	Concentric left ventricle myocardial hypertrophy
7	Paris, Vasseur-Tramond	Apical left ventricle aneurysm (Fig. 4)
8	Paris, Vasseur-Tramond	Apical left ventricle aneurysm
9	Paris, Vasseur-Tramond	Cardiac adipositas
10	Paris, Vasseur-Tramond	Cardiac adipositas

Discussion

In the 19th century, medical diagnosis was essentially clinical, lacking state-of-the-art imaging facilities, and therapeutic options were indeed limited. At this time, Florence and Coimbra Universities had high-quality medical schools, which adopted teaching tools such as anatomical and pathological wax models, admired throughout Europe. It was mainly for educational purposes that scientific ceroplastics was developed in the 18th and 19th centuries [6, 8, 9]. A pliable, widely accessible material, wax allowed the creation of realistic and durable models. Until the advent of colour photography, there existed no other medium capable of accurately reproducing diseases, and recording improvement or deterioration of patient conditions.

The Florentine heart wax models were created by Egisto Tortori (1829-1893) and Giuseppe Ricci, while the heart wax models in Coimbra were acquired from the Vasseur-Tramond workshop in Paris. They were accomplished either by direct observation of the diseased heart at autopsy in Florence, or by reproduction of a former wax model in Coimbra [4, 5].

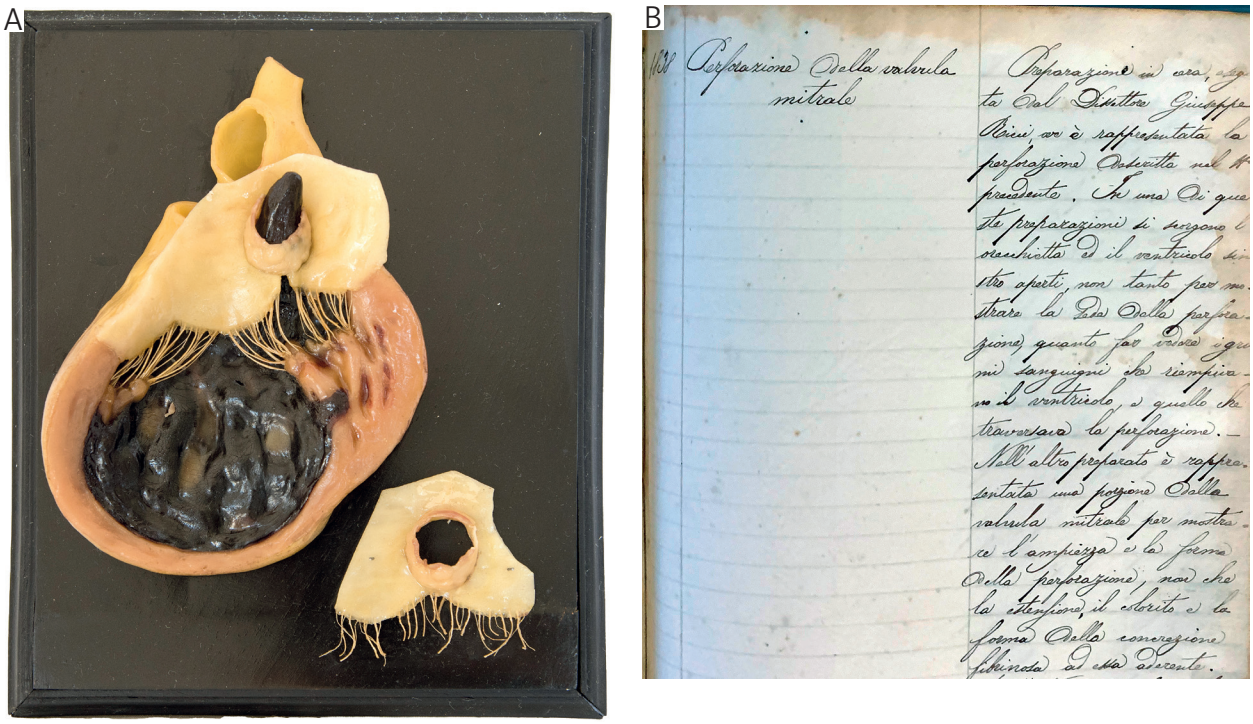

Fig. 3. Heart wax model by Giuseppe Ricci (**A**) and post-mortem report (**B**) of mitral valve perforation complicating infective endocarditis in a 38-year-old woman, who died five weeks after giving birth (source: Pathology Museum of the University of Florence)

Egisto Tortori, pupil of Luigi Calamai (1800-1851), was the last wax modeller from the La Specola workshop, where he started as an apprentice at the age of fifteen. Between 1771 and 1893 collections of both normal and diseased body parts were produced for the La Specola Museum and also for Italian and foreign universities. Tortori's hearts are among the last vestiges of a long tradition of scientific ceroplastics, which flourished in Florence and subsequently spread across Europe. In the mid-19th century, other countries replaced Italy as the major producers of anatomical wax models [4, 5].

The Vasseur-Tramond workshop was founded in Paris by Pierre Vasseur, who was joined by his son-in-law Gustave Tramond (1846-1905) in 1878. As in Florence, artists collaborated closely with anatomists in order to make accurate models that, according to Gustave Tramond, "are not useful for studying anatomy but rather for remembering it once it has been learnt" [4, 5].

The types of cardiac disease and the presence of similar wax models in both series testify to the relevance of these disorders at that time. Indeed, congenital malformations, dilation, infections and their consequences were major causes of morbidity and mortality. Are these wax models still of any use?

Congenital heart malformations were a leading cause of death, particularly during the neonatal period and early infancy [10, 11, 12]. Nowadays, survival has greatly increased due to echocardiography and

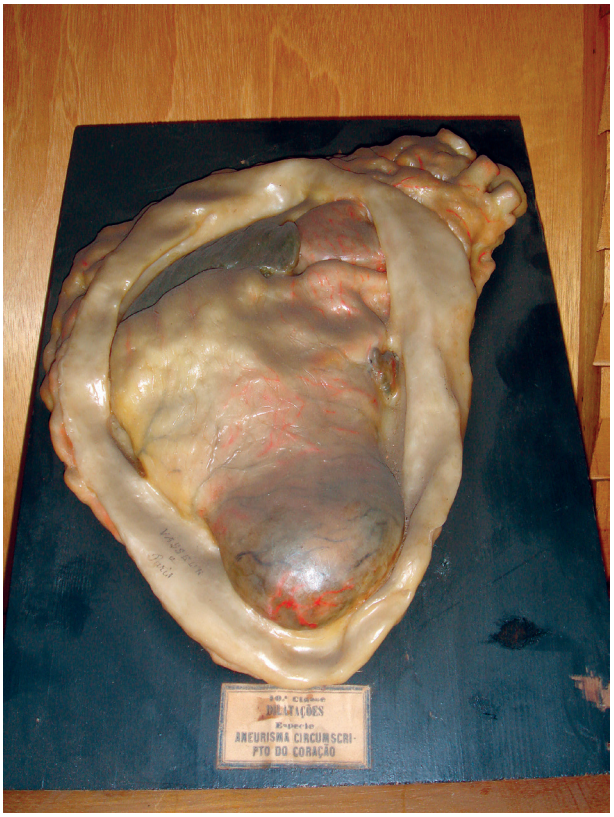

Fig. 4. Heart wax model from the Vasseur-Tramond workshop in Paris, representing apical left ventricular aneurysm (source: Pathology Museum of the University of Coimbra)

surgical/haemodynamic procedures [13]. However, adequate visualization of the malformed heart helps to easily recognize the defect and decide on the best corrective approach. Therefore the use of wax models during medical training is undoubtedly advantageous. Interventricular communication (ventricular septal defect = IVC = VSD) is the most frequent congenital cardiopathy (25-64%) [14, 15]. Survival into adulthood and old age may cause further complications, such as endocarditis, paradoxical embolization, cardiac insufficiency, arrhythmias and sudden death [13, 16, 17, 18, 19]. Univentricular heart, defined as a heart with "only one chamber fulfilling the criteria of a ventricle (the main chamber), with or without a coexisting rudimentary outlet chamber or trabecular pouch", has a poor prognosis [20]. Over the years, the concept of purely morphological malformations has changed to that of functionally univentricular hearts, as has palliative surgery to more definitive corrective procedures [20, 21, 22].

Myocardial hypertrophy (eccentric, concentric, bilateral, right or left) was first described by the French physician Jean-Baptiste de Sènac (1693-1770) [23]. It may be primary or secondary to altered conditions of haemodynamic overload. Gross and microscopic characterization is therefore mandatory in order to establish the causal diagnosis, provide appropriate care and offer familial counselling in genetic-based diseases, such as hypertrophic cardiomyopathy (HCM) [24, 25, 26, 27, 28]. Exercise-induced hypertrophy is currently a crucial issue and investigation is imperative to avoid sudden cardiac death in sportspersons [29, 28, 30].

Sedentarism and change in dietary habits are two of today's social/health "cancers", since they have aggravated and/or introduced a variety of pathological disorders, namely obesity cardiomyopathy [31]. Emphasis has been laid upon the role of visceral fat as a cause of morbidity and in the development/vulnerability of coronary artery plaques [32]. Due to either genetic predisposing factors such as familial hypercholesterolaemia (then unknown) or excessive food consumption, epicardial fat deposition (cardiac adiposity, Quain fatty heart) has been observed and recorded [33, 34].

Endocarditis was another common cause of death, primarily because the principles and rituals of antisepsis both in and out of medical settings were in the early stages of development and mass antibiotic treatment with penicillin was not introduced until 1945 [35, 36, 37, 38]. All the valves could be involved (mitral valve > 25%), at times associated with systemic and/or local complications, such as leaflet rupture/perforation, cord and/or papillary muscle rupture and septic embolism [39, 40]. Endocarditis may be caused by several bacterial or fungal agents [41, 42, 43, 44]. Rheumatic valvular disease was once the usual underlying lesion, but nowadays this has been supplanted by degenerative processes, e.g. aortic valve calcification and mucoid degeneration of the mitral valve. Endocarditis can be isolated or associated with myocarditis, leading to dilated cardiomyopathy in 9-50% of cases and sudden death in 2.7-10%, or else manifest as pancarditis, with a mortality rate as high as 40% [45, 46, 47, 48, 49].

Incidence of tuberculous pericarditis is estimated between 1 and 4%, with a mortality rate of 90% without medical care versus 12% if timely diagnosed and adequately treated [50]. Since the Bacillus Calmette-Guérin (BCG) vaccine, developed in 1906, was only licenced for human use in 1921, and streptomycin was first used shortly after the Second World War in 1946, tuberculous pericarditis was a prevalent life-threatening disease [51, 52]. Nowadays, it occurs less frequently with exuberant clinical symptoms and fatal outcome, except in the context of immunosuppression [53, 54, 55].

Other causes of pericardial disease were progressively identified and linked to the relevant morphological pattern such as fibrinous pericarditis (also known as "bread and butter" pericarditis) in viral infections and autoimmune disorders following myocardial infarction or chronic renal failure (uraemic) [56, 57, 58, 59]. Despite improvements in renal replacement therapy, pericardial involvement may still occur in the setting of previously undiagnosed advanced kidney disease or when patients are ineffectively dialysed [60].

Ventricular aneurysms are usually complications of several diseases, namely cardiac infections, chest trauma or ischaemic myocardial scarring/deformation [61, 62, 63, 64]. This last scenario is of utmost importance, since out-of-hospital cardiac arrest due to ischaemic heart disease accounts for 50-60% of cases [64]. Left ventricle aneurysms in ischaemic hearts occur in 4-20% of cases, 85% of which are anterior, with apical and/or septal involvement [65, 66, 67, 68]. Associated mortality may result from thromboembolism, heart failure, rupture or arrhythmias [67].

This museological survey highlights the relevance of pathology museums and their impressive collections, which offer a unique glimpse into the past of cardiovascular pathology and a documentation of diseases currently ranking first in overall mortality rates in the Western world.

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