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The autopsy of King George II following his sudden death in 1760: Historical Perspectives on Aortic Dissection

La autopsia del Rey Jorge II tras su repentina muerte en 1760: perspectivas históricas sobre la disección aórtica

A autópsia do Rei George II após sua morte repentina em 1760: perspectivas históricas da dissecção aórtica

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Summary: Two hundred sixty years have passed since Frank Nicholls' history-making observations and publication generated by the autopsy findings on King George II following his sudden death in 1760. Several decades later, the disease was named, attaching – for the first time – the terms *dissection* and *dissecting* to an aortic disease process. Another century went by before effective surgical treatment was developed. In sharp contrast, the evolution during the last 20+ years has been fast-paced and amazing. Our understanding of AD, while not yet complete, has improved dramatically. The introduction of nonsurgical endovascular therapy has had a profoundly transformative impact. Stent-graft repair has already largely replaced open surgery in the treatment of complicated type B AD. Predictably, such endovascular techniques will also have major future impact for patients presenting with acute type A dissection. Finally, and amidst the plethora of "good news," it is appropriate to reflect on the formidable challenge that endovascular therapies face as they gear to compete with the much-improved optimal medical therapy in the management of patients with acute uncomplicated type B dissection. Long-term gains may well become the winning card when and if the late results of TEVAR can be shown to improve on the rather compromised outlook of medically treated dissection patients.

Resumen: Han pasado doscientos sesenta años desde las observaciones históricas de Frank Nicholls y la publicación generada por los hallazgos de la autopsia del rey Jorge II después de su muerte repentina en 1760. Varias décadas después, la enfermedad recibió un nombre, uniendo, por primera vez, los términos disección y disección a un proceso patológico aórtico. Pasó otro siglo antes de que se desarrollara un tratamiento quirúrgico efectivo. En marcado contraste, la evolución

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durante los últimos 20 años ha sido rápida y asombrosa. Nuestra comprensión de la DA, aunque aún no está completa, ha mejorado drásticamente. La introducción de la terapia endovascular no quirúrgica ha tenido un impacto profundamente transformador. La reparación con endoprótesis ya ha reemplazado en gran medida a la cirugía abierta en el tratamiento de la DA de tipo B complicada. Previsiblemente, estas técnicas endovasculares también tendrán un gran impacto futuro para los pacientes que presentan disección aguda de tipo A. Por último, y en medio de la plétora de "buenas noticias", es apropiado reflexionar sobre el formidable desafío que enfrentan las terapias endovasculares a medida que se preparan para competir con la terapia médica óptima, muy mejorada, en el tratamiento de pacientes con disección aguda no complicada de tipo B. Las ganancias a largo plazo pueden convertirse en la carta ganadora cuando y si se puede demostrar que los resultados tardíos de TEVAR mejoran las perspectivas bastante comprometidas de los pacientes con disección tratados médicamente.

Resumo: Duzentos e sessenta anos se passaram desde as observações históricas de Frank Nicholls e a publicação gerada pelas descobertas da autópsia do Rei George II após sua morte repentina em 1760. Várias décadas depois, a doença foi nomeada, anexando - pela primeira vez - os termos dissecção e dissecação a um processo de doença aórtica. Mais um século se passou antes que um tratamento cirúrgico eficaz fosse desenvolvido. Em nítido contraste, a evolução durante os últimos 20 anos foi rápida e surpreendente. Nossa compreensão da DA, embora ainda não completa, melhorou drasticamente. A introdução da terapia endovascular não cirúrgica teve um impacto profundamente transformador. O reparo de enxerto de stent já substituiu amplamente a cirurgia aberta no tratamento da DA tipo B complicada. Previsivelmente, essas técnicas endovasculares também terão grande impacto futuro para pacientes que apresentam dissecção aguda tipo A. Finalmente, e em meio à infinidade de "boas notícias", é apropriado refletir sobre o desafio formidável que as terapias endovasculares enfrentam à medida que se preparam para competir com a terapia médica ótima muito melhorada no tratamento de pacientes com dissecção aguda não complicada do tipo B. Os ganhos a longo prazo podem muito bem se tornar a carta vencedora quando e se os resultados tardios do TEVAR puderem ser mostrados para melhorar a perspectiva bastante comprometida dos pacientes com dissecção tratados clinicamente.

It was in mid-2009 that, serendipitously, I came across a most interesting historical occurrence related to the death and subsequent autopsy of England's King George II. This led in turn to the writing of an editorial piece published in Vascular Disease Management (VDM) in November of 2011. The topic and its continuing intrigue have remained foremost in my mind ever since…

George II (George Augustus) was King of Great Britain and Ireland, Duke of Brunswick-Lüneburg (Hanover) and a prince-elector of the Holy Roman Empire from June 11, 1727, until his death in 1760 (at age 76). George was born in the city of Hanover in Germany. His parents were George Louis, Hereditary Prince of Brunswick-Lüneburg (later King George I of Great Britain), and Sophia Dorothea of Celle. Their marriage was dissolved in 1694 on the pretext that Sophia had abandoned her husband. She was henceforth confined to Ahlden House and denied access to her children, who probably never saw their mother again. George spoke only French until the age of four, after which he was taught German by one of his tutors. He additionally learned English and Italian, and studied military history and battle tactics with special diligence. His father George I died on June 22, 1727 during one of his visits to Hanover, and George II succeeded him as King and Elector at the age of 43. George II was crowned at Westminster Abbey on October 11, 1727. He reigned for 33 years, 4 months, and 15 days. And was succeeded by his grandson George III (since his son Frederick preceded him in death).

"On the morning of October 25, 1760 the King arose from bed, had a cup of hot chocolate, and went alone to his privy for a bowel movement. His valet heard a loud crash and found him unresponsive on the floor, his only sign of trauma being a cut on his face presumably related to the fall. The house surgeon, Mr. Andrews, was immediately brought in but was unable to revive the King despite multiple attempts at bloodletting. As it was apparent that the King had died, an autopsy was ordered to rule out any nefarious cause of death. Dr. Nicholls was directed to perform the autopsy and embalm the royal body the following day." (Frank Nicholls was an English physician, born in 1699. He attended Exeter College at Oxford, obtaining his Bachelor of Arts degree in classics and physics at the age of 19. He earned his master's degree at 21 years, a Bachelor of Medicine at 25, and his Doctorate at 30 years. Prior to graduating in Medicine, he lectured at Oxford in anatomy, focusing mostly on what was then referred to as 'minute anatomy'. His academic career unfolded with distinction, and in 1753, he was appointed as Physician to King George II).

> During the autopsy, the abdomen was opened first, with no major abnormalities noted… then the skull. Next, upon opening the chest, Dr. Nicholls encountered a hemopericardium, the blood coagulated, "… nearly sufficient to fill a pint cup (568 ml)". Upon removing this blood, *a round orifice appeared in the middle of the upper side of the right ventricle of the heart, large enough to admit the extremity of the little finger*." Dr. Nicholls surmised that the proximate cause of death was cardiac tamponade and that the King "… must, therefore, have dropped down, and died instantaneously." After opening the (ascending) aorta, "*an approximately 1.5 inch transverse fissure on its inner side was identified through which some blood had recently passed under its external coat and formed an elevated ecchymosis"*. These observations along with a low-toned engraving illustration of such findings were

published the next year (Nicholls L. Observations concerning the body of his late majesty. *Philos Trans 1761;52:265-274.*).

Until relatively recently, it was assumed and widely accepted that Nicholls' excellent account of the intimal tear in the ascending aorta, allowing blood to enter the wall, creating an "elevated ecchymosis" under the adventitia, represented indeed the earliest recording and most clear description of the pathological entity we now recognize as aortic dissection (AD) – a term not yet available to Nicholls at the time as we shall see a bit later. It was further assumed the said dissection had caused the fatal aortic rupture into the pericardium causing the death of George II. This is intriguing when one considers that the autopsy findings do not support such contention as the aortic wall was not obviously ruptured despite the intimal tear and mural bulging. It seems, almost shockingly, that the description of "…a round orifice in the middle of the upper side of the right ventricle…" was somehow overlooked, escaping closer scrutiny at the time of explaining the presence of the large hemopericardium (that obviously caused the fatal cardiac tamponade). Recent re-examination of these issues has led to the development of these thoughtful discussion points that favor an alternative explanation for the causation of the hemopericardium and, consequently, of the King's death:

- The aorta was found to be indeed dissected but not ruptured;
- There was a hole in the anterior wall of the right ventricle which likely represented myocardial rupture from severe ischemic disease;
- It was appreciated in retrospect that the King was most probably suffering from cardiac symptoms for some time ("frequent distresses and sinkings about the region of the heart"). Unfortunately, there was no way for Dr. Nicholls to infer the possible etiology as angina would not be described until 1768 (by Heberden) and not published until 1772. The relationship of angina to coronary artery disease would have to wait another 140 years.
- Dr. Nicholls' attempt at explaining ventricular rupture (from aortic dilatation causing compression of the pulmonary artery…) was articulated in his autopsy report, but it was without substance when considered in light of modern knowledge and understanding of cardio-pulmonary physiology.

- *Postinfarction myocardial rupture* is today a well-known albeit rare consequence of massive coronary ischemia.

Acute AD is the most frequent and potentially catastrophic manifestation of the so-called acute aortic syndrome which also includes intramural hematoma (IMH) and penetrating aortic ulcer (PAU). The incidence is said to be no less than 30 cases per million individuals per year (in the western world). In its natural evolution, without treatment, acute type A AD carries an enormous mortality risk of about 1% per hour during the first 2 weeks after onset, with half of the patients expected to be dead by the 3rd day, and almost 80% by the end of the 2nd week. Death

rates are lower but still significant in acute type B AD: 10% minimum at 30 days, and 70% or more in the highest-risk groups.

The diagnosis of AD and the characterization of its type and precise extent have been refined to an exquisite degree by virtue of computed tomography (CT), especially when performed with intravenous contrast administration and 3-dimensional reconstruction (CT angiography or CTA for short) that can clearly depict the entire aorta and its branches. Magnetic resonance imaging (MRI) is quite useful as well and expected to become fully competitive or even better than CT in the future.

With few exceptions, the management of acute type A AD continues to be a prime example of life-saving emergent open-heart surgery. The operation often involves graft replacement of the dissected ascending aorta, with or without aortic valve repair or replacement. In patients presenting with extensive type A dissection, cardiac surgeons have more recently been considering more extensive operations extending into the arch.

Etiology and Pathogenesis of Aortic Dissection

The aorta is a rather complex organ with a 3-layered anatomic configuration. The intima is a metabolically intensive, monolayered endothelial liner that is supported by a fairly loose connective tissue sublayer which permits the motion of the intima relative to the media when the aorta expands and contracts during the cardiac cycle. The media is composed of some 50 layers of fenestrated, lamellar elastic fibers. Collagenous fibers and smooth-muscle cells are interposed. Elastin is highly stretchable. This enables its fibers to lengthen 2 to 3 times without rupturing, permitting the aorta to exhibit its impressive distensibility and elasticity. Both characteristics are essential to optimal aortic function. Quite opposite are the collagenous fibers, which have an estimated stiffness 5,000 times greater than that of elastin. Their role is to support aortic integrity and resist shearing forces as flowing blood is pumped powerfully by the left ventricle. Outermost is the adventitia, a tough layer of collagen and connective tissue that contributes substantially to aortic integrity. The vasa vasorum within this adventitial layer provide nutritional circulation to a thick vascular wall that cannot rely solely on the diffusion of nutrients from the flowing blood in the lumen.

It is widely accepted that AD occurs when an intimomedial tear, or *entry tear*, allows blood flow to enter the aortic wall, thereby creating a new secondary channel: the false lumen (FL). The FL propagates distally in a spiraled (most often) or straight manner. The FL can also propagate proximally all the way to the aortic valve. Not infrequently, the true lumen (TL) becomes compressed by the pressurized FL, sometimes to the point of collapse that can lead to ischemic complications below (malperfusion). Whereas the proximal thoracic aorta is almost always the site of the entry tear, secondary or reentry tears (fenestrations) can occur either distally in the thoracic

aorta or in the abdominal aorta or iliac arteries. Why and how all this occurs is somewhat mysterious and incompletely understood. However, a diseased or weakened vessel wall is a probable prerequisite, rendering the aorta vulnerable when exposed to the tremendous burden of severe or uncontrolled hypertension. The well-documented increased risk of AD in several inherited aortic diseases (such as Marfan syndrome) supports this assumption. Data from the International Registry of Acute Aortic Dissection (IRAD) have identified several well-defined risk factors for the development of acute AD: male sex, age in the 60s and 70s, hypertension, prior cardiac surgery (particularly aortic valve repair), bicuspid aortic valve, and a history of Marfan syndrome. Less than 10% of the time, acute AD occurs in individuals younger than 40: they are often normotensive, but they typically have a history of cardiac surgery or a bicuspid aortic valve, Marfan syndrome, Ehlers-Danlos syndrome, or similar conditions. Or are frequent users of crack cocaine. Probably belonging in the same disease spectrum are IMH and PAU, which often present with similar symptoms. They may be linked through a common pathogenesis. IMH originates from a hemorrhage within the wall of the aorta, but without a demonstrable intimomedial tear or flap. Many experts think of it as a precursor of AD. In fact, IMH evolves into full AD (with a double-barrel aorta) in nearly 20% of cases. Two thirds of IMH cases involve the descending aorta (rather than the ascending segment. The converse is true in AD. Of note, the overall prognosis and 25% risk of death at 1 year are about the same for both IMH and AD. PAU can form anywhere along the aorta; however, most develop in the descending thoracic portion. Patients tend to be elderly and show evidence of significant atherosclerosis throughout. The ulcer can precede AD and be associated with IMH. The concomitant occurrence of PAU and IMH is dangerous and may warrant early intervention and repair. PAUs behave unpredictably and can lead to rupture and catastrophic hemorrhage. Thoracic endovascular aortic repair (TEVAR) is rapidly emerging as a feasible less-invasive treatment option because most ulcers develop in areas that are anatomically suitable for endovascular repair and endografting. It is increasingly agreed that intervention is justified and should be pursued (if reasonable and feasible) for ulcers larger than 3 cm in diameter, as well as for all symptomatic aortic ulcers of any size.

Anatomic Classification

The extent of the dissection process along the aorta defines the type. The DeBakey classification was the first to be proposed (in 1965). Three main types were recognized: types I and II affect the ascending aorta; type III, distal dissection, begins distal to the left subclavian artery, sparing the proximal arch and ascending aorta. DeBakey's insight in distinguishing types IIIa (down to or ending above the visceral segment) and IIIb (extending downward to involve the abdominal aorta and iliac arteries) has proved to be extremely valuable in the 21st century because of the substantial impact on the prognosis and long-term results after TEVAR. The simpler and more recent Stanford Classification has also become well established, especially outside the cardiothoracic surgical community. It describes only 2 types of AD: type A, which signifies involvement of the ascending aorta; and type B, in which the ascending aorta is not affected.

Stanford type A is equivalent to DeBakey types I and II, and Stanford type B is equivalent to DeBakey types IIIa and IIIb. Approximately two thirds of cases of acute AD are type A, and the rest are type B.

Type B Aortic Dissection: Complicated versus Uncomplicated

Complicated dissection refers to evidence of thoracic aortic rupture (defined as the presence of blood outside the aortic wall), malperfusion (ischemia that involves the viscera, kidneys, spinal cord, or lower extremities), or rapid expansion in the distal arch or proximal descending aorta to a total aortic diameter of 4.5 cm or greater. These findings constitute a clinical imperative for intervention,because they immediately threaten life or limb. Approximately 30% of patients who present with acute type B AD have a complicated dissection. Malperfusion is perhaps one of the most intriguing and unique complications of acute AD, especially when it affects the visceral and renal vascular beds. The classic descriptions of the alleged pathogenesis of aortic branch closure include static mechanisms (branch vessel compression by the pressurized FL) and dynamic mechanisms (protrusion of a dissection flap into the branch-vessel origin). Various remedial techniques and approaches have been developed, such as surgical and endovascular fenestrations. Today, we know that malperfusion is largely the result of severe proximal compression or collapse of the TL in the chest by the pressurized and bulging FL. This understanding has enabled the present-day treatment of most patients with the relatively simpler, seemingly more effective approach of relining the TL in the proximal (and mid) descending aorta with a stent-graft to obliterate the entry site and redirect all blood flow down the TL exclusively. Another result of such new understanding and modern therapy has been the greatly diminished role of direct branch-vessel revascularization (stenting), and the use of fenestrations only rarely. In addition to the unequivocal and crucial diagnostic components of complicated dissection, it is unfortunately not unusual to see or hear mentions of other findings and softer criteria that only doubtfully justify intervention. These include unrelenting pain, uncontrolled hypertension, extension of the dissection, and image worsening. Most or all the 70% of patients who present with uncomplicated dissection should be treated medically, in adherence with currently available scientific evidence. Modern anti-impulse and antihypertensive pharmacologic therapy produces very satisfactory results in the acute stage, with an expected 30-day mortality rate of 10% or less at present. However, subsequent clinical follow-up and serial aortic imaging over time are crucial because AD patients are exposed to long-term life-threatening risks – including the formation of dissecting thoracic aneurysms in 20% to 30% of such patients.

Historical Overview

Daniel Sennert (1572-1637, a German physician and chemist, University of Wittenberg) was first to describe AD in 1628, although in retrospect this was a rather preliminary and simplistic

description… One hundred and thirty-three years later, in 1761, history came upon the most clear and irrefutable description with Frank Nicholls' publication of the autopsy findings of the King George II as discussed above. But it would not be properly named, "aortic dissection", until J-P Maunoir did so in 1802. Maunoir was a Swiss surgeon and ophthalmologist from Geneva, little known outside his own inner circles, so his contribution went essentially unnoticed, being promptly forgotten after 1819 when Laennec proposed the term "dissecting aneurysm" – a major disservice indeed as it injected great confusion in the definition of dissection versus aneurysm, a problem cardiovascular specialists continue to deal with to this day! René Laennec, inventor of the stethoscope, had become a medical celebrity in Europe by then, ensuring that his proposed label for AD would be almost immediately accepted, totally obliterating Manoir's much earlier and better designation which of course was completely correct from the beginning. The next major breakthrough was to take place in Houston more than a century later, on 7 July 1954, when the all-star team of DeBakey, Cooley, and Creech performed the first successful surgical resection of a dissecting thoracic aortic aneurysm. DeBakey and his associates went on to accumulate a vast clinical and surgical experience in the management of AD patients, reporting on a 20-year follow-up of 527 surgically treated patients in 1980. It was ironic that Michael DeBakey himself underwent and survived open surgery for type A dissection at the age of 97.

The contemporary recognition and treatment of AD was ushered in by 2 major developments. The first was the creation of the IRAD in 1996, which proved to be crucially important as the collaboration of 20 international centers of excellence and dedicated clinical investigators in 9 countries produced an astonishing amount of information and solid data. Their contributions on many levels constitute the biggest share of knowledge and understanding that we have gained about AD during the last 20 years. The second was the publication on the May 2, 1999, issue of the New England Journal of Medicine containing two back-to-back landmark papers reporting some of the earliest clinical experiences and results with endovascular stent-graft intervention for acute type B AD. They showed how the surgical treatment standard could and should be challenged moving forward. These papers (by Nienaber et al. and Dake et al.) heralded the endovascular era in AD management.

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