Prevention of Venous Thrombosis and Pulmonary Embolism

J. G. Sharnoff

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To
Ian Douglas-Wilson
Former Editor of the Lancet

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Preface

This book has been prompted by recent advances in the safe prevention of thromboembolism by subcutaneous heparin prophylaxis, in particular postoperatively. It has been correctly called by S. Sherry a major breakthrough in medicine. Although thromboembolism was first recognized by Laennec in 1819 and defined by Virchow in 1846, its development was not well understood and its prevention escaped our best efforts until now. This all-too-common, sudden, unexpected and unwanted form of morbidity and mortality, always referred to in the surgical patient as postoperative pulmonary thromboembolism, has now become the major complication of all surgery. However, it occurs with equal frequency in hospitalized non-operative patients as well, so the latter are also in need of this prophylaxis if this calamity is to be avoided. The mass of literature generated in the past few years has produced some confusion as to which of a number of methods of heparin prophylaxis gives the best results. It is the intention of this book to help clarify the situation and thereby resolve the problem by offering a procedure of subcutaneous heparin prophylaxis which is acceptable, safe and simple to administer. Recent technical advances offer the hope of attaining this goal.

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Epidemiology

Although Laennec first described the pulmonary infarct in 1819 and Virchow explained its development as a deep vein thrombosis and embolism to the pulmonary arteries in 1846[63], little has been accomplished since then in the prevention of this often fatal phenomenon. The reason for this is attributable to the fact that the development of thrombosis still remains partially understood. This is despite the fact that Virchow in 1856[64] described a triad for thrombosis which is still valid today; he suggested that blood stasis, vascular wall injury and alteration of the blood constituents are the main causes of thrombosis. Many complex explanations have been and are being suggested to explain the last mentioned; but so far our best efforts have failed.

With the advent of antibiotics came the conquering of most common bacterial infections and the control of some viral infections, so thromboembolism has assumed a more prominent position as a cause of morbidity and mortality. Thus, it has been estimated that more than 200 000 thromboembolisms occur in the United States annually[39,65]. In the event of death, the autopsy has disclosed an almost equal frequency of thromboembolism in both medical and surgical patients. In the latter it often occurs after operation and has therefore been referred to as postoperative pulmonary thromboembolism. However, it may also be observed with some frequency during or immediately following surgery and producing cardiopulmonary arrest and death. These deaths are to this

day usually attributed to anaesthesia[53,56] which has been the case since the introduction of anaesthesia with chloroform and ether. Autopsy evidence indicates that pulmonary thromboembolism is the most frequent (70%) cause of death[48]. It was deTakats[17] who described the death so aptly as 'found suddenly dead in bed'. In most instances anaesthesia is not implicated and death occurs without premonitory signs within minutes, leaving no time for embolectomy or thrombolytic therapy[11,14,23,45,59]. This makes the need for prophylactic treatment mandatory.

Fatal pulmonary thromboembolism is undoubtedly chiefly a hospital-based problem[36]. In this respect it lends further support to one of Virchow's postulates in the development of thrombosis, namely blood stasis. The hospitalized patient is greatly restricted in his movements and is often completely immobilized[48]. This induces blood stasis in the veins of the lower limbs. Although older patients are at the greatest risk, fatal pulmonary thromboembolism can occur at any age[19,31,32]. It has on occasion been reported in neonates[66], but is more frequently observed beginning with the teenage years and increasing in frequency with advancing age. Orthopaedic patients requiring surgery for hip fractures, total hip replacement and leg amputations[20,21,45–47,52] are at the greatest risk, followed by patients requiring thoracoabdominal surgery[15,26,67] and women during the early post-partum period[1,7,42]. The non-surgical patients at greatest risk in order of frequency are those with acute myocardial infarctions, congestive heart disease,[22,25,34,54,58,68] septicaemias[16], diabetes mellitus[11,36], cerebrovascular accidents, malignancy[2,8,10,40,55], obesity, and young women using oral contraceptives[3,12,13,39,44,61,62].

Fatal pulmonary thromboembolism has also been observed at post-mortem following minor surgery including dentistry. This is usually in cardiopulmonary arrest cases in association with general anaesthesia as mentioned above. Although rarely observed in non-hospitalized healthy individuals, it has been noted in individuals during prolonged airplane travel[57], television watching, and in sedentary occupations and prolonged rest periods[27,50,68]. Fatal pulmonary thromboembolism may occur in these individuals with or without the presence of lower extremity varicosities[9].

Recent reports indicate that the incidence of fatal pulmonary thromboembolism is greater in Great Britain and on the European continent than in the United States. This may not be a statistical discrepancy but a reflection on the difference in hospital care. Length of hospitalization in Great Britain is as a rule longer than in the United States. As mentioned above longer periods of immobilization are a distinct hazard[50].

Other conditions which may be associated with thromboembolism are anaemia and polycythaemia. There also appears to be a seasonal variation in the frequency of pulmonary embolism in the temperate zones, the greatest incidence being in the spring and fall[51]. It has been postulated that this seasonal difference is due to the renewed physical activity following periods of greatest inactivity during the cold of winter and the heat of summer. Tropical and subtropical areas on the other hand appear to have a lower incidence of thromboembolism. This may be explained by the lack of seasonal difference and the constant activity in all seasons[24,30,44,60]. The above is consistent with the findings in the classic study of Morris *et al.* on London bus employees[38]. The authors reported a higher incidence of coronary artery thrombosis in the sitting bus drivers compared with the lower incidence observed in the physically active conductors. Although these were cases of arterial thrombosis, they may well reflect the cumulative effect of coagulation factors in the immobile bus drivers and the development of hypercoagulable blood according to the third postulate of Virchow (see below).

Statistical findings tend to be either conflicting or unrevealing[14,40]. The reported incidence of pulmonary embolism as a cause of death, from the US National Center for Health Statistics, is very inaccurate. This is because of the low autopsy rate and the difficulty in differentiating pulmonary embolic death from other forms of sudden death. Thus the Vital Statistics of the United States for the year 1976[65] reported only 11 513 deaths from pulmonary embolism and infarctions, which is probably a very inaccurate figure.

The data for the incidence of pulmonary embolism according to sex reveal little difference. The US Vital Statistics indicate an