

INTRODUCTION

The first published description of SVCS is generally credited to William Hunter who in 1757 described the syndrome as consequence of a syphilitic aneurysm of the ascending aorta. At autopsy, Hunter noted that the SVC and the innominate vein were "both so much compressed by the dilated artery, as hardly to have any thing left of their natural capacity and appearance. One of the first clinical descriptions of SVCS caused by a malignancy was made by William Stokes in 1837. Stokes described a patient whose face was bloated, pale, and slightly edematous....with an appearance of the eyes as if the balls were protruded from their sockets and a marked dilatation of nostrils during breathing, gave an expression of distress and suffering(1),(2)

SVCS was regarded as a relatively rare clinical entity before the mid-20th century. In a review by McIntire and Sykes (1948), provided an extensive review of all literature published on SVCS, McIntire and Sykes felt that superior vena cava obstruction occurs much more frequently than is generally suspected and the many cases are doubtless hidden because of being reported as cases of the primary pathology(3)

The dominant process causing SVC obstruction before 1948 was infections. nearly two thirds of cases were from benign etiologies such as syphilitic aortic aneurysms, chronic fibrous mediastinitis from tuberculosis or syphilis, and phlebitis with thrombus formation, whereas only one third of the cases were from primary thoracic tumors. with improved antibiotic management of infectious disease, syphilis and tuberculosis are now extremely rare causes of SVCS. Paralleling the dramatic increase in

lung cancer incidence, thoracic malignancies have become far and away the leading cause of SVCS(3)