Aneurysmal Dilatations of the Superior Vena Caval System *

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PRIOR to the use of autogenous vein grafts to span defects produced by excision of arterial segments, the term *aneurysm* was seldom, if ever, applied to purely venous structures. The production of *significant aneurysmal dilatation* in such autogenous vein grafts when not completely surrounded by parallel bundles of voluntary muscle resulted in restriction of autogenous venous grafts for reconstruction of arterial flow in the four extremities. True or pure primary venous aneurysms were not described in textbooks of pathology or surgery prior to 1962.[34]

Our interest in this subject was aroused by the finding of a pure or primary (congenital) aneurysm of the superior vena cava in a university student in February of 1948.[2] Review of the literature revealed a single previous report of the pure venous aneurysm, that of Harris.[28] He described "a congenital venous cyst of the mediastinum" which was found at autopsy of a five-month-old infant. This sacculation had direct connections via feeder veins with the left innominate, inferior thyroid and right jugular veins. Following a visit to the University of Istanbul, Turkey, in 1962, we became aware of the presentation of a similar type case to the Turkish Medical Association in 1948.[15] This was a large solitary, saccular, congenital aneurysm above and behind the ear of a 14-year-old boy, which enlarged significantly with exercise or fatigue. Simple ligation of the single vein of entry and exit produced cure. In 1956, Lawrence and Burford[32] reported the successful excision of a solitary venous aneurysm which communicated via a 2 mm. stalk with the superior vena cava immediately above the entrance of the azygos major vein. The senior author (O. A. A.) has the privilege of including herein the initial presentation of two further examples of this type of primary or pure venous aneurysm studied and treated by Scannell[51] and Robertson.[47] The first of these was a soft, vascular mass in the right supraclavicular region which communicated with the external jugular vein by one small venous channel. The pathologist chose to term this *a venous hemangioma*. Robertson's case occurred in a woman and proved to be a massive venous sacculation communicating directly into the left innominate vein. Aneurysm of the splenic vein has been reported by Lowenthal.[35]

The above six cases had no known antecedant trauma to the area involved, demonstrated no arteriovenous communications, and were not the result of an associated cardiac anomaly which could produce abnormal pressure or blood flow within the involved structures. A solitary area of vein sacculation connected by a single channel to a major venous structure, or marked fusiform dilatation occurring in the course of a major vein, does not properly fall under the classification of venous angiomata. We believe that such lesions might be better

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classified as two basic sub-types of pure venous aneurysms.

Consideration of these cases developed an appreciation for the need of an over-all classification of aneurysmal and other significant dilatations of veins. Although this presentation is confined to a description of such pathology as it involves the superior vena caval system, it is hoped that the outline suggested would apply to the inferior caval system as well. Pathologic dilatation of the pulmonary veins and the coronary venous system might require a separate classification.

The basic purpose for this discussion, therefore, will be threefold: 1) To emphasize the existence of venous aneurysms and the need for clarification of our understanding of the same; 2) to present new exemplary cases and review the pertinent

TABLE 1. *Proposed Classification of Aneurysmal Disease Superior Vena Caval Systems*

I. Congenital lesions
  A. Primary or simple
    1. Fusiform (1*)
    2. Saccular or *diverticular*—(2)
  B. Secondary or complex (resulting from associated anomalies)
    1. Total anomalous pulmonary venous return—(9)
    2. Anomalous vena cavae
      a. Hypoplasia or agenesis inferior vena cava—(3)
      b. Persistent levoatrial cava
      c. Complete transposition one or both cavae
  C. Obstructive anomalies (intravenous diaphrams, etc)
  D. Miscellaneous

II. Acquired lesions
  A. Primary
    1. Fusiform—unsupported vein graft arterial replacement
  B. Secondary
    1. Trauma (*false* type)—(1)
    2. Obstruction (mediastinitis, cirrhosis): varices—(2)
    3. Adjacent neoplasms—(1)

III. Pseudo-aneurysms
  A. Transient
    1. Cardiac failure (numerous)
    2. Cardiac failure plus Budd-Chiari Syndrome—(1)
  B. Venous neoplasms (angiomata)—(2)

IV. Arteriovenous aneurysms
  A. Congenital
    1. Systemic vessels
    2. Systemic and pulmonary vessels (1)
    3. Cirsoid
  B. Acquired
    1. Trauma—(6**)
    2. Surgical—purposeful, non-purposeful—(4)
    3. Associated disease (lues)

* Numbers in parentheses indicate cases of that type encountered by the authors.
** Includes only one of numerous cases previously reported by Elkin.

literature; and 3) to analyze the clinical significance of the individual types as well as the appropriate diagnostic and therapeutic measures recommended.

A suggested classification is presented in Table 1. This is the result of a study of the various lesions reported to date and is subject to considerable change inasmuch as the present literature is rather limited.

Type I.A. congenital lesions of primary type are those in which there is no associated abnormality producing increased blood flow or elevation of venous pressure. The fusiform lesions include those reported by ourselves[1,2] and Dorken.[15] A similar type lesion of the splenic vein has been presented by Loewenthal and Jacob.[35] The saccular type are represented by those described by Harris,[28] Lawrence and Burford[32] and the two new ones herein reported through the courtesy of Scannell[51] and Robertson.[47] This latter group may cause some dissension by pathologists who prefer to include them among the various venous angiomata. The fact that they are large solitary sacculations and have a single communication with the major venous trunk might suggest the term *venous diverticula.* They are neither cirsoid nor neoplastic and are free of arteriovenous components. We believe they deserve to be classified as true venous aneurysms.

Type I.B. The second group included under Type I (congenital lesions) includes those congenital abnormalities of the heart, pulmonary veins or vena cava which produce an inordinate flow within a venous structure or persistently elevated venous pressure. One might consider these as *congenital venous aneurysms of an acquired type.* The literature is replete with descriptions of this type of lesion. Excellent summaries in this regard can be found in the books of Taussig,[58] Gould[26] and Nadas.[40] Total anomalous pulmonary venous drainage can be of several types, but the type involving a single collecting trunk which empties via the left innominate vein into the superior vena cava is that one most prone to produce huge aneurysmal changes. The lesions produced by actual vena caval anomalies are less common, but they include a hypoplasia or agenesis of the inferior vena cava;[5,17,38] Tuchman[59] described a persistent left vena cava entering the left atrium, while Taussig[58] has so well depicted the situation wherein both venae cavae empty into the left atrium along with the pulmonary veins. The cavae can show marked aneurysmal dilatation under any of the above situations. Of considerable interest here is the recent report of Fox and Goss[22] who produced transposition of the left superior vena cava in the offspring of rat dams injected with trypan blue solution 8½ days past insemination.

Type I.C. Significant local or widespread dilatation of veins as the result of a congenital obstructive process within a major veni is rare. Rafinski[43] described one in the superior vena cava causing death in a five-month-old infant, while Cucci[13] removed an anomalous diaphragm-like valve which was producing obstruction of a left subclavian vein.

Type II.A. The second grouping includes sundry acquired lesions. The only pure, primary type would involve those instances wherein autogenous vein grafts have been used to span segmental arterial defects in the thorax or abdomen of experimental animals. On rare occasions these have been used in humans, but such use now would be confined to unusual emergency situations, and then only until an adequate prosthesis may be obtained to replace it.

Type II.B. The first subtype in this group includes *false* aneurysms of veins due to trauma or caused by adjacent neoplasms. Previous reference to the same has not been found in the literature. However, the authors present herein individual examples of each of these. The second subtype has been previously reported by us[33] as the result of cirrhosis of the liver. Renbouen[46] described this as the result of a thrombosis

of the superior vena cava of 50 years' duration.

Type III. In the third basic group are found the pseudoaneurysms. Fleischner and Udis [21] stated that the normal width of the azygos shadow is rarely more than 5 mm. and may enlarge to as much as 1.3 mm. with chronic heart failure. One of our patients who has been presented elsewhere [54] presented an azygos shadow 3 cm. in width as the result of chronic heart failure superimposed upon Budd-Chiari's syndrome of high inferior vena cava thrombosis. Other reports of azygos dilatation simulating mediastinal tumor include those of Sayer,[49] Schmidt,[52] Stauffer,[56] and Bronte-Stewart,[10] and Magbitang.[36] Venous angiomata and leiomyosarcomata can produce massive venous dilatations and may appropriately belong in this group.

Type IV.A. A final grouping includes the arteriovenous fistulae. The writings of Reid,[45] Elkin,[19] and Holman [29, 30] are replete with instances of various types of these lesions occurring throughout the body. The congenital lesions of this type are relatively rare but such have been described which involve the 1) phrenic; [20, 27] 2) pericardial; [14] 3) tonsillar; [6] 4) subclavian;[57] and 5) internal maxillary and pterygoid,[24] as well as other vessels. These have involved systemic arteries and veins. Still another group of congenital arterio-venous aneurysms include those joining bronchial arteries to pulmonary veins,[4] and finally in this category are systemic vein to pulmonary artery (reported herein), and pulmonary vein to systemic vein (levo-atriocardinal vein anomaly).[18] Cirsoid aneurysms of the chest wall [8, 11, 37, 42] and scalp [44] have been described.

Type IV.B. The acquired group of arteriovenous fistula emphasizes the role of surgery as well as nonpurposeful trauma. Surgery may purposely produce one temporarily to promote unilateral overgrowth of one extremity [31] or as an attempt to increase coronary collateral circulation.[9] However, Elkin [19] emphasized that "trauma incident to en masse ligation of vessels during surgical operation has become recognized as a cause of arteriovenous fistula." Some of the branches of the superior vena caval system which have become so involved by surgery or by accidental trauma include the 1) phrenic; [20, 27] intercostal (reported below); 3) internal mammary; [25] 4) thyroid; [16] 5) subclavian; [61] 6) vertebral; [53] 7) cervical; [41] 8) humeral; [7] 9) axillary; [55] and other vessels.

Under rare circumstances arteriovenous aneurysms can be produced by specific disease. Franklin and Pollock [23] have reviewed a total of 120 cases of thoracic aorto-caval aneurysms reported to date. These have been mostly luetic and had a life duration of from one to 526 days after onset, with average expectancy of 45 days.

Case Material

To date we have encountered a group of 32 cases which could fall within the aforementioned classification. These are specified in Table 1. For the sake of brevity we have chosen to limit specific description to the more unusual cases.

Type I.A. Congenital Lesions.

A. Fusiform. H. S., a 19-year-old student—lesion involved superior vena cava reported in detail previously.[2] Exemplary roentgenograms are shown in Figure 1 a–d. Lesion was wrapped with reactive cellophane July 25, 1948, and adjacent normal venous structures protected by nonreactive material. Patient has remained well for 15 years, no detectable abnormalities of venous pressure or circulation time. Lesion has remained stable in size on roentgenograms since surgery (Fig. 1 a–d).

Saccular or Diverticular. Two cases through the courtesy of colleagues. Relative to the first of these Scannell [51] wrote, "The patient was 11 years old . . . noted a soft fluctuant mass above the right clavicle. This mass was particularly noticeable after exercise, and seemed to have increased during the antecedent two years . . . a definite bruit beneath the clavicle . . . was later pointed out to be a normal venous hum. There was no particular evidence of circulatory embarrassment in the patient's right arm. . . . When an incision was made over the clavicle, a soft vascu-

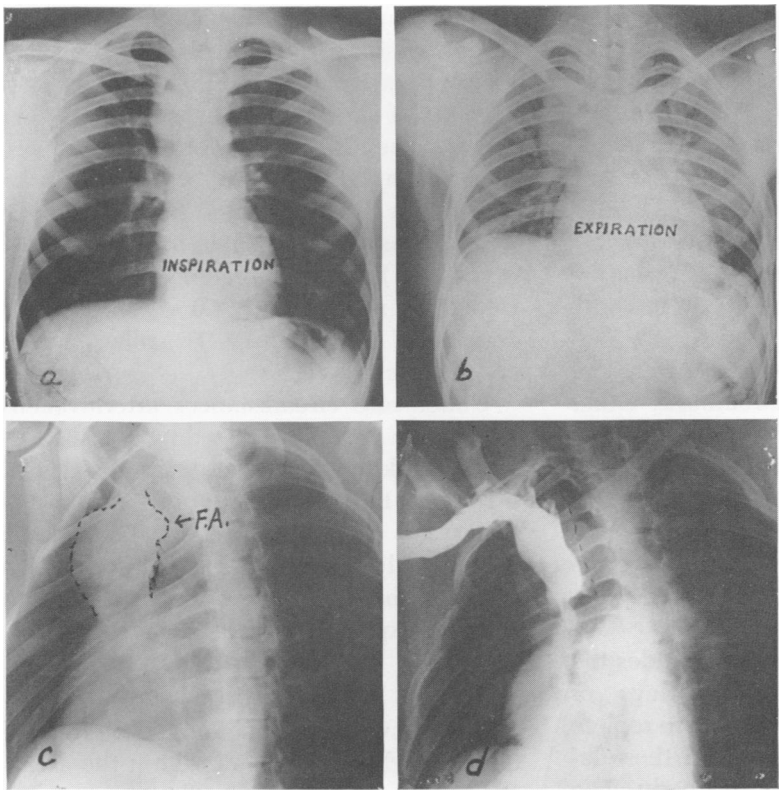

FIG. 1. (H. S.) a, b. Inspiration and expiration. Note changes in size and shape of congenital primary fusiform aneurysm of superior vena cava (type I.A.1) in right upper paramediastinal area. c. Oblique view of superior vena caval aneurysm. Borders outlined by dotted lines reveal false aneurysm (F. A.) probably produced by catheter. d. Angiographic visualization of aneurysm 2 years after circumferential wrapping with reactive cellophane.

lar mass was discovered communicating with the external jugular vein by one small venous channel. It was not really an aneurysm but rather a venous hemagnioma; and this was confirmed by the pathological report."

The second case history in this category was described by Robertson.[47] This patient was a young woman who noticed some dyspnea. Roentgenogram revealed a large opacity occupying the upper two-thirds of the left hemithorax. In-

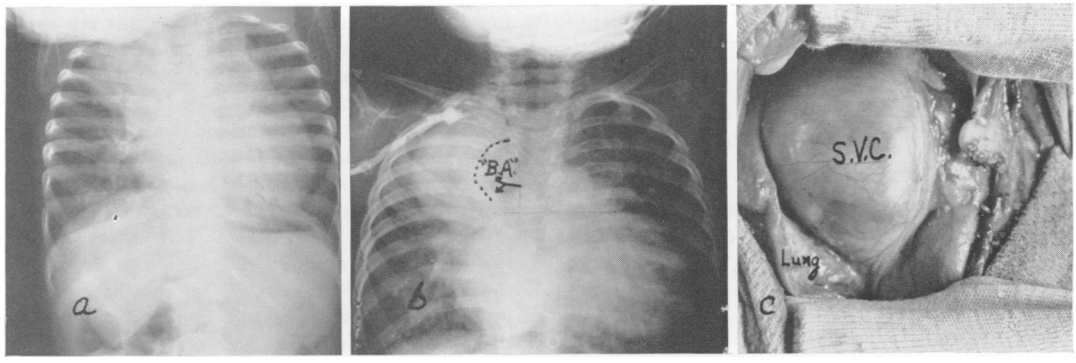

FIG. 2. (N. C.) a. Postero-anterior roentgenogram portraying "figure-of-eight" or "snowman shadow" considered characteristic of total anomalous pulmonary venous drainage. Unusual amount of shadow seen in right upper paramediastinal area was produced by massive aneurysmal changes in superior vena cava. b. Venous angiogram revealing huge aneurysm of superior vena cava area. "B.A." ("blackout" or "washout area") demonstrates region of decreased concentration of contrast media produced by inflow of pulmonary venous blood through orifice of left innominate vein. c. Photograph taken at time of thoracotomy showing large, thin-walled aneurysm of superior vena cava (S.V.C.) compressing adjacent lung tissue.

vestigation and surgical exploration revealed the lesion to be a massive, solitary, cystic structure containing venous blood. This structure communicated with the left subclavian and innominate vein juncture by a single short communicating structure, so short as to suggest the lesion to be an actual diverticulum of the main venous trunk.

Type I.B. Congenital Lesions.

B. Secondary or Complex, Resulting from Associated Anomalies. Total anomalous pulmonary venous return. We have encountered nine such cases in our clinic of various types. The first case seen (June 1948) happened to show the most advanced aneurysmal changes of the superior vena cava (Fig. 2 a–c). This 12-month-old girl suffered from progessively more severe dyspnea and tachycardia. Standard postero-anterior roentgenograms showed the *snow-man, figure-of-eight* described by Taussig.[58] The opacification of the upper half of the right hemithorax was more extensive than usually seen. Angiocardiograms revealed the aneurysm of the superior vena cava and showed the characteristic *black-out* of the medial aspect of the contrast media filled area due to significant inflow of blood from the left innominate vein. This patient was subjected to surgery prior to the days of cardiopulmonary bypass, and the procedure performed was unsuccessful. The massive aneurysm as it appeared during surgery was photographed (Fig. 2c); a dramatic turbulence of admixed venous and oxygenated blood within the thin-walled aneurysm was a conspicuous finding. Subsequent cases of a similar nature have been operated upon and the

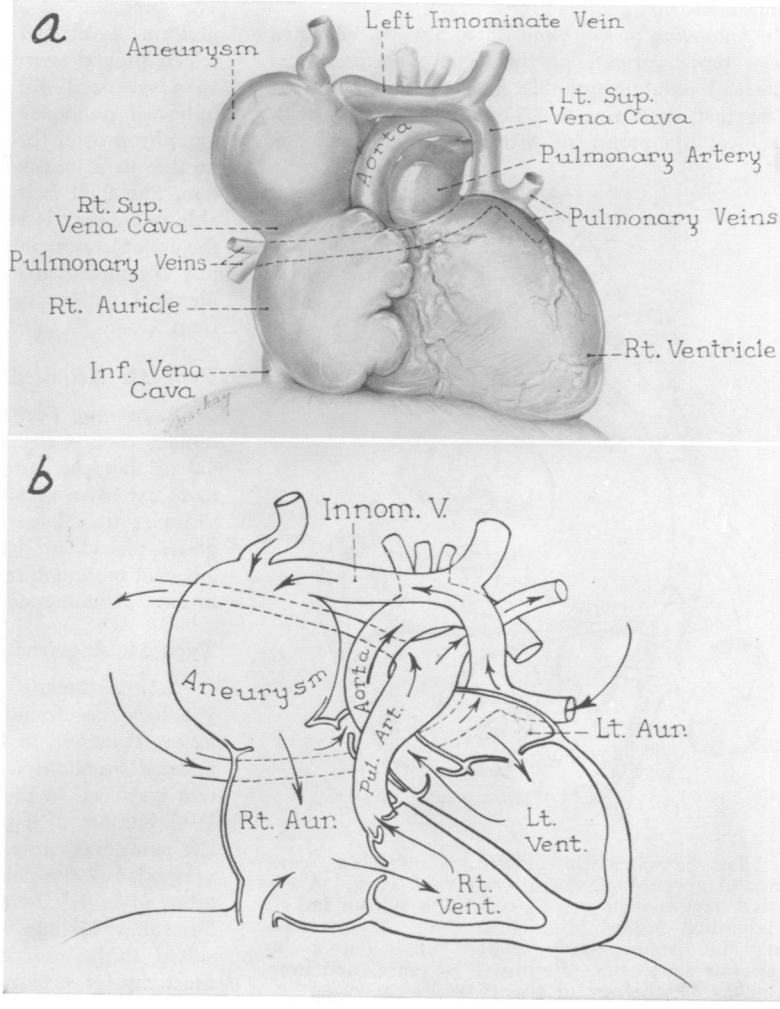

FIG. 3. a. Drawing of anatomico-pathologic situation present in N. C., wherein all pulmonary venous drainage enters common collecting trunk behind heart, thence via another venous trunk to left innominate vein and finally into aneurysmal superior vena cava. b. Outline drawing used to clarify cardiovascular anomaly shown in a.

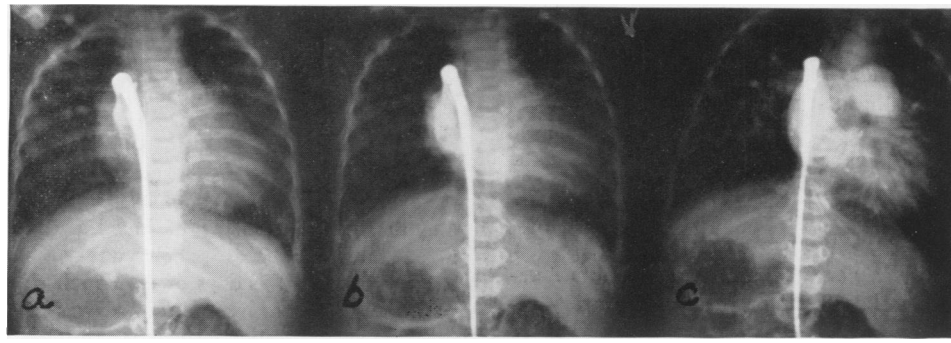

FIG. 4. Series of angiograms obtained in study of patient with congenital pulmonic stenosis and "congenital absence of inferior vena cava." a. Cardiac catheter introduced via femoral vein enters right atrium through azygos major vein. Initial injection outlines area of azygos entry into right atrium. b. Right atrium and early right ventricular filling. c. Post stenotic dilatation of pulmonary artery demonstrated.

aneurysmal dilatation of the superior vena cava diminished markedly (Fig. 3 a, b).

Anomalies of the Vena Caval System. We have seen three patients of this type who presented classical roentgenographic findings (Fig. 4 a–c). The first case was a 25-month-old infant with classical symptoms of tetralogy of Fallot. The vena caval status was defined by angiography. The presence of a right aortic arch led us to perform a Blalock operation on the left side, which proved successful. Our second patient was an 11-year-old girl with severe cyanosis due to universal pulmonary arteriovenous fistulae. Angiography proved the right paramediastinal mass to be due to a massively dilated azygos venous system. She died from her extensive and uncorrectable pulmonary lesions, and postmortem confirmed the roentgenographic findings. A third infant has just completed angiographic studies and has the identical pattern so well portrayed in a drawing from Gould [26] (Fig. 5).

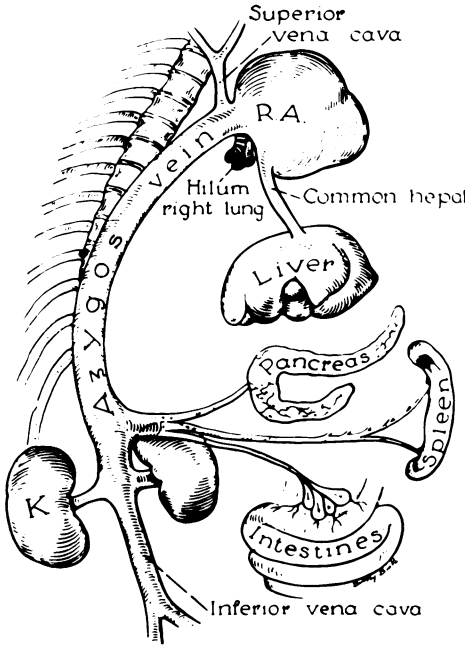

FIG. 5. Schematic drawing illustrating "congenital absence of the inferior vena cava." A dilated azygos vein acts as collecting system for all abdominal organs other than liver. Azygos vein may be "aneurysmally dilated" at it enters the superior vena cava. (Reprinted by permission from Gould's "Pathology of the Heart.")

Type II. Acquired Lesions

A. Primary Fusiform. Vein grafts in animals to replace excised segments of iliac arteries, abdominal or thoracic aorta have developed fusiform or more extensive aneurysmal changes in our laboratories as they have elsewhere.[39] Autogenous vein grafts placed in extremities and surrounded by adjacent musculature have not shown such changes to date in numerous human cases.

Type II. Acquired Lesions (Secondary)

B.1. Secondary to Trauma (False Type). We have not found a report to date of such a lesion. However, in 1949 we did see the following unusual situation: J. F., a 29-year-old Negro man, was first seen in the Grady Memorial Hospital in 1947 because of a penetrating stab wound in the left parasternal area. An extensive hemothorax developed, but the patient refused further treatment after an initial thoracentesis and was discharged. Screening 72 mm. roentgenograms in 1949 resulted in his re-admission and exploratory thoracotomy for suspected *anterior mediastinal tumor*

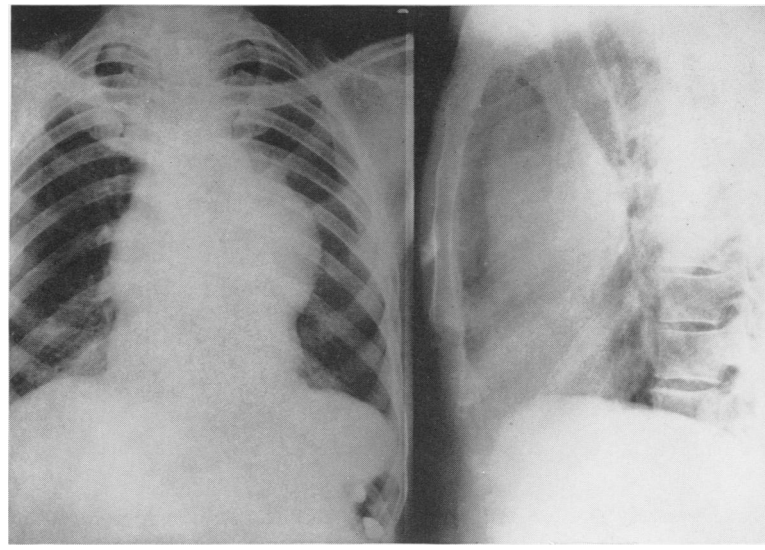

FIG. 6. (J. F.) PA and lateral films demonstrating a false aneurysm of left internal mammary vein; study performed 2 years following stab wound by ice pick in left parasternal area.

(Fig. 6). No angiographic studies were performed prior to surgery. At surgery the lesion proved to be a false aneurysm arising from the left internal mammary vein, presumably from the trauma which occurred two years previously. Delicate dissection proved the lesion to be totally separate from the internal mammary artery which was actually left functional and intact within the patient. The sac contained well organized clot and had been non-pulsatile on fluoroscopic examination (Fig. 6 a, b).

B.2. Varices. Secondary to Mediastinitis, Cirrhosis of Liver. Two patients in this category have been seen, in both of whom the underlying pathology was severe hepatic cirrhosis. In one patient (Fig. 7) the basic lesion was diffuse aneurysmal enlargement of the hemiazygos system. In the other there was a remarkable degree of massive varices of the posterior mediastinum (Fig. 8 a, b). The patients have been previously described.[33]

B.3. Secondary to Neoplasms. This 32-year-old man was seen at the Atlanta VA Hospital in 1954. Major chest injury occurred in 1945 and roentgenograms at that time revealed some abnormality of the left upper paramediastinal area. Actually a somewhat similar lesion was noted on re-examination of his induction films in 1942. We performed venous angiography in 1954. A distinct, saccular aneurysm of the inferior surface of the left innominate vein was demonstrated. Important accessory findings were the irregular edges of this sac and its incomplete emptying of contained contrast material throughout the later stages of this study. At surgery a vascular mass was found to occupy the thymus gland which was firmly adherent to the inferior surface of the left innominate vein, and a secondary saccular or diverticlate projection of this vein had been produced. Thymectomy and surgical resection of the *diverticulum of the vein* with reconstruction of the innominate vein wall was well tolerated. The excised specimen revealed a diffuse malignancy of the thymus, well encapsulated, and *adherent vein aneurysm wall.*

Type III. Pseudo-Aneurysms (Transient and Miscellaneous Dilatations)

III.A. Transient. The enlargement of the azygos vein with cardiac failure especially when examined radiographically in the supine position has been

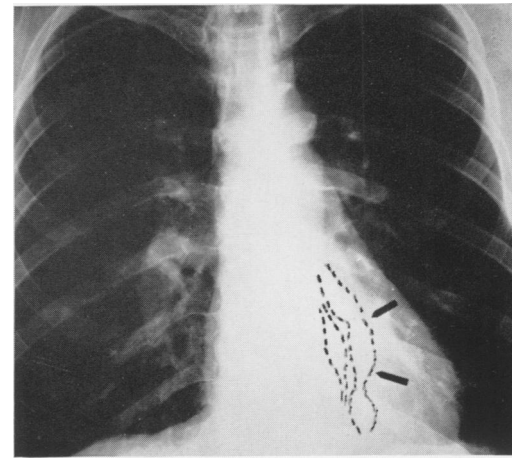

FIG. 7. (P. B.) Overexposed Buckey roentgenogram of chest revealing diffuse, fusiform dilatations of hemiazygos venous system in a patient with hepatic cirrhosis. Venous dilatations here are more fusiform and less saccular than those in Figure 8.

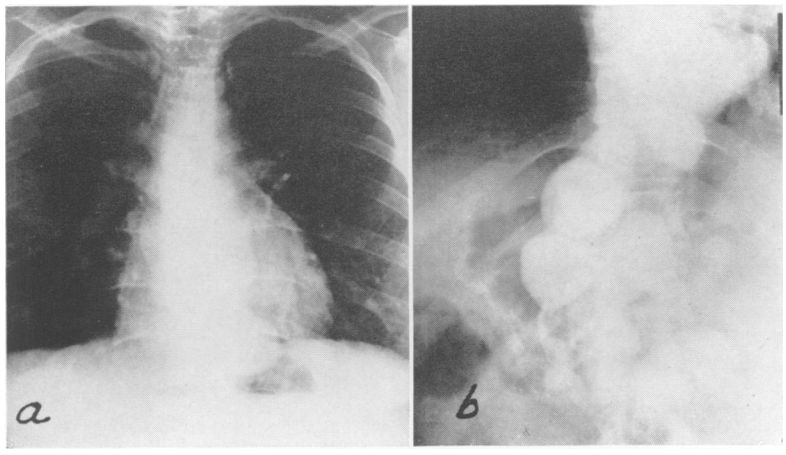

FIG. 8a, b. (P. B.) a. Film of chest. b. Spot film (angiogram) demonstrating massive aneurysmal dilatations (varices) of hemiazygos venous system in association with cirrhosis of liver. (Courtesy of Dr. W. Molner.)

seen frequently in our institution.[2] An unusual case seen in our medical school occurred in a 49-year-old white man in April 1957 and has been previously described.[54] The patient had suffered an extensive thrombosis of the inferior vena caval system up to the level of the right renal vein 22 years previously. The superimposition of chronic cardiac failure due to hypertensive and cardiovascular renal disease in 1957 produced a remarkable distention of the azygos vein (Fig. 9 a–c). This lesion was nonpulsatile. Following extensive therapy a remarkable decrease in the size of this lesion occurred.

III.B. Venous Neoplasms. J. L., a 49-year-old man, presented in 1958 with an extensive opacity in the left chest (Fig. 10 a, b). Following extensive study, exploratory thoracotomy was performed. The left chest wall, entire mediastinum, and diaphragm were covered with huge, tortuous, vein-like structures with interspersed vascular neoplastic tissue. Biopsy revealed it to be an angiosarcoma. Subsequent radiation therapy was somewhat palliative.

Type IV. Arteriovenous Aneurysms

IV.A. Congenital–Simple (Systemic and Pulmonary Vessels). In 1948, J. S., an 11-year-old white boy, presented with classical symptoms and findings of tetralogy of Fallot. Dr. R. B. Logue heard a localized continuous murmur in the third left interspace about 8 cm. lateral to the sternum. He suspected a bronchial artery to pulmonary artery fistula. At the time of a left Blalock operation (patient had a right aortic arch), we were surprised to find a vascular mass in the left upper lobe. Dissection proved this to be an aneurysm

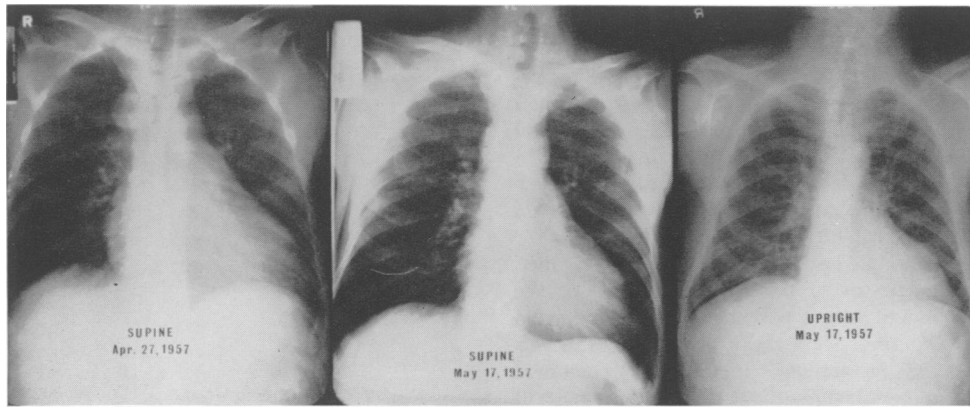

FIG. 9. (J. R. B.) Roentgenographic studies demonstrating azygos vein dilatation simulating a mediastinal tumor. a. Pseudo-aneurysm as it appeared prior to treatment. b. and c. Marked decrease in size of azygos phlebectasia following 20 days of medical therapy. Note larger size of lesion when studies are made in supine position.

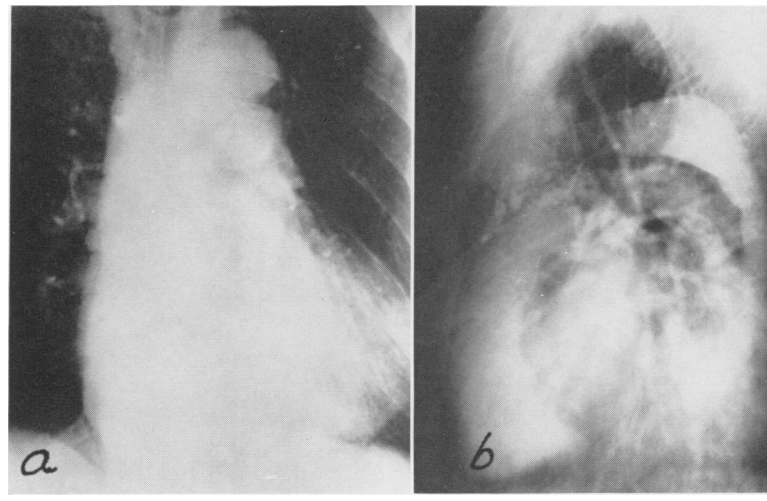

FIG. 10. (A. S. R.) a. Antero-posterior and b. lateral angiograms demonstrating massively dilated venous channels widespread in left hemithorax. Exploration revealed irrsectable angiosarcoma.

which had formed at the juncture of the segmental artery to the superior segment of the left upper lobe and an aberrant vein connecting to the left innominate vein. This partially imitated the persistent left cardinal vein situation wherein a persistent vein drains the superior pulmonary venous system into the left innominate vein. We have seen this latter situation on two occasions (one in association with persistent ductus arteriosus, the other in a patient with interatrial septal defect and transposition of right pulmonary veins). Fortunately, in the former instance findings were docu-

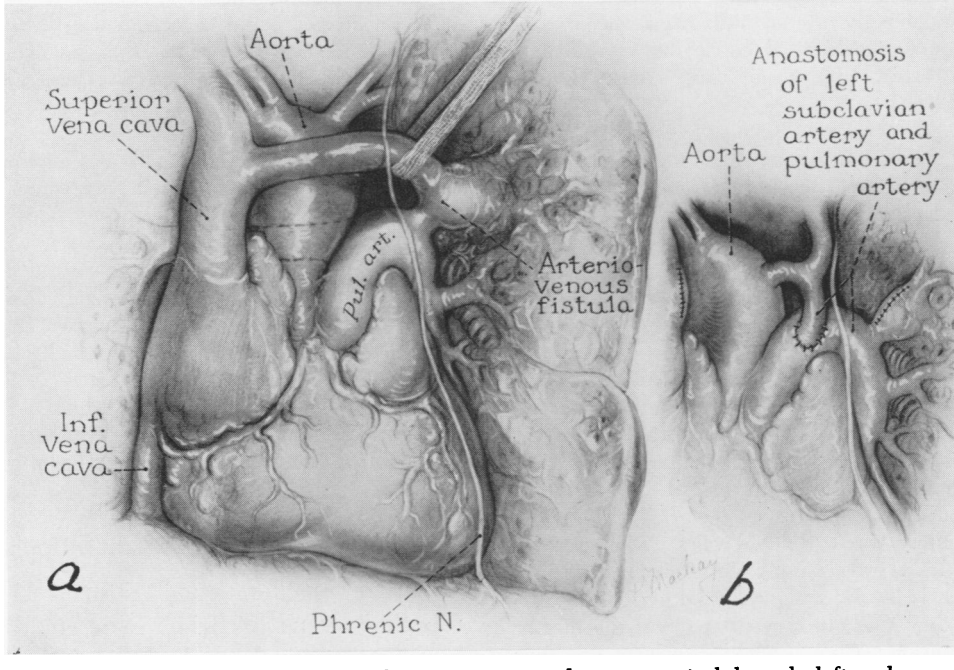

FIG. 11. Patient J. S. a. Unusual communication between apical branch left pulmonary artery and left innominate vein. Note dilatation at venopulmonary artery junction labeled "arteriovenous fistula." Patient had tetralogy of Fallot with right aortic arch. b. Following transection of fistulous communication and preservation of pulmonary artery, left subclavian arising from left innominate artery was anastomosed to pulmonary artery (Blalock-Taussig procedure).

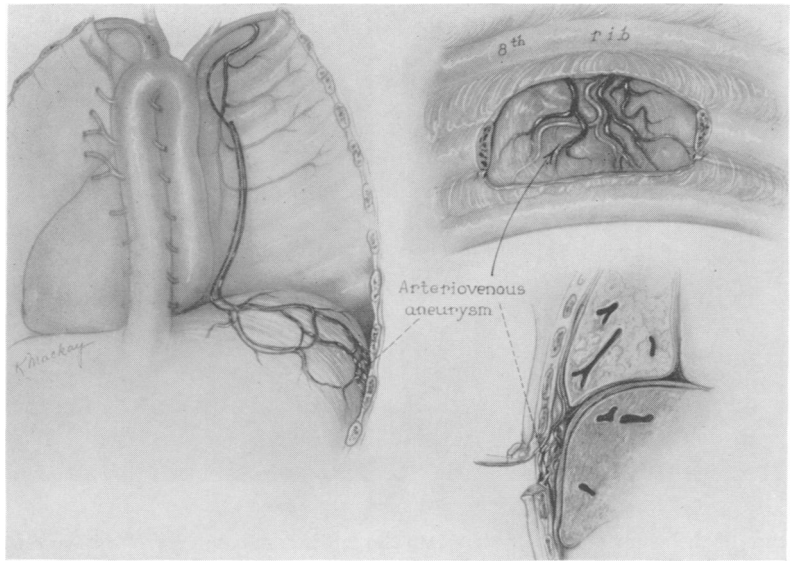

FIG. 12. Arteriovenous aneurysm occurring after multiple thoracenteses. (Originally published by Elkin, D. C., J.A.M.A., 141:531, 1949.)

mented by motion pictures and was truly an anomaly involving the left pulmonary arterial system. Pressure studies suggested that blood was flowing from the systemic vein to pulmonary artery. The fistula was excised with reconstruction of the pulmonary arterial wall. A classical Blalock operation was performed employing the left subclavian artery. Improved oxygenation resulted (Fig. 11 a, b).

IV. B1. Acquired–Trauma. Figure 12 is taken from a previous communication by Elkin [20] concerning a patient treated at Emory Hospital in 1949. She had undergone numerous antecedent thoracenteses considered to be the cause for this arteriovenous fistula of the phrenic vessels. In 1960 we saw D. S., a 29-year-old white man, who had been treated for traumatic hemothorax elsewhere requiring multiple thoracenteses. Six months after recovery from this episode he was noted to have a continuous murmur over the posterior aspect of the left eighth rib (the site of previous thoracenteses). Local compression could occlude the murmur. Attempts to record the same by phonocardiography were only partially successful. Angiography was of limited value. Total excision of the lesion was simply performed (Fig. 13). The vessels involved proved to be perforating vessels as they came to the surface over the erector spinae muscles. Cure resulted. We have had four arteriovenous fistulas of the subclavian artery and vein occurring following stab wounds. This has been satisfactorily handled by the recommended surgical approach with excision of the clavicle.[61]

IV. B.2. Acquired–Surgical (Purposeful or Nonpurposeful). Recently we have seen this type of lesion in a patient who had undergone corrective surgery at our institution three years previously for coarctation of the aorta. The continuous murmur in this instance was heard just lateral to the apex of the heart and could be occluded by local pressure. The fistula is considered to involve intercostal vessels at the site of rib resection. Furthermore, we have a child under observation at the Grady Memorial Hospital who underwent closure of an interventricular septal defect at another hospital. An arteriovenous fistula has appeared at the site of ligation of the internal mammary artery and simulates the lesion described by Glenn and Steinberg.[25] We have on four occasions attempted to produce pleural arteriovenous fistula at the time of surgery for pulmonary atresia. In two instances excellent long-term results have occurred following staged bilateral pleurectomy of this type. In two instances death ensued, in one of which a sudden embolic occlusion of a thumb-sized bronchial artery was the cause of fatality nine days after such surgery.

Roentgenologic Considerations

Earlier considerations in this regard were reported by us in 1954,[33] wherein discussion was confined to lesions of this type appearing within the thorax. On plain postero-anterior roentgenograms the aneurysms usually appear as a smooth, rounded structure. There may be significant change in

size and shape on inspiration-expiration film studies. We have been somewhat impressed with the tendency for even large venous aneurysms to be much less distinct in contour, or even seemingly absent, on studies in the lateral projection. Fluoroscopy has shown interesting pulsations which were paradoxical in timing relative to ventricular contraction in the pure fusiform lesion without associated anomalies. This is in contrast to the lack of pulsation seen in the simple saccular or diverticular type reported. We have been impressed with the absence of pulsations in aneurysmal dilatation of the superior vena cava due to total anomalous pulmonary venous drainage and in the *pseudo-aneurysm* due to combined cardiac failure and high inferior vena caval thrombosis. High, constant flow or pressure in these lesions seems to decrease the tendency to pulsations. Venous aneurysms of the superior mediastinum may enlarge when studied in the supine position; and such enlargement would be accentuated by the Mueller or Valsalva maneuvers.

Ultimately all venous aneurysms require venography for final diagnosis. The evidence at surgery of recent, walled-off perforation by a cardiac catheter in Patient H. S.[2] has led us to recommend that patients suspected of superior vena caval aneurysm should have the contrast media injection at a site not requiring entrance of the catheter into the lumen of the aneurysm. Total anomalous pulmonary venous drainage can be well studied by injecting through a catheter placed in the right atrium via the femoral vein. Our solitary instance of a residual opacity noted after angiography of an aneurysm in association with a neoplasm suggests the possible significance of *delayed emptying time.* If a catheter had been placed in this area, the residual opacity might have been interpreted as being the result of penetration of the vein wall.

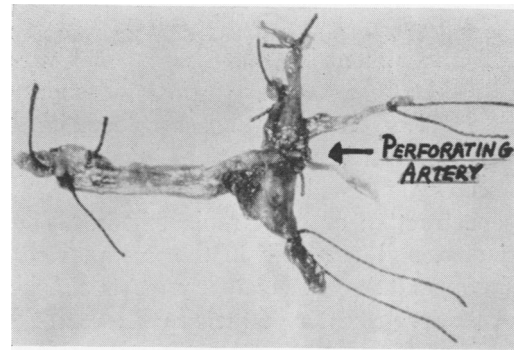

FIG. 13. Specimen excised from chest wall of D. S. Lesion presented after multiple thoracenteses performed for the management of a hemothorax, resulting from trauma to another area of chest.

Clinical Characteristics

The clinical features of these lesions vary according to the size, position and associated lesions which may be present. The pure venous aneurysms of the periauricular tissues[15] and the supraclavicular space[51] became distended with fatigue and effort as well as showing progressive enlargement. The pure fusiform superior vena caval aneurysm[2] was asymptomatic. The saccular or diverticular lesion of Lawrence[32] caused local pain. That of Robertson[47] caused dyspnea due to its size.

The secondary congenital lesions associated with causative anomalies basically owe their symptom complex to the underlying cardiovascular abnormality. In a similar way the acquired lesions relate their symptoms to the responsible disease state. The solitary *false* aneurysm was not symptomatic. Pseudo-aneurysms again relate their complaints according to the predisposing cause.

Arteriovenous aneurysms are the only lesions of this group which produce a continuous murmur, bruit, or thrill. Several excellent studies have portrayed in complete detail the symptoms, findings and physiologic effects of these lesions.[12, 29, 30, 45, 48, 60]

Therapy

The treatment of venous aneurysms varies markedly with their type. Whensoever the involved vein may be sacrificed, local excision is preferable. Lesions involving essential veins will require reinforcement, reconstruction or graft replacement.[50] The fundamental principle in the management of congenital venous aneurysms is to obtain exact definition of their cause. In those resulting from associated anomalies, correction of the causative abnormality controls the venous aneurysm. This basic fundamental underlies the therapy of the acquired lesions and the pseudo-aneurysms. Complete excision with or without graft replacement is curative for the various arteriovenous fistulae.

Summary and Conclusions

The authors have presented evidence from the literature and from their personal cases which appears to prove that venous aneurysms are a true entity. A suggested classification has been presented. The place for venous angiomata and neoplasm in such a classification is debatable. From a review of the literature as well as a review of 32 personal cases, we have attempted to present the radiologic criteria, clinical features, and recommended therapeutic measures. From this material we think we are justified in concluding that:

1. Aneurysmal disease of veins is less rare than previously suspected.

2. A useful classification has been presented whose major weakness involves the status of venous angiomata and neoplasms.

3. Catheterization can be a specific hazard in these lesions unless such studies have been so planned as to avoid actual insertion of the catheter with the lumen of the aneurysmal vein.

4. Roentgenographic and fluoroscopic study reveals varying findings according to the specific type of lesion being investigated. Venous aneurysms may appear vague or absent on lateral roentgenograms. Dilatation of intrathoracic veins can imitate mediastinal tumors. Final diagnosis requires venography.

5. The clinical pictures varies markedly according to type, location, and size of the lesion.

6. Treatment is excisional or reparative in pure primary, *false* venous aneurysms, and arteriovenous fistulae. For other types the major effort should be directed towards corrrection of the underlying patho-physiology; and, as such, the therapy may be basically medical or surgical.

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