Gangrene of the Extremities of Venous Origin
Review of the Literature with Case Reports

By Henry Haimovici, M.D.

Gangrene of the extremities of venous origin is reviewed; it is a rare but distinct clinicopathologic entity hitherto not widely known. A classification of the cases of thrombophlebitis complicated with gangrene is presented, based on the presence of the peripheral pulses: (1) cases with palpable arteries; (2) cases with nonpalpable but patent arteries. Patency of the entire arterial system of the involved extremity, as verified by anatomic findings, is necessary for correct diagnosis. Complete blockage of the venous system appears to be the initiating and main cause, angiospasm playing a secondary role. The gangrene usually remains superficial and limited. Hence, a conservative surgical attitude is emphasized.

GANGRENE of the extremities due to vascular disturbances can almost invariably be ascribed to either pure arterial occlusion or mixed arterial and venous occlusions. Gangrene as a result of venous obstruction alone is a third variety, but its possibility is not widely recognized. This paper will deal with this latter variety only.

The common clinical course of the occlusion of the main vein of an extremity is well known. The absence of anoxemia of the tissues in thrombophlebitis or phlebothrombosis is explained by the ease with which the collateral circulation is re-established owing to the abundant venous pathways. When, however, in addition to the main vein most, if not all, of the tributaries are also occluded, marked anoxemia of the tissues leading to gangrene may ensue from the sole obstruction of the venous tree.

Gangrene following extensive thrombophlebitis without arterial occlusion is, however, of rare occurrence. A perusal of the literature has uncovered only 27 such cases, with adequate clinical and pathologic information. Two of these were observed by the author.

HISTORICAL DATA

As early as 1593, Fabricius Hildanus1 seems to have been the first to recognize the possibility of gangrene of venous origin. Ever since, more particularly during the eighteenth and early nineteenth centuries, there has been much discussion on this subject as attested by many case reports. However, most of these reports were probably those of cases of gangrene due to mixed arteriovenous obliteration. Undoubtedly, the description of these cases suffered from the confusion which existed at that time regarding the whole subject of gangrene and its mechanism. In the latter part of the nineteenth century the clinical and pathologic descriptions of some of these cases seem to leave no doubt about their diagnosis.

Cruveilhier,2 in his “Traité d’Anatomie Pathologique Générale” (1862), pointed out the extreme rareness of this condition and emphasized that only very extensive venous thrombosis of both the main and collateral trunks can cause it.

In 1859, Huetel3 published the report of a case and gave an excellent account of the clinical and pathologic criteria of this type of gangrene. In 1894, Gaillard4 reported a similar case and reviewed the previously published observations. Most of the latter, however, upon careful analysis cannot be accepted as bona fide cases of gangrene due to venous occlusion alone. Three years later, Rey5 observed another case and again reviewed the subject. Thereafter it is surprising to note that for more than three decades the literature carried only one new observation published in 1903 by Fons.6

In 1924, in his book, Buerger7 merely mentioned that “complete obstruction of the chief veins of a part, without occlusion of the arteries, may also lead to moist gangrene, although this is of rare occurrence.” In a further chapter he simply emphasized the fact that “in the veins only extensive thrombosis over large territories is effective in producing gangrene of an extremity or portions of an extremity.”

Bergendal8 revived the subject in 1931 by reporting another case of “gangrene of the foot and lower part of leg in consequence of venous thrombosis.” In the following years, several clinical and pathologic reports were published by Bergeret, Guillaume, and Delarue in 1932; Wertheimer and Frieh in 19359; Gutzeit in 193610; Fontaine, Israel, and de Souza-Pereira in 193611; Salmon, Audier, Jouve, and Haimovici in 193812; Gregoire in 193813; Pringle in 193814.

In 1937, Fontaine and de Souza-Pereira14 reproduced experimentally this type of lesion in the dog by the division of all the veins at the root of the
<table>
<thead>
<tr>
<th>Case Author Date</th>
<th>Sex</th>
<th>Age</th>
<th>Etiologic bases</th>
<th>Site of the venous occlusion</th>
<th>Clinical aspect of the venous occlusion</th>
<th>Peripheral pulses</th>
<th>Extent of gangrene</th>
<th>Treatment</th>
<th>Result</th>
<th>Anatomic findings of the vessels of the affected extremity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1, Huetter, 1859</td>
<td>M</td>
<td>43</td>
<td>Unknown</td>
<td>RLE</td>
<td>a. Phl. a. dol. b. 11 days later phl. caer. dol.</td>
<td>Ant. tib. felt. Post. tib. n.r.</td>
<td>Foot &amp; lower leg</td>
<td>—</td>
<td>Died. Pulm. emb.?</td>
<td>Thrombosis of all veins distal to com. iliae</td>
</tr>
<tr>
<td>2, Guillard, 1894</td>
<td>F</td>
<td>27</td>
<td>Gastric ca.</td>
<td>LLE</td>
<td>Phl. caer. dol.</td>
<td>Only fem. felt</td>
<td>Foot</td>
<td>—</td>
<td>Died</td>
<td>Patency. Dissection distal to fem.</td>
</tr>
<tr>
<td>3, Rey, 1897</td>
<td>M</td>
<td>18</td>
<td>Pulmonary T.B.</td>
<td>RLE</td>
<td>a. Phl. a. dol. b. 21 days later phl. caer. dol.</td>
<td>Only fem. felt</td>
<td>Foot</td>
<td>—</td>
<td>Died</td>
<td>Patency. Dissection from vasa azygos to fem.</td>
</tr>
<tr>
<td>4, Pons, 1905</td>
<td>M</td>
<td>35</td>
<td>Post-traum. (fracture of femur)</td>
<td>RLE</td>
<td>Phl. caer. dol.</td>
<td>Only fem. felt</td>
<td>Foot &amp; lower of leg</td>
<td>Mid thigh amput. 9th day after accident</td>
<td>Recovered</td>
<td>Thrombosis of all veins of amput. specimen</td>
</tr>
<tr>
<td>5, Bergendal, 1931</td>
<td>M</td>
<td>33</td>
<td>Unknown</td>
<td>RLE</td>
<td>Phl. caer. dol.</td>
<td>Only fem. felt</td>
<td>Foot &amp; lower leg</td>
<td>Leg amput. (36th day)</td>
<td>Recovered</td>
<td>Thrombosis of all veins of amput. specimen</td>
</tr>
<tr>
<td>6, Bergeret, Guillaume &amp; Delarue, 1932</td>
<td>F</td>
<td>52</td>
<td>Postop. (appendectomy)</td>
<td>RLE</td>
<td>a. Phlebothrombosis b. 9 days later phl. caer. dol.</td>
<td>Absent pedal pulses. Absent oecil. readings</td>
<td>Foot &amp; 1/2 of leg</td>
<td>Thigh amput.</td>
<td>Died</td>
<td>Patency of anti. &amp; post. tib., &amp; personal. Active bleeding from proximal end of fem. during amput.</td>
</tr>
<tr>
<td>8, Wertheimer &amp; Frisch, (Case 2) 1935</td>
<td>M</td>
<td>44</td>
<td>Post i-v infusion</td>
<td>?UE</td>
<td>Cyanosis &amp; motor loss of hand</td>
<td>Absent radial pulse</td>
<td>Hand &amp; lower 1/2 of forearm</td>
<td>—</td>
<td>Died</td>
<td>Patency</td>
</tr>
<tr>
<td>9, Carcassonne &amp; Audier, 1935</td>
<td>F</td>
<td>57</td>
<td>Unknown</td>
<td>RUE</td>
<td>Phl. caer. dol.</td>
<td>All pulses felt</td>
<td>Second finger</td>
<td>Perivascular sympathectomy of brachial vessels</td>
<td>Recovered</td>
<td>Thrombosis of brachial veins</td>
</tr>
</tbody>
</table>

GANGRENE OF THE EXTREMITIES OF VENOUS ORIGIN
<table>
<thead>
<tr>
<th>No.</th>
<th>Fontaine, Israel, &amp; Sousa-Pereira, 1936</th>
<th>M</th>
<th>Unknown</th>
<th>RLE</th>
<th>Phl. caer. dol.</th>
<th>Only fem. felt but arteriogram showed patent arteries</th>
<th>Foot</th>
<th>Thigh amput.</th>
<th>Recovered</th>
<th>Thrombosis of all veins distal to com. iliac (operation &amp; dissection of amput. specimen)</th>
<th>Patent. Arteriogram &amp; dissecion</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Gutzeit, 1936</td>
<td>M Child (age?)</td>
<td>Post-traum.</td>
<td>LLE</td>
<td>Phl. caer. dol.</td>
<td>Only fem. felt (pulpit pulsatling at operation)</td>
<td>Foot &amp; lower 1/2 of leg</td>
<td>Amput.</td>
<td>Recovered</td>
<td>Thrombosis of all veins of amput. specimen</td>
<td>Patent</td>
</tr>
<tr>
<td>12</td>
<td>de Vernejoul, Audier, &amp; Picaud, 1936</td>
<td>M</td>
<td>Lung abscess</td>
<td>RLE</td>
<td>Phl. caer. dol.</td>
<td>Absent pedal pulses. Normal osc. read. only above lower 1/2 of leg</td>
<td>Foot</td>
<td>—</td>
<td>Died</td>
<td>Thrombosis of all veins distal to external iliac</td>
<td>Patent</td>
</tr>
<tr>
<td>14</td>
<td>Tilley (Case 1), 1938</td>
<td>F</td>
<td>Postpartum</td>
<td>RLE</td>
<td>a. Phl. a. dol. b. 3 days later phl. caer. dol.</td>
<td>n.r.</td>
<td>Foot &amp; leg</td>
<td>Thigh amput.</td>
<td>Recovered</td>
<td>Thrombosis of all veins</td>
<td>Patent</td>
</tr>
<tr>
<td>15</td>
<td>Tilley (Case 2), 1938</td>
<td>F</td>
<td>Postpartum</td>
<td>LLE</td>
<td>a. Phl. a. dol. b. 4 days later phl. caer. dol.</td>
<td>n. r.</td>
<td>Foot</td>
<td>Midleg amput.</td>
<td>Recovered</td>
<td>Thrombosis of all veins</td>
<td>Patent</td>
</tr>
<tr>
<td>17</td>
<td>Pringle (Case 1), 1938</td>
<td>M</td>
<td>Post-traum. (foot)</td>
<td>LLE</td>
<td>Cyanosis but no edema. Superficial veins thrombosed</td>
<td>Tib. pulses not felt</td>
<td>Foot &amp; leg</td>
<td>—</td>
<td>Died 48-hour after accident</td>
<td>Thrombosis of all veins distal to fem.</td>
<td>Patent</td>
</tr>
<tr>
<td>Case Author Date</td>
<td>Sex</td>
<td>Age</td>
<td>Etiologic bases</td>
<td>Site of thrombosis</td>
<td>Clinical aspect of the venous occlusion</td>
<td>Peripheral pulses</td>
<td>Extent of gangrene</td>
<td>Treatment</td>
<td>Result</td>
<td>Anatomic findings of the vessels of the affected extremity</td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>-----</td>
<td>-----</td>
<td>-----------------</td>
<td>-------------------</td>
<td>----------------------------------------</td>
<td>-------------------</td>
<td>------------------</td>
<td>-----------</td>
<td>--------</td>
<td>-------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>22, Gutermuth, 1942</td>
<td>M</td>
<td>45</td>
<td>Ca. of left lung</td>
<td>LUE</td>
<td>Wrist pulses felt</td>
<td>Hand</td>
<td>—</td>
<td>Died</td>
<td>Thrombosis of all veins of the entire extremity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23, Morales-Aparicio (Case 1), 1944</td>
<td>F</td>
<td>30</td>
<td>Post partum</td>
<td>LLE</td>
<td>a. Phl. a. dol. b. 20 days later phl. caer. dol.</td>
<td>Only fem. felt</td>
<td>Forefoot</td>
<td>Lirsevere amput. of foot</td>
<td>Recovered</td>
<td>No other anatomic findings except pulses felt after subsidence of edema</td>
<td></td>
</tr>
<tr>
<td>24, Morales-Aparicio (Case 2), 1944</td>
<td>F</td>
<td>36</td>
<td>Bronchopneumonia</td>
<td>LLE</td>
<td>a. Phl. a. dol. b. 24 hours later phl. caer. dol.</td>
<td>All pulses felt bilaterally. Normal oscil. readings</td>
<td>RLE: Foot &amp; lower 1 of leg LLE: All toes except 4th</td>
<td>Conservative</td>
<td>Recovered</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>25, Fontaine &amp; Forrest (Case 1), 1946</td>
<td>M</td>
<td>40</td>
<td>Postop.</td>
<td>LLE</td>
<td>RLE: 15 days post-op. phl. a. dol. A few days later: phl. caer. dol. of both lower extremities LLE: 7 days post-op. phl. a. dol.</td>
<td>All pulses felt bilaterally. Normal oscil. readings</td>
<td>RLE: Foot &amp; lower 1 of leg LLE: All toes except 4th</td>
<td>Conservative</td>
<td>Recovered</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>26, Fontaine &amp; Forrest (Case 2), 1946</td>
<td>M</td>
<td>8</td>
<td>Post-trauma.</td>
<td>LUE</td>
<td>Phl. caer. dol.</td>
<td>Only brachial felt. Absent oscil. readings in forearm</td>
<td>Hand &amp; lower 1 of forearm</td>
<td>Midarm amput.</td>
<td>Recovered</td>
<td>Thrombosis of all veins of amput. specimen</td>
<td></td>
</tr>
<tr>
<td>27, Haimovici &amp; Sufferness, 1948</td>
<td>M</td>
<td>60</td>
<td>Postop.</td>
<td>LLE</td>
<td>a. Phl. a. dol. b. 4 days later phl. caer. dol.</td>
<td>All pulses felt. Normal oscil. readings</td>
<td>All 5 toes &amp; 2 plantar areas</td>
<td>—</td>
<td>Spontaneous amput. of toes. Recovered</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

Thr. phl. = Thrombophlebitis.
Phl. a. dol. = Phlegmasia alba dolens.
Phl. caer. dol. = Phlegmasia caerulea dolens.
RLE = Right lower extremity.
LLE = Left lower extremity.
RUE = Right upper extremity.
LUE = Left upper extremity.
n.r. = Not recorded.
The lower extremities were involved in 79.3 per cent of the cases, while in the remaining 20.7 per cent the upper extremities were affected.

The clinical picture of a thrombophlebitis resulting in gangrene is characteristic. The thrombophlebitic process may go through three distinct phases: (1) phlegmasia alba dolens or blue thrombophlebitis, and (3) gangrene.

1. Phlegmasia Alba Dolens.—This phase is well known and needs no further description. It preceded the blue thrombophlebitis by a few days to a couple of weeks in 44 percent of the cases only. In the remainder of the cases, it failed to appear or may have been overlooked.

2. Phlegmasia Caerulea Dolens.—Phlegmasia caerulea dolens, or blue thrombophlebitis, was present as an initial manifestation in 56 per cent of the cases but was seen at a later stage in all instances irrespective of the onset. This phase of the thrombophlebitic process is usually typical in its manifestations.

Edema, considered as the pathognomonic sign of venous occlusion, may be absent at the inception in some cases. But soon thereafter, swelling usually becomes very marked and may extend beyond the groin or the axilla. The main feature is its rapidly progressive development. The skin of the affected limb is glossy, exceedingly tense, and the edematous portion is of a characteristic woody consistency. In a patient with swelling due to cardiac decompensation, phlebitic edema could be easily overlooked, but on closer examination the size of the affected limb is noted to be larger than that of the unaffected limb.

Cyanosis was the most characteristic sign. It appeared very early, developed rapidly, and extended to the entire extremity. Its maximum intensity was at the distal parts (toes, heel, fingers) where areas of bluish dark or red discolorations were noted.

Pain was a constant feature which accompanied cyanosis. Its onset may be sudden or progressive. In the former case it was often mistaken for an embolic pain. Irrespective of its type of onset, this pain was usually more intense than in the common form of thrombophlebitis. Sometimes in patients with
poor general condition, however, pain may go unnoticed. 

Local temperature was sometimes normal and it contrasted with the presence of cyanosis. However, in most cases, coolness of the distal parts of the limb was marked. Loss of motor power and hypesthesia, more frequently than anesthesia, were noted.

Arterial pulses were palpable in one-third of the cases (9 out of 27). In the remaining two-thirds, distal arteries, although patent, could not be felt or their patency was not recorded. It should be borne in mind that the exploration of the pulses in these cases is fraught with difficulties because of the usually excessive edema in the foot, around the ankle, and in the popliteal space. Besides the edema, which may account at least partly for the nonpalpable arteries, the presence of arteriospasm may be another reason for the absent pulses. Suffice it to mention at this point, that in these cases, the patency of all the arteries was verified by careful dissection and also sometimes by arteriography.

Oscillometric exploration of the distal arteries is fraught with the same difficulties as that of the palpation of the pulses. In addition, the application of the cuff is often painful and furthermore may be inadvisable because of possible danger resulting from the pressure on friable venous thrombi. In the cases in which oscillometric readings were taken, the information obtained paralleled that of the pulse findings.

Arteriography, when used, revealed the integrity of the arterial tree despite absent pulses and oscillometric readings. This means of exploration is not commonly used, but in such cases might be helpful in identifying the exact nature of the circulatory disturbances.

Multiple venous occlusions involving two or more extremities were not infrequent. In 2 out of the 27 cases, gangrene occurred in both lower extremities. In addition, in 8 out of the 27 cases, concomitant simple uncomplicated thrombophlebitis affecting other extremities was also noted. In one instance, the patient had a typical migrating thrombophlebitis the first manifestation of which was that of priapism. Then, in addition to the blue thrombophlebitis and gangrene of the right lower extremity, he developed venous occlusions of the left lower and upper extremities. Gutermuth’s case is a similar example of extensive involvement of the venous system (both lower extremities, left upper extremity, and the veins of the neck). The time of appearance of these multiple venous occlusions varied. They rarely appeared simultaneously. In most cases, they either preceded by a few days or weeks the blue thrombophlebitis or followed it from a few days to two months.

3. Gangrene.—The course of this type of blue thrombophlebitis was typical. Gangrene of the distal parts of the extremity appeared within four to eight days after the onset of the anoxic manifestations. It is well to remember, however, that blue thrombophlebitis does not necessarily lead to gangrene. A certain number of such cases, with transient cyanosis and ischemia occurring in the course of thrombophlebitis, have been reported. When gangrene occurs, as in the present group, it is in most instances of the “moist” variety, but “dry” necrosis is not incompatible with a venous occlusion.

Usually gangrene was limited to the distal parts of the extremities and, most important of all, it remained superficial. The necrotic and surrounding areas presented a characteristic bluish discoloration. Blebs were often noted and lymphangitis also developed. The line of demarcation at the early stage was not always very sharp. If infection accompanied the ischemic disorders of the limb, the latter offered the classical picture of wet gangrene.

Clinical Types

Thrombophlebitis complicated with gangrene can be differentiated into two main clinical types according to the presence of the peripheral pulses: (1) cases with palpable distal arteries and (2) cases with nonpalpable but patent arteries.

The following 2 cases, observed by the author, will best illustrate the two different clinical types.

Thrombophlebitis Complicated With Gangrene (Distal Arteries Palpable).* M. L., a 62 year old white
man, was admitted, on May 26, 1946, to the Alexian Brothers Hospital, Elizabeth, N. J., for neoplasm of the bladder. The patient had had hematuria for about two months prior to his admission. Except for a right inguinal hernia and some degree of chronic bronchitis, the patient's general condition was good.

At the time of his admission an electrocardiogram showed no abnormality and the blood pressure was 150/70. On May 27, 1946, a midline supraperine cystotomy was performed. The tumor was resected and five radon seeds of 1.5 mc. were inserted. The patient was ambulatory forty-eight hours after the operation.

On the fourth postoperative day the patient developed hiccoughs, for which he received carbon dioxide. Three days later congestion of the right lower lobe was diagnosed and confirmed by a roentgen-ray plate exposed on the following day. The patient was running a slight fever (101 F.) but there was no tenderness along the lower extremities. He was given 20,000 units of penicillin every three hours. On June 7 the left calf appeared infiltrated and tense. The diagnosis of thrombophlebitis became apparent. The patient was then given dicumarel (100 mg. the initial dose) and papaverine (1/2 grain administered subcutaneously every four hours), and his leg was elevated. During the following three days the calf was distinctly tense and the foot was warm. The patient's general condition was good.

On June 11 (fifteenth postoperative day) at 5 p.m., the patient experienced a sudden pain in the left leg and foot, more marked in the latter. Upon examination, the entire left lower extremity, from the hip down, was found to be swollen. It was also warm, with the exception of the foot and lower third of the leg which were cold and mottled. The circumference at the calf was 38.5 cm. as against 32 cm. on the right side. The dorsalis pedis and the posterior tibial arteries were felt and of good volume. Oscilometric readings were:

<table>
<thead>
<tr>
<th></th>
<th>Ankle</th>
<th>Calf</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>2.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Right</td>
<td>2.25</td>
<td>3.5</td>
</tr>
</tbody>
</table>

A paravertebral block with 1 per cent novocain was performed at the first, second, third, and fourth lumbar vertebrae with good immediate results. The severe pain, present before the block, subsided and the patient remained comfortable during the next twenty-four hours. Nevertheless, on the following day, all the toes appeared much more cyanotic than on the previous examination. Another paravertebral block with novocain was performed with immediate good results. Despite that, within the next twenty-four hours, the first and fifth toes appeared completely black, while the second, third, and fourth were black only distally. The areas of gangrene, involving the toes, were most marked on the plantar region. In addition, the ball of the foot and a circumscribed area near the heel also appeared gangrenous (fig. 1). Two days later, another block was performed with the same apparent favorable response. The patient was much more comfortable. A line of demarcation became evident four to five days after the onset of gangrene.

On June 19, the circumference at the calf was 33.5 cm. (5 cm. less than eight days previously). The edema of the entire left lower extremity was subsiding. The areas of gangrene on the plantar region (ball and heel of the foot) showed some fading, while those of the toes remained unchanged.

Fig. 1.—Dorsal and plantar regions of left foot. Gangrene involving the five toes, more extensive on the plantar than dorsal surface. Additional superficial necrotic lesions are present on the ball of the foot and near the heel.

On July 1 (three weeks after the onset of the acute ischemic episode) the patient was allowed to be out of bed, and six days later he was discharged from the hospital. His general condition was good. The edema of the lower extremity had completely subsided, and the gangrene of the left five toes was dry and well demarcated. No active surgery was contemplated, for it was assumed that spontaneous amputation would eventually occur. Indeed, the lesions of the ball and heel remained superficial and separated in a few months. As to the toes, after the demarcation between the normal and dead tissues began, the separation of the gangrenous distal phalanges took several months (from Oct. 9, 1946, to Feb. 3, 1947). The stumps (fig. 2) of the different toes were completely healed in May, 1947, eleven months after the onset of gangrene.

While at home, on Sept. 2, 1946, two months after his discharge from the hospital, the patient developed a typical iliofemoral thrombophlebitis of the right lower extremity. The entire limb was markedly swollen, hot, and painful. The patient was running a fever of 101.5 F. The next day a right paravertebral
block was performed and repeated twenty-four hours later. Within twenty-four hours after the last block, the temperature became normal and the pain subsided. The edema of the extremity subsided within a week. Unlike the thrombophlebitis of the left lower extremity, that of the right side was not accompanied by ischemic phenomena and followed a normal course.

When last seen in June, 1947, all pulses were present in both feet and the oscillometric readings were normal. It is to be emphasized that throughout the entire course of the disease the foot arteries remained patent and the oscillometric readings were normal.

The course.

When masia conception, the entire extremity subsided. After amputation and marked edema, the patient was hospitalized for cardiac decompensation. Among other manifestations, she presented edema of both lower extremities, bilateral pleural effusion, reduced diuresis, and allied symptoms.

While in the hospital she had a left iliofemoral thrombophlebitis characterized by pain along the medial aspect of the thigh, hydramniosis of the knee joint, and marked edema of the entire extremity.

Eight days later, during the night, she was seized suddenly with a violent pain in the left foot, and cyanosis up to the knee was noted. During the next few hours the pain became excruciating, the cyanosis more prominent, and edema more intense and painful to the touch. These troubles were most marked in the foot. In addition, coldness was also present in the foot and lower half of the leg. Loss of motor power was noted. The femoral artery just below Poupart's ligament could be palpated. None of the other pulses in this limb could be felt. Oscillometric readings were absent below the knee but were obtainable in the thigh. Fifteen to sixteen hours after the onset of this acute vascular occlusion syndrome, surgical exploration, under local anesthesia, of the femoral vessels was performed. The common femoral artery appeared normal; the superficial femoral was reduced in size and was pulsating feebly. However, no clot was present. Stripping of the adventitia over 4 inches released the spasm; the artery resumed its normal diameter and was pulsating freely. (It is noteworthy that as soon as the stripping of the adventitia was completed the patient stated that her pain disappeared). The femoral vein, in its entire length, was distended and filled with a firm clot.

After the operation a paravertebral block with novocain was performed. The leg became warmer but the foot remained cyanotic and cold. After a few days, the general condition grew worse, gangrene of the "wet" type became apparent, and an midleg amputation was performed. At the site of the amputation all the arteries were patent while the veins appeared thrombosed. An arteriogram of the amputated specimen showed a normal arterial tree in the leg and foot (fig. 3). Careful dissection of the vessels revealed that all the veins, large and small alike, were thrombosed. Microscopic examination showed a thrombophlebitic process at some levels, while the arteries appeared quite normal. This patient died a few weeks later from her cardiac decompensation.

* This case was reported in collaboration with Salmon, Audier, and Jouve.12

Thrombophlebitis Simulating Arterial Embolism and Resulting in Gangrene (Distal Arteries not Palpable but Patent).*—The patient, a woman, 50 years of age, with a previous history of several attacks of rheumatic heart disease, was hospitalized for cardiac decompensation. Among other manifestations, she presented edema of both lower extremities, bilateral pleural effusion, reduced diuresis, and allied symptoms.

FIG. 2.—The same foot as shown in figure 1. These photographs were taken eleven months after the onset of gangrene. Note the healed stumps of the five toes after spontaneous amputation.

In this case, the clinical course went through four distinct phases. First phase: At the inception, during the first few days, the thrombophlebitic process appeared as a typical phlebitis alba dolens. Second phase: On the fifth day the patient experienced a sudden pain and his left leg and foot became cold and cyanotic. The clinical picture took the aspect of a phlebitis caerulea dolens or blue thrombophlebitis. Third phase: Three days later, the toes and two areas of the plantar surface became gangrenous. Fourth phase: Regression and demarcation of the lesions began four to five days after the onset of gangrene. The spontaneous amputation of the five toes and the healing of the plantar lesions required several months. At present, there are no postthrombophlebitic sequelae and the walking capacity of the limb is normal. A similar instance in which there was only minimal loss of tissues (spontaneous amputation of the toes) was also reported by Fontaine and Forster20.
As in the preceding observation, the sequence of events in this case was typical: (1) phlegmasia alba dolens, (b) eight days later, phlegmasia caerulea dolens with acute ischemia and absent distal pulses simulating arterial embolism, (3) after a few days, wet gangrene.

This case is a typical example of a thrombophlebitis simulating at its onset a sudden arterial occlusion. A similar pseudoembolic onset was observed in several of the reviewed cases. In my observation the etiologic circumstances and the acute occlusive vascular syndrome made the diagnosis rather difficult. The pathologic findings during the operation and those of the amputated specimen were decisive in establishing the true nature of this case of gangrene.

**Diagnosis**

Gangrene of venous origin should be suspected whenever a thrombophlebitis is complicated with necrosis. Since this type of gangrene tends to remain superficial, an early diagnosis may be instrumental in avoiding an amputation. To make a diagnosis of gangrene of venous origin, it is necessary to establish the fact that there is thrombophlebitis without arterial occlusion. As already emphasized above, the type of venous occlusion most likely to induce gangrene is the blue thrombophlebitis. Its manifestations are typical. They consist of: (1) onset usually sudden; (2) cyanosis, appearing early, developing rapidly and extending to the entire extremity; (3) excessive edema of a woody consistency; (4) skin temperature, sometimes conserved, contrasting with the other signs of vascular deficit; (5) patent peripheral arteries. The latter are either palpable (one-third of the cases) or their patency is disclosed by an arteriogram or by dissection. When the distal pulses are felt the diagnosis is relatively easy. When the distal arteries are not palpable the diagnosis is difficult or not feasible on the clinical data alone. In the latter instance, other types of occlusive vascular disease should therefore be ruled out.

**Differential Diagnosis**

*Reflex Arteriospasm:* Iliofemoral thrombophlebitis associated with a reflex arteriospasm may simulate blue thrombophlebitis. The clinical course of the ischemic manifestations in reflex arteriospasm is variable. In some instances the symptoms of local blood-deficiency may disappear spontaneously after several hours and the diagnosis of phlegmasia
GANGRENE OF THE EXTREMITIES OF VENOUS ORIGIN

...literans, or arteriosclerosis obliterans) followed by gangrene may not be easy to distinguish from cases of gangrene due to venous occlusion alone. A previous history of intermittent claudication or a straightforward history of acute arterial occlusion preceding the venous occlusion may be in favor of the diagnosis of mixed arteriovenous occlusive syndrome. However, only careful dissection of the vessels of the affected extremity will reveal the decisive factor.

Among the reported observations of so-called puerperal gangrene, a good number appear to be due to mixed arterial and venous obstructions or to the use of ergot. The clinical and particularly the anatomic information accompanying most of such observations is not adequate. It is probable that some of these cases were gangrene of venous origin.

Summary

From this review it appears that in some cases the diagnosis may be difficult to establish on the basis of clinical information alone. It is therefore, important to re-emphasize that in these cases the anatomic findings of the vessels of the affected extremity as revealed by a careful post-mortem examination or dissection of the amputated specimen are paramount in establishing the true nature of the gangrene. It is obvious that the patency of the arteries in an amputated limb does not necessarily exclude the possibility of arterial origin of the gangrene because the arterial closure might have been proximal. For this reason, active bleeding from the proximal end of the main artery during the amputation should be an additional criterion in favor of the diagnosis of gangrene of venous origin. In the selection of the cases tabulated in this paper, all these criteria were met.

Pathologic Physiology

Data Supporting the Concept of Gangrene of Venous Origin

Clinicopathologic Data: From the foregoing description, it appears that the prominent features of this clinicopathologic entity consist of a syndrome of venous obstruction associated with gangrene and patency of the arterial tree.

alba dolens becomes apparent. In other instances the ischemic phenomena are checked either by the use of papaverine and paravertebral block or by periarterial stripping. In all these instances, following these procedures, normal color of the extremity and the distal pulses return. The diagnosis of reflex arteriospasm then becomes evident. Contrasting with the latter cases, in those of blue thrombophlebitis resulting eventually in gangrene, the ischemic manifestations cannot be reversed completely by the above procedures.

Acute Peripheral Circulatory Failure: Acute peripheral circulatory failure caused by acute thrombophlebitis is characterized by loss of pulsations in the arteries of all extremities and should be easy to differentiate if this rare clinical syndrome is kept in mind (Dennis and Morgan and co-workers). In these cases, the arterial pulsations disappear only transiently. Indeed, after proper treatment of the shock, pulses reappear and the diagnosis becomes evident.

Peripheral Arterial Embolism: Blue thrombophlebitis, particularly when associated with severe pain and marked arteriospasm, may closely simulate embolic occlusion. In order to differentiate one condition from the other, the following findings may be helpful. In embolic occlusion, the patient experiences acute pain with Blanching of the foot and leg and coldness of the extremity. In such cases, there is usually evidence of a heart condition, either auricular fibrillation or acute coronary thrombosis. In blue thrombophlebitis, the objective findings already mentioned above, that is, enlargement of the extremity, prominence of superficial veins, and the marked cyanosis, should help to distinguish the two conditions. At the inception of the thrombophlebitic process, when edema may be slight, the error might, however, be unavoidable. Indeed, in some cases the nature of the occlusive vascular syndrome has been established only during surgical exploration of the main vessels.

Mixed Arterial and Venous Occlusions: These may present the most difficult problem in the differential diagnosis. Cases of acute thrombophlebitis associated with arterial occlusion (simple arterial thrombosis, thrombangiitis obliterans, or arteriosclerosis obliterans) followed by gangrene may not be easy to distinguish from cases of gangrene due to venous occlusion alone. A previous history of intermittent claudication or a straightforward history of acute arterial occlusion preceding the venous occlusion may be in favor of the diagnosis of mixed arteriovenous occlusive syndrome. However, only careful dissection of the vessels of the affected extremity will reveal the decisive factor.

Among the reported observations of so-called puerperal gangrene, a good number appear to be due to mixed arterial and venous obstructions or to the use of ergot. The clinical and particularly the anatomic information accompanying most of such observations is not adequate. It is probable that some of these cases were gangrene of venous origin.
While the clinical data may be very suggestive, however, the main evidence in support of the concept of gangrene of venous origin is furnished by the pathologic findings. Indeed, careful dissection of the entire vascular system (table 1) disclosed an extensive occlusion of the whole venous tree and the absence of any organic lesion of the arterial tree.

Microscopic studies of the involved blood vessels have confirmed the gross findings and offered a rational basis for the understanding of some of the clinical manifestations. From a review of the observations reporting detailed histologic information concerning the blood vessels, it is possible to emphasize the following points of interest.

1. The histologic data on the venous system are based on the study of its various segments: femoral, popliteal, anterior and posterior tibial, and muscular and saphenous veins.

The lumen is entirely occupied by a blood clot which is not always homogeneous. At certain points the thrombus is of recent date while at other levels of the venous system the organization of the clot is at an advanced stage. In most cases, the large-sized veins present a recent thrombophlebitic process, with or without a beginning of organization, while the smaller-sized veins present a more advanced process.

The study of the wall of the occluded veins offers evidence of phlebitis of variable intensity. Endophlebitis and interstitial hemorrhagic infiltration are more or less associated and their degree varies with the case and the level of the venous tree. Of special interest to be mentioned is the diffuse inflammatory infiltration of the adventitia. The infiltrative process of this periphlebitis (venous adventicitis) may propagate and extend to the adjacent arterial adventitia. This histopathologic finding is the most probable explanation for the associated arterial spasm which accompanied the thrombophlebitis in some of the cases.

2. The arterial system is usually intact. The lumen is empty and the wall appears microscopically normal, with the exception, in some cases, of the adventitia. In a case reported by Salmon and his collaborators, a slight degree of adventicitis of the femoral, popliteal, and posterior tibial arteries was noted.

While these anatomic findings suggest a causal relationship between the venous occlusion and gangrene of the extremity, the possibility of gangrene due to venous occlusion alone is not readily accepted. In order to understand more clearly the pathogenesis of this type of gangrene, it is important to review briefly some facts regarding the anatomy of the venous system and the data concerning the experimental reproduction of such lesions.

Experimental Data: The anatomy of the collateral channels of the venous system is well known and need not be reviewed. It is, however, pertinent to mention briefly that in both the upper and lower extremities there are two sets of veins, superficial and deep, which anastomose frequently with each other. The ease with which the collateral circulation is re-established, owing to the abundant venous pathways, is one of the main reasons for the absence of anoxemia of the tissues or gangrene in thrombophlebitis.

Indeed, it has long been apparent, both in man and in animals, that simple ligations or even multiple obstructions of the main veins of the limbs induced no important obstacle to the venous circulation. Leriche and Jung14 and Fontaine and de Souza-Pereira15 studied in the dog the effects of resection and sclerosing of one or several segments of the venous system (saphenous, femoral, and vena cava). These obstacles to the venous circulation resulted only in a temporary edema not exceeding three to four weeks in duration. Only the division of all veins at the root of the hind leg of the dog was followed by gangrene of the limb of the wet type. From their experimental work, Fontaine and de Souza-Pereira concluded: "When the return of the circulation is completely blocked, the resulting stagnation of the venous blood is such that gangrene follows rapidly and the arteriograms are a proof of its pure venous origin."

It appears therefore, from these experimental data, that the completeness of the blockage of the venous return is an essential prerequisite for the occurrence of gangrene. These experimental facts shed interesting light on the ob-
servations made on the pathologic process in human beings.

Pathogenic Mechanism of Gangrene of Venous Origin

Three factors seem to be involved in the pathogenic mechanism of this type of gangrene: circulatory arrest, venous stasis, and vasomotor disturbances.

Role of the Circulatory Arrest: Gangrene as a result of arterial obstruction, regardless of its cause, is ascribed to the interference of blood distribution to the involved areas. In the case of gangrene as a result of venous obstruction alone, blood can apparently reach the tissues. Despite the patency of the arterial system, however, there is circulatory arrest caused by blockage of the return blood flow due to the extensive venous occlusion (fig. 4). In the final analysis it appears that the underlying mechanism of gangrene of venous origin is essentially an obstacle which prevents arterial oxygenated blood from reaching the tissues.

Role of the Venous Stasis: The venous stasis which accompanies the thrombophlebitic process seems to be an important contributing factor to the ischemia. Clinico-pathologic and experimental data on infarction of various viscera caused by venous occlusion (gut, spleen, kidney, brain, breast, and of striated muscle) seem to emphasize the role of venous stasis in their genesis. Ricker has shown that stasis and anoxemia can be directly responsible for necrosis with hemorrhage. From their work on muscular infarction due to venous occlusion, Wertheimer and associates hold a similar view. In a broader sense, visceral infarcts due solely to venous thrombosis are other illustrations of gangrene obeying the above mechanism.

Role of the Vasomotor Disturbances: On the basis of clinical and experimental evidence, Leriche and Kunlin and Ochsner and DeBakey believe that many of the clinical manifestations in venous thrombosis are due to vasoconstriction of the arterial and venous systems and that the vasoconstricting impulses originate in the thrombophlebitic segments.

It is not generally appreciated that the veins, like the arteries, respond to many types of stimuli. Malméjac and Haimovich have shown in the dog that stimulation of the lumbar sympathetic chain induces, among other responses, venous constriction as expressed in terms of blood stasis and increased volume of the limb.

The role of venous stasis in the causation of the circulatory disorders occurring in this type of thrombophlebitis, however, appears difficult to evaluate. It is possible to assume that in some cases it may only contribute to complete the already extensive venous occlusion.

In several of the observations reported in this series, arteriostenosis accompanied the thrombophlebitis as was definitely established during surgical exploration of the main arteries or as was disclosed by arteriography. In all these instances, the arteriostenosis was always preceded by the thrombophlebitic process. On the basis of the available information it is difficult to determine whether or not this spasm extended also to the collateral branches. As to its nature, the histologic evidence presented above points to its reflex origin. Indeed, the propagating adventicitis from the vein to the artery is in favor of this view. Since the presence of arteriostenosis accompanying the venous occlu-
sion, in some of these cases, is undeniable, the question arises as to what extent it was responsible for the gangrene. From the available data the arteriospasm appears to play rather a secondary role in its causation. Two sets of facts seem to lend support to this view. First, there are a number of cases of gangrene of venous origin without accompanying arteriospasm. Second, it has been shown experimentally that it can be reproduced by blocking only the entire venous system of the limb. It appears, therefore, that while angiospasm may be an aggravating factor, it is not the initiating cause in the mechanism of this gangrene.

**Prognosis**

The outcome in the 27 cases, recorded above, leaves much to be desired. Eleven out of the 27 patients died, their general condition being apparently responsible for this poor prognosis. Among the 16 remaining patients, only 3—having 4 involved extremities—had minor gangrene and recovered without surgery. In the other cases, major amputations were performed. It is to be hoped that modern treatment (antispasmodics and anticoagulants) and the knowledge that this type of gangrene usually remains superficial will improve the prognosis.

**Treatment**

Treatment will vary according to the stage of the clinical manifestations. In the presence of blue thrombophlebitis, the treatment should be directed against: (1) the angiospasm, (2) the propagation of the intravenous clotting, and (3) the underlying condition. There is no need to describe in any detail the different means for the management of these factors. However, it suffices to mention them briefly.

The control of the angiospasm can be obtained by the use of papaverine, Etamon, Priscoline, Dibenamine, or by paravertebral blocks of the sympathetic ganglia with procaine. The latter procedure can be repeated if necessary every twenty-four hours for as many consecutive days as is deemed advisable.24

It is generally agreed that anticoagulant therapy is important in the treatment of a sudden vascular occlusion. As indicated above, in the cases of thrombophlebitis followed by gangrene, the entire venous system of the limb was filled with thrombi. Since this event is mainly responsible for the gangrene, it is of the utmost importance to institute anticoagulant therapy (heparin, dicumarol) as soon as possible. The duration of its administration will, of course, be guided by the clinical manifestations.

In addition to the above measures, the treatment of the underlying etiologic condition should also be undertaken concurrently when indicated.

When gangrene sets in, and its venous origin is suspected or definitely established, it is important to bear in mind the fact that these lesions may remain superficial. Gangrene of the deeper tissues may be much less extensive than is apparent from observations of the skin.24, 29, 30 Amputation should therefore be delayed unless signs of toxemia are present. Antibiotics should prove beneficial under these circumstances. Demarcation of the necrotic areas and their spontaneous elimination may occur.24, 30 If lesions appear more extensive, a major amputation may become unavoidable.

**Summary and Conclusions**

A study of 27 cases of gangrene of the extremities following acute thrombophlebitis without arterial occlusion is presented. From the review of the literature it appears that this complication is rare.

The clinical picture of a thrombophlebitis resulting in gangrene is characteristic. Phlegmasia alba dolens, or milk leg, as an initial manifestation was seen in 44 per cent of the cases. Phlegmasia caerulea dolens or blue thrombophlebitis was present in 56 per cent of the cases at the onset but was seen at a later stage in nearly all instances. The latter syndrome is usually typical and consists of edema, often of a wooden consistency, marked and extensive cyanosis, and intense pain. Arterial pulses were palpable in one-third of the cases, while in the remaining two-thirds the distal arteries, although patent, could not be felt to pulsate. In these cases, the exploration of the pulses as well as the oscillometric readings is fraught with difficulties because of the usually excessive edema in the foot around the ankle.
and in the popliteal space. Multiple venous occlusions involving two or more extremities were not infrequent (10 out of 27 cases).

The course of this type of blue thrombophlebitis was typical, gangrene of the distal parts of the extremity occurring within four to eight days after the onset of the anoxic manifestations. In most instances this gangrene is of the "moist" variety, and it usually remains limited and superficial.

The cases can be divided into two main clinical types: (1) thrombophlebitis followed by gangrene with palpable distal arteries; (2) thrombophlebitis simulating arterial embolism and resulting in gangrene with non-palpable but patent distal arteries. To make a diagnosis of gangrene of venous origin it is necessary to establish the fact that there is thrombophlebitis without arterial occlusion. This condition should be differentiated from: reflex arteriospasm, acute peripheral circulatory failure caused by acute venous thrombosis, peripheral arterial embolism, and mixed arteriovenous occlusions. In some instances the diagnosis can be established only on the basis of the anatomic findings in the vessels of the affected extremity (postmortem examination or dissection of the amputated limb and active bleeding from the proximal end of the main artery during amputation).

From the clinicopathologic and experimental data it appears that the complete blockage of the venous return is the initiating and main cause of this type of gangrene. The angiospasm, when present, may be an aggravating factor, hence its role in the causation of the lesions appears to be secondary. Prognosis depends not only on the degree of the local factors (circulatory arrest, venostasis, and vaso-motor disturbances), but also on the underlying etiologic condition. The latter is mainly responsible for the poor prognosis as to life. Management by conservative surgical procedures should always be attempted except in the cases of extension of the lesions and the presence of signs of toxemia.

ADDENDUM

Since this article was submitted for publication, another cases of gangrene of venous origin came under my observation. The patient, a woman 42 years of age, was hospitalized at Montefiore Hospital for paraplegis due to intramedullary cord tumor. During her stay at the hospital she had several operations. On February 1, 1949, a few days after a laparotomy, both lower extremities became cyanotic and markedly edematous, and the right foot and lower third of the leg became gangrenous. Only the femoral pulse was felt. The patient died seven days later. On postmortem examination, an arteriogram and the dissection of the vascular system revealed patency of the entire arterial tree and thrombosis of the venous tree from the vena cava inferior down to the toes.

REFERENCES

5 ReyT: Sur les gangrènes d’origine veineuse, Paris, 1897. (Thesis in Medicine.)
14 Salmon, M., Audier, M., Jouve, A., and Hal-
MOVICI, H.: Sur un cas de gangrène du membre inférieur par thrombose veineuse. Sympathétoc- 
tomie péri-artérielle et veineuse (fémorales) in-
chir. de Marseille 12: 1, 1938.

(a) Grégoire, R.: La répercussion de l'infla-

mation des veines sur le système artériel col-

(b) ———: La phlébite bleue. Presse méd. 46:

1313, 1938.

PRINGLE, J. H.: Massive ischaemic gangrene 

with thrombosis of veins and patent arteries. Glas-

gow M. J. 129: 126, 1938.

Fontaine, R., and de Souza-Pereira, A.: Ob-

litérations et résections veineuses expéri-

mentales; contribution à l'étude de la circu-

lation collatérale veineuse. Rev. de chir., Paris 75:

161, 1937.

Wertheimer, P., Dechaume, J., and Freih, P.: 

Infarctus musculaire d'origine veineuse. Docu-

ments expérimentaux. Lyon chir. 34: 224, 1937.

(a) Audier, M., and Haimovici, H.: Les gan-

grènes des membres d'origine veineuse. Presse 

méd. 46: 1403, 1938.

(b) ———, and ———: Les phlébites des mem-

bres avec gangrène. Arch. d. mal. du coeur 58:

992, 1938.

Decoux, P., and Bastien, P.: Gangrène par 

spasme artériel au cours d'une phlébite. Ann. 
d'anat., path. 16: 353, 1939.

Favre, M., Rochet, Freih, and Godinot, C.: 

A propos d'un cas de gangrène cutanée étendue 

par lésions veineuses. Lyon chir. 36: 723, 1940.

Gutermuth, W.: Über venothrombotische Gan-


Tilley, J. H.: Gangrene of extremities in 


36: 157, 1938.

Morales Aparicio, L.: Flebitis azul. Rev. espa-

ño. de cir. traumat. y ortop. 1: 218, 1944.

Haimovici, H., and Suffness, G.: Gangrene of 

the extremities of venous origin; Case report. 


Bunn, W. H.: Case of thrombosis of inferior vena 

cava and extensive skin necrosis following scar-

let fever; recovery. Ohio State M. J. 29: 485, 

1933.

Meyer, O.: Latent phlebitis as cause of gangrene. 

München med. Wochensch. 87: 581, 1940.

Smith, B. C., and Quimby, E. H.: The use of 

radioactive sodium as a tracer in the study of 

peripheral vascular disease. Radiology 45: 335, 

1945.

Allen, E. V., Barker, N. W., and Hines, E. A. 

Jr.: Peripheral Vascular Diseases, Philadelphia, 

W. B. Saunders Company, 1946 p. 635.

Wright, I. S.: Vascular Diseases in Clinical Prac-


p. 383.

Fontaine, R., and Forster, E.: Deux nouvelles 

observations de gangrène des membres d'origine 


Laewen, A.: Arteriospasms bei akuter massiver 


61: 1681, 1934.

Audier, M.: Thromboses veineuses aiguës simul-

tant l'embolie artérielle des membres. Paris 

méd. 1: 384, 1936.

Edwards, E. A.: Nonarterial disorders simulating 

disease of the peripheral arteries. New England 


(a) Haimovici, H.: Les Embolies Artérielles des 


(b) ———: Les Occlusions Artérielles Aigu’s des 


Magendie, J., and Tinguad, R.: Phlébite à forme 

pseudo-embolique (phlébite bleue de Grégoire). 

Bordeaux chir. 3-4: 112, 1945.

Ochsner, A., and DeBakey, M.: Thrombophle-

bitis: The role of vasospasm in the production of 

the clinical manifestations. J.A.M.A. 114: 117, 

1940.

Nicole, R.: Arteriospasmus bei akuter Venen-

thrombose. Schweiz. med. Wochenschr. 65: 676, 

1935.

Pallin, G.: The differential diagnosis: arterial 


Scandinav. 65: 588, 1929.

Pampari, D.: Thrombophlebitis acuta degli arti e 

fenomeni pseudo-embolici, Il Policlinico. Sez. 

chir. 45: 470, 1938.

Sackenreiter, G.: Observations à propos de la 

"Phlegmatia coerulea dolens." Presse méd. 45:

575, 1940.

Cadenat, F.: Phlébite à phénomènes pseudo-

emboliques. Mém. Acad. de chir. 64: 436, 1938.

De Vernejoul, R., Audier, M., and Picaud, A.: 

Quoted by Audier, M.22

Dennis, C.: Disaster following femoral vein liga-

tion for thrombophlebitis: relief by fasciotomy; 

clinical case of renal impairment following crush 


Morgan, E. H., Allen, E. V., and MacCarty, 

C. S.: Acute peripheral circulatory failure caused 

by venous thrombosis. Proc. Staff Meet., Mayo 


Am. J. Obst. & Gynec. 27: 785, 1934.

Gutman, P. E.: Puerperal gangrene of extremities. 


O'Leary, J. L.: Peripheral gangrene following 

pregnancy. Am. J. Obst. & Gynec. 33: 662, 

1937.

McNally, F. P.: Puerperal gangrene of extremi-


Leriche, R., and Jung, A.: Recherches expéri-

mentales sur les oedèmes chirurgicaux des mem-

bres d'origine phlébitique. J. de chir. 37: 481, 

1931.
GANGRENE OF THE EXTREMITIES OF VENOUS ORIGIN


Ricker, G.: Quoted by Wertheimer and associates.17


Gangrene of the Extremities of Venous Origin: Review of the Literature with Case Reports

HENRY HAIMOVICI

Circulation. 1950;1:225-240
doi: 10.1161/01.CIR.1.2.225

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1950 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/1/2/225

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/