Acute Inferior Vena Cava Thrombosis*

Early Results of Heparin Therapy

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Study objective: To determine, during heparin therapy, the embolic risk associated with acute inferior vena cava thrombosis compared with noncaval thrombosis.

Design: Prospective controlled study.

Setting: University-affiliated general hospital.

Patients: Of 68 consecutive patients considered, 18 with caviographically proved inferior vena cava thrombosis and 45 with phlebography-proved noncaval proximal thrombosis met all other eligibility criteria and completed the study.

Interventions: All patients received adjusted continuous IV heparin therapy for ten days.

Measurements and Results: All 63 patients underwent systematic baseline and "day 10" perfusion lung scanning and phlebography. None suffered pulmonary embolism within the ten days, but 11/63 patients showed thrombus extension on day 10 phlebograms. Retrospectively, no significant difference could be found between the groups with and without extension.

Conclusions: (a) The early embolic risk associated with heparin-treated venous thromboses appears low and does not seem to depend on the location (caval or more peripheral) of venous clots. (b) Thrombus extension may occur in spite of apparently "adequate" anticoagulation with heparin.

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patients with acute thrombosis of the IVC often undergo early prophylactic IVC interruption in addition to anticoagulant therapy to prevent severe embolic events.14 The complication rate associated with IVC interruption is relatively well documented;4 however, the risk of embolization of IVC clots during anticoagulation alone is virtually unknown.

In our experience, approximately 15 percent of patients with proximal DVT have IVC thrombosis, either isolated or associated with more peripheral thrombi. Thus, it appears that a substantial number of patients with DVT might undergo a purely prophylactic additional procedure whose justifications are not based on adequate prospective data. Furthermore, IVC interruption is not a routine procedure in noncaval DVT8 and, in a previous study, we had found a low incidence of early recurrent pulmonary embolism during adequate anticoagulation in 50 patients with pulmonary embolism plus proximal noncaval DVT9.

Therefore, it appeared of interest to appreciate the embolic risk specifically associated with acute IVC thrombosis during heparin therapy. A prospective surveillance was given to 18 consecutive patients with IVC thrombosis who did not undergo early prophylactic IVC interruption and to a control group of 45 consecutive patients with proximal but noncaval DVT similarly treated and evaluated during the same period. Finally, systematic repeated phlebography was also organized for better evaluating the local evolution of heparin-treated proximal deep venous clots.

Material and Methods

Inclusion Criteria and Recruitment of Patients

During a 30-month period, all patients admitted to the department of pulmonary diseases were included in the study if the following criteria were met: proximal deep vein thrombosis (popliteal, femoral, iliac, or caval) proved by phlebography and/or caviography within the preceding 24 h; no venous thrombus above renal veins; no contraindication to anticoagulant treatment; no severe pulmonary embolism requiring thrombolytic agents or surgical embolectomy; no contraindication to repeated contrast or isotopic studies; no acute severe associated disease; age ≥18 years; and informed consent obtained. Sixty-eight patients met these criteria; IVC was involved in 18 of them.

The recruitment of patients is described in Figure 1. At our institution, all patients with confirmed pulmonary embolism underwent systematic bilateral lower limb phlebography within 36 h of diagnosis, even in the absence of signs or symptoms of DVT. Similarly, all patients with confirmed DVT underwent systematic baseline perfusion lung scanning, even in the absence of signs or symptoms of pulmonary embolism. The 68 patients included in the present study approximately represent one third of all patients with proved thromboembolism admitted to our department during the time of the study. The percentage of patients with IVC thrombosis (8 percent) was especially high in this study, because some were referred to our department from other hospitals because of their supposedly specific therapeutic problems, including eventual prophylactic IVC interruption.

Diagnostic Methods

Phlebography: The technique used in this study has been

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Manuscript received March 22; revision accepted July 1.

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Acute Inferior Vena Cava Thrombosis (Girard et al)
Previously described. Briefly, it was performed on recumbent patients by bolus injection of contrast medium in a dorsal vein of each foot, which allowed analysis of the deep venous system only. A Valvava maneuver or an abdominal compression together with the elevation of both legs was systematically performed before taking the last film to obtain better visualization of the iliac veins and vena cava. All films were anteroposterior views.

The diagnosis of acute thrombosis was based on the appearance of a constant intraluminal filling defect within an opacified vein. The total length of proximal deep clots (calf deep veins excluded) was measured as an index of the total volume of proximal thrombi.

Second phlebography was systematically performed after ten days of treatment ("day 10" phlebography).

Cavography was performed in all patients in whom the visualization of IVC was judged insufficient on phleograms, in case of suspected IVC thrombosis, or as a complementary investigation after pulmonary angiography.

It was performed at inclusion in a total of 27 patients, either by percutaneous catheterization of one or both femoral veins at the groin (five patients) or by retrograde positioning of a pulmonary angiography catheter in the IVC (22 patients).

Second cavography was performed in 22 patients (18 with IVC thrombosis plus four with insufficient visualization of the IVC) and, in those 18 patients with IVC thrombosis on initial cavography, the same techniques were used as in the first procedure so that data would be comparable. Caval clots were classified as "floating" when associated with a 5-cm or greater proximal segment of thrombus outlined on each side by contrast medium on anteroposterior and profile views. Injections for obtaining profile views of the IVC, however, were required only in those cases that fulfilled the criteria for "floating" clots on anteroposterior cavograms.

Angiography was performed at inclusion in all patients with clinically suspected pulmonary embolism or abnormal baseline perfusion lung scintigraphy, and it constituted the only diagnostic end point for pulmonary embolism (Fig 1). The contrast medium was injected in the right and left pulmonary arteries successively (selective opacification). The incidence for each injection was chosen on the basis of clinical or scintigraphic data. The pulmonary artery catheter was inserted in a basilic vein (90 percent of angiographies), a femoral vein (8 percent), or an internal jugular vein (2 percent). Eighty-eight percent of angiographies were "conventional," and the remaining 12 percent were obtained with the digital subtraction technique. In our experience, both techniques appear to have similar accuracy in the diagnosis of pulmonary embolism. The scoring system described by Miller et al was used to evaluate the degree of angiographic vascular obstruction.

Perfusion lung scintigraphy: Technetium 99m-labeled albumin microspheres were injected IV with the patient in the supine position. At least four views were available (anterior, posterior, and right and left lateral).

It was performed systematically in all patients at inclusion ("baseline" scans) and after the first ten days of heparin treatment ("day 10" scans). It was also performed in case of clinically suspected pulmonary embolism during these ten days. During the study, baseline perfusion lung scans were noted as "normal" or "abnormal." Abnormal scintigraphic perfusion should be confirmed by contemporary pulmonary angiography for diagnosing pulmonary embolism; subsequent perfusion lung scans were compared only with the immediately preceding one and noted as "presence" or "absence" of new defect or defects. "Presence" of new defect or defects required angiographic confirmation.

Interpretation and Outcome Events

All phlebographic, angiographic, and scintigraphic documents were interpreted at the time of performance by several trained chest physicians and radiologists. During the reviewing period at the end of the study, all of these documents were interpreted again by two independent pairs of chest physicians and radiologists without knowledge of clinical history and without knowledge of the order of performance of phlebographies and perfusion lung scans in an individual patient. Only minor differences were found between the two successive interpretations and between the pairs, and there was no diagnostic change in particular with respect to the diagnoses of (a) pulmonary embolism during anticoagulant treatment, and (b) proximal extension of thrombosis during anticoagulant treatment, which were the two outcome events in this study. Extension was diagnosed if the proximal growth of the thrombus was 1 cm or more. Pulmonary embolism, suspected on the appearance of new defect or defects on perfusion lung scan, should be confirmed by pulmonary angiography.

Protocol

Treatment protocol: As soon as the diagnosis of venous thromboembolic disease was suspected, a continuous IV infusion of sodium heparinate was started (500 IU/kg/day). Further doses were then adjusted on the basis of coagulation tests performed at least once a day for ten days. The aim was to increase the Howell time to 1.5 to 2 times the control value and the thrombin time to more than 2 min. In the event of inadequate anticoagulation, the Howell time was rechecked 4 to 6 h after any therapeutic change. Patients
Table 1 — Main Clinical Characteristics of Patients on Entry into the Study

<table>
<thead>
<tr>
<th>Feature</th>
<th>Caval Thrombosis</th>
<th>Non-caval Thrombosis</th>
<th>Total, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>18</td>
<td>45</td>
<td>63</td>
</tr>
<tr>
<td>Mean age, yr</td>
<td>54.2</td>
<td>53.7</td>
<td>(24-84)</td>
</tr>
<tr>
<td>Sex ratio (M/F)</td>
<td>7/11</td>
<td>29/17</td>
<td>35/28</td>
</tr>
<tr>
<td>Associated PE, no. (%)</td>
<td>6/33</td>
<td>23/51</td>
<td>29/46</td>
</tr>
<tr>
<td>Mean Miller index (range)</td>
<td>8.1/34</td>
<td>13.2/24</td>
<td></td>
</tr>
<tr>
<td>Asymptomatic PE</td>
<td>3/6</td>
<td>9/23</td>
<td></td>
</tr>
</tbody>
</table>

Risk factors†
- Previous DVT 5 12 17 (27)
- Recent surgery 3 7 10 (16)
- Immobilization <1 mo 4 5 9 (14)
- Recent delivery <1 mo 4 1 5
- Evolving neoplasms‡ 2 7 9 (14)
- Varicose veins 2 8 10 (16)
- Nephrotic Syndrome‡ 2 0
- Protein C deficiency 1/11 0/18
- Protein S deficiency 0/9 1/15
- AT III deficiency 1/15 2/23
- Polycythemia vera 0 1
- Femoral catheterization 2 2
- Recent long journey 1 3
- None 4 18 22 (35)

*PE = pulmonary embolism; DVT = deep venous thrombosis.†Each patient may have more than one of these conditions.
‡Either already known or discovered during hospital stay.

Tables 1

Patients were allowed to become ambulant, using venous elastic support, after two consecutive days of adequate anticoagulation had been completed.

Oral anticoagulation was started only after the performance of day 10 perfusion lung scintigraphy and phlebography, in an overlap between heparin and oral anticoagulants of four days or more.

Surveillance: Patients were given daily clinical examinations. Perfusion lung scanning was systematically performed on days 0 and 10 and phlebo- and/or cavography was also repeated on day 10 after entry into the study. Any clinically suspected venous thrombus extension or pulmonary embolism led to repeated phlebography or perfusion scanning respectively. Any "new defect" on perfusion lung scan led to pulmonary angiography. Any proved thrombus extension or pulmonary embolism led to the immediate assessment of contemporary pulmonary perfusion or lower limb venous circulation respectively.

Plasma concentrations of antithrombin III, protein C, and protein S were systematically measured in patients under the age of 50, in patients without an evident cause of venous thrombosis, and in patients with a familial history of venous thromboembolic disease. The techniques of measurement of protein C, and, subsequently, protein S concentrations became available at our institution during the time of the study, and this explains the different numbers of patients in whom these measurements were obtained (Table 1). Neoplasia as a causative agent was sought only in patients with biologic or clinical anomalies.

RESULTS

The delay between the initiation of IV heparin and the diagnosis of acute proximal DVT was 36 h or less in all patients.

Among the 68 patients who initially met the inclusion criteria, five suffered major hemorrhagic complications within the first six days of anticoagulation, resulting in the interruption of heparin treatment and immediate interruption of IVC. None of these five patients showed clinical evidence of pulmonary embolism or thrombus extension before the hemorrhagic accident occurred, and they did not significantly differ from the remaining 63 who completed the study and whose main clinical data on entry are presented in Table 1.

The data obtained on initial phlebocavograms are presented in Tables 2 and 3.

Day 10 phlebocavography and perfusion lung scintigraphy were obtained in all 63 patients eight to 12 days after entry to the study. None showed evidence of pulmonary embolism (Table 4). One patient with multiple emboli on baseline angiogram, however, showed angiography-confirmed "spurious scintigra-

Table 2 — Initial Phlebocavography: Upper Limit of Venous Clots

<table>
<thead>
<tr>
<th>Location of DVT</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Popliteal</td>
<td>6 (8)</td>
</tr>
<tr>
<td>Femoral</td>
<td>28 (41)</td>
</tr>
<tr>
<td>Iliac</td>
<td>15 (22)</td>
</tr>
<tr>
<td>Caval</td>
<td>18 (28)</td>
</tr>
<tr>
<td>Total</td>
<td>67*</td>
</tr>
</tbody>
</table>

*This total exceeds 63 because four patients had bilateral proximal DVT.

Table 3 — Caval Clots: Initial Clinical and Phlebocavographic Data

<table>
<thead>
<tr>
<th>Total no. of patients</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic venous thrombosis</td>
<td>11</td>
</tr>
<tr>
<td>Totally occlusive caval thromboses</td>
<td>0</td>
</tr>
<tr>
<td>C + I + F + P* thromboses</td>
<td>9</td>
</tr>
<tr>
<td>C + I + F thromboses</td>
<td>5</td>
</tr>
<tr>
<td>C + I thromboses</td>
<td>2</td>
</tr>
<tr>
<td>Isolated caval thrombus</td>
<td>1</td>
</tr>
<tr>
<td>&quot;Floating&quot; caval thrombus</td>
<td>7†</td>
</tr>
</tbody>
</table>

* = caval; I = iliac; F = femoral; P = popliteal.
†Identification by biplanar cavography performed in 12 patients.

Table 4 — Main Results After 10 Days of IV Heparin Therapy

<table>
<thead>
<tr>
<th>Patients with Caval Thrombosis, No. (%)</th>
<th>Other Patients, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary embolism</td>
<td>0</td>
</tr>
<tr>
<td>Thrombus extension</td>
<td>5/18 (28)</td>
</tr>
</tbody>
</table>

*NS, not significant (χ² = 1.84).
Clinical symptoms (pleuritic chest pain or hemoptysis or both) within the first six days of therapy, leading to repeated perfusion lung scintigraphy: none of these

Table 5—Potential Risk Factors for Thrombus Extension*

<table>
<thead>
<tr>
<th>Factor</th>
<th>Patients with Thrombus Extension, No. (%)</th>
<th>Patients without Thrombus Extension, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no.</td>
<td>11</td>
<td>52</td>
</tr>
<tr>
<td>Evolving neoplasm</td>
<td>2 (18)</td>
<td>7 (13)</td>
</tr>
<tr>
<td>AT III deficiency</td>
<td>2/5</td>
<td>1/30</td>
</tr>
<tr>
<td>Protein C deficiency</td>
<td>0/5</td>
<td>1/24</td>
</tr>
<tr>
<td>Protein S deficiency</td>
<td>0/4</td>
<td>1/20</td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Polycthemia vera</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Persistent immobilization</td>
<td>5 (45)</td>
<td>NS</td>
</tr>
<tr>
<td>Associated PE on entry</td>
<td>6 (54)</td>
<td>13 (25)</td>
</tr>
<tr>
<td>into the study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Length of proximal thrombus, cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(range)</td>
<td>20.7 ± 18†</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>(3-65)</td>
<td></td>
</tr>
<tr>
<td>Mean daily dose of heparin, IU</td>
<td>35,300 ± 750†</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>(2-78)</td>
<td></td>
</tr>
<tr>
<td>Mean percentage of adequate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>coagulation tests</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First 3 days, %</td>
<td>62</td>
<td>62</td>
</tr>
<tr>
<td>First 10 days, %</td>
<td>77</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Each patient may have more than one of these conditions.
†Mean ± SD.
scans revealed the appearance of new defect or defects, and this was taken to exclude the diagnosis of appreciable recurrent pulmonary embolism.

Proximal extension of thrombosis was diagnosed in a total of 11 patients (Table 4 and Fig 2 to 4). An IVC interruption was performed in only nine of them, because, during the study, the slight extension (<2 cm) of thrombosis in two patients had been attributed to inadequate anticoagulation at the onset of therapy. In eight of those nine patients with IVC interruption, a Greenfield filter was inserted, and one patient (see Fig 2) underwent a venous thrombectomy prior to the placement of a Adams-De Weese clip. The extension was of 5 cm or less in all patients but one.

Potential risk factors for thrombus extension were compared between the groups with (11 patients) and without extension (32 patients). This comparison is presented in Table 5 and does not allow to find any predictive factor for thrombus extension during heparin treatment. Among three patients in whom antithrombin III deficiency was diagnosed, however, two showed an extension. In these three patients, the diagnosis of deficiency was unknown on entry into the study, was suspected on the basis of a first measurement during heparin therapy, and was confirmed during subsequent oral anticoagulation.

Discussion
The specific therapeutic problems of acute IVC thrombosis have been discussed, to our knowledge, in only one recent study. This was a retrospective work, which showed that among 25 patients those 17 without systematic early IVC interruption did rather well. This study led us to consider the therapeutic problem of acute IVC thromboses in a prospective and objective way, because considerable subjectivity exists in this field, especially regarding the so-called free-floating venous clots.

It would have been of interest to undertake a randomized trial comparing the efficiency and the safety of anticoagulation with and without systematic complementary IVC interruption. In a previous work including 50 patients with proximal DVT plus pulmo-
nary embolism, however, we found an incidence of recurrent thromboembolic events (4 percent within the first 15 days of therapy) that precluded such a comparative study, because it would have required an excessive number of patients. To demonstrate a significant reduction in recurrence rate, eg, from 5 percent in the group treated only by anticoagulation to 1 percent in the group with an additional procedure, observations of at least 716 proved cases of acute proximal DVT would be necessary. Therefore, we chose to compare a group in which IVC interruption is not a routine procedure because of an expected low embolic risk (ie, patients with proximal DVT but without caval involvement), with a group in which IVC interruption is usually recommended on the basis of a supposed high risk of severe embolism. Consequently, however, our results provided data only concerning the feasibility of treating IVC clots without IVC interruption and should not lead to therapeutic recommendations.

The absence of systematic IVC interruption in patients with acute, eventually free-floating IVC thrombosis raised some ethical questions, since pulmonary embolism, a potentially fatal complication, could be prevented by a simple procedure, IVC interruption. These questions were answered by using five main arguments. (1) The above-mentioned retrospective study on acute IVC thrombosis had found rather encouraging results. (2) Our previous work on patients with proximal DVT plus pulmonary embolism had found a low embolic risk, with only one fatal recurrence related to both inadequate anticoagulation and severe initial pulmonary vascular obstruction. (3) A close surveillance of coagulation tests seems to improve both the safety and the efficiency of anticoagulant therapy. (4) The complication rate of IVC interruption is appreciable and has been recently evaluated as high as 17.4 percent with the Greenfield filter, with a reported operative mortality ranging from 0 to 5 percent. (5) Because IVC interruption does not replace the anticoagulant treatment, the respective risks associated with both therapeutic procedures are added. Our therapeutic attitude thus appeared ethically defensible. Finally, from a financial point of view, the reservation of IVC interruption to strict and limited indications might also contribute to diminish the cost of venous thromboembolic disease.

In 18 selected patients under close surveillance, we...
found that the embolic risk of IVC clots within the first ten days of anticoagulation was nil. In this regard, our patients with IVC thrombosis appeared similar to those 45 with more peripheral thrombosis.

The embolic risk associated with proximal DVT prior to anticoagulant treatment is high. The systematic performance of ventilation-perfusion lung scanning in such patients demonstrates that about 50 percent of them have "high probability" perfusion defects. Although we used a slightly different diagnostic approach, we found similar results in this study (Table 1). During anticoagulant therapy, however, the embolic risk associated with proximal DVT appears poorly determined. In the literature, this risk varies from 0 to 28 percent within the first seven to 15 days of treatment. This variation probably has several explanations, some of which, such as the method of heparin administration and the adequacy of anticoagulation, have been investigated in prospective comparative studies. Other explanations might include the absence, in most of the above-mentioned studies, of a systematic contemporary assessment of baseline lung perfusion and lower limb deep vein permeability, because this results in poorly documented diagnoses of "recurrent thromboembolic events." In our previous prospective work, we used systematic scintigraphic surveillance, with pulmonary angiography as the sole diagnostic end point for both initial pulmonary embolism and eventual recurrence. This careful diagnostic approach for evaluating the embolic risk was also employed in the present work, and we found similar results. After the initiation of this study, other authors have published prospective studies in which less than 4 percent of patients with DVT were reported to suffer pulmonary embolism during IV heparin treatment, without any fatal embolic event. These results also are consistent with ours, and confirm the low incidence of pulmonary embolism in patients with proximal DVT treated by closely monitored IV heparin.

The aspect of venous clots on phlebograms has been considered a prognostic factor for embolization. Jones et al recommend IVC interruption for free-floating iliofemoral clots because 60 percent are said to embolize in spite of anticoagulation. This rate of 60 percent, however, represents three of five patients extracted from a retrospective study, and our results in patients with free-floating caval thrombi, as well as those of other investigators who do not modify their therapeutic decisions on the basis of the anatomic results of phlebography, strongly support different conclusions. According to our present data, the phlebographic aspect of venous clots seems of little if any prognostic value, regarding their embolic risk during anticoagulant treatment. This hypothesis, however, should be confirmed in further prospective studies including a greater number of patients.

Extension of venous clots was found in 5/18 and 6/45 patients with IVC and noncaval proximal thrombosis, respectively. Others have demonstrated the occurrence of thrombus extension in spite of "adequate" anticoagulation, and such discrepancies between the anticoagulant and the antithrombotic effects of heparin have been described already and commented on. We were unable to find any predictive factor for this extension: location and aspect of thrombi, doses of heparin, percentage of adequate coagulation tests, an index of total volume of proximal thrombi and associated pulmonary embolism were not significantly different between the groups with and without extension (Table 5). Individually, however, extension might have been suspected in a few patients: antithrombin III deficiency, rapidly evolving neoplasms, and nephrotic syndrome are clinical situations associated with a recognized altered efficiency of heparin. Further, more patients might have revealed statistically significant differences. At present, our findings only suggest the usefulness of repeated phlebocavography for detecting thrombus extension, because the incidence of this complication appears appreciable.

The relationship between thrombus extension and an eventually increased embolic risk remains speculative. Pollak et al suggested such a relationship on the basis of two cases in which thrombus extension was proved before recurrent pulmonary embolism. In fact, the indication to IVC interruption in our patients with thrombus extension was both the hypothesis increased risk of pulmonary embolism and the proved failure of anticoagulant therapy, i.e., thrombus generation in spite of "adequate" doses of heparin.

Oral anticoagulation therapy is now routinely started after one to seven days of IV heparin. In this study, we have introduced oral anticoagulation only after ten days of IV heparin therapy, and the effect of an earlier introduction of oral anticoagulants on the phlebographic evolution remains to be determined. In terms of recurrent thromboembolic events, however, short (one day) and long (seven days) anticoagulation by IV heparin before the start of oral anticoagulants showed similar results in a recent careful, prospective, randomized study.

Finally, our study provides data that confirm the poor diagnostic value of clinical symptoms for diagnosing pulmonary embolism: 42/63 patients had no signs of pulmonary embolism on entry into the study, whereas 12 of them (25 percent) actually had angiography-documented pulmonary emboli (Table 1). Accordingly, none of those five patients with clinically suspected recurrent embolism during the prospective surveillance was found to have new scintigraphic defects. In our view, this further supports the recom-
mendation for a systematic contemporary baseline assessment of both the deep venous system and the pulmonary perfusion in patients with acute venous thromboembolism.

Our results do not allow any recommendation on the use of prophylactic IVC interruption in the treatment of patients with acute IVC thrombosis, and systematic IVC interruption remains acceptable and perhaps justified in these patients, especially when a close clinical and biologic surveillance cannot be performed. Our study, however, suggests that the location of venous clots should not constitute per se an indication to a relatively expensive and potentially dangerous additional procedure, namely: IVC interruption. Nevertheless, "close surveillance" partly consists of procedures enabling the detection of thrombus extension during therapy (repeated phlebocavography). The development of less invasive methods for detecting thrombus extension might considerably improve our therapeutic approach to this frequent clinical problem, and the effect of an earlier introduction of oral anticoagulants perhaps should be investigated.

ACKNOWLEDGMENTS: We are indebted to Drs. S. Salmeron, F. Brenot, C. Philippoteau, P. Herve, T. Chinet, and P. Duroux for their efficacious support during and after the study, and to M.F. Bouissacu for typing the manuscript.

REFERENCES


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