Edema Fluid and Coagulation Changes During Fulminant Pulmonary Edema*

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Edema fluid and a coagulation profile were obtained in 45 patients (17 to 87 years) during fulminant pulmonary edema. Left ventricular failure and/or volume over-load accounted for edema in 18 patients. In another 27 patients, edema developed in association with other features that typify the adult respiratory distress syndrome (ARDS). In the ARDS group, multiple disorders were implicated in the genesis of edema, including shock, bacteremia, drug overdose, and aspiration. Assessment of edema fluid and coagulation measurements is useful to classify and to determine the severity of the edema process. ARDS is characterized by permeability pulmonary edema that usually stems from a combination of multisystemic disorders. Permeability pulmonary edema and coagulation changes appear to be interrelated. However, it is not clear the extent to which the coagulation disturbances are a cause or a result of the alterations in the alveolar-capillary membrane.

The adult respiratory distress syndrome (ARDS) has been characterized as acute pulmonary dysfunction that occurs with a variety of life-threatening injuries or illnesses; including perfusion failure (shock) especially in association with hemorrhage, trauma, or systemic bacterial infection, intoxication with heroin or other drugs, aspiration of gastric contents, and pulmonary embolism. A marked reduction in lung compliance and impairment of oxygen exchange together with clinical and roentgenographic signs of pulmonary edema are typically observed. It has been postulated that the pulmonary edema is due to a marked increase in pulmonary capillary permeability to protein (permeability edema). Therefore, the protein content of edema fluid is likely to be similar to plasma. This concept is supported by an electronmicroscopic study of the alveolar capillary membrane of patients who died with this syndrome in which substantial damage to the endothelial and epithelial barriers was demonstrated. However, the mechanisms that account for this damage have not been clearly defined. Several studies have pointed to changes in the coagulation system, microemboli, neurohumoral factors, as well as chemical injury of the airway.

Pulmonary edema may also stem from the effects of unbalancing the hydrostatic and oncotic pressures across the pulmonary microvascular membrane. This form of edema is termed hemodynamic or high pressure edema and is most commonly due to left ventricular failure and/or volume overload. During hemodynamic pulmonary edema, the alveolar capillary membrane continues to be an effective barrier to protein. Therefore, in this form of edema, the protein content of edema fluid that accumulates in the interstitium and subsequently floods the airspaces is likely to be approximately 50 percent of plasma. In addition, the edema fluid usually contains red blood cells that may be due to microvascular rupture and focal hemorrhage. It is possible, therefore, that in fulminant hemodynamic edema, the focal microvascular damage may lead to activation of the coagulation cascade by exposure of collagen to circulating clotting factors and platelets. However, the extent to which these potential coagulation changes are found in association with severe high pressure edema has not been studied.

**MATERIALS AND METHODS**

We prospectively studied 45 patients ranging in age from 17 to 87 years with florid pulmonary edema who were admitted to the USC Center for the Critically Ill-Hollywood Presbyterian Medical Center from August 1974 to August 1979. This population was selected from more than 300 patients with pulmonary edema in our service during that interval. We identified ARDS in 27 patients as acute respiratory failure that developed in a critically-ill patient with multisystem illness and/or injury in association with a marked decrease in oxygenation, a reduction in lung compliance, and clinical and roentgenographic signs of pulmonary edema. High pressure or hemodynamic pulmonary edema that was
related to left ventricular failure due to unequivocal clinical and historical signs of ischemic heart disease, hypertensive crisis, alcoholic, and cardiomyopathy and/or volume overload was documented in another group of 18 patients. Aside from cardiopulmonary arrest (CPR) and cardiogenic shock, we could identify no other clinical condition accounting for edema in this group.

The trachea of each patient was intubated, and all patients received mechanical ventilation at the time of the study. Positive end-expiratory pressure (PEEP) to levels of up to 20 cm H2O were employed in 24 patients during the study. Effective static or dynamic lung compliance was determined. Arterial blood gas levels were measured; we used the arterial to alveolar oxygen tension ratio (PaO2/PAO2; oxygenation index) as a measure of ventilation to perfusion mismatch or shunting. The normal oxygenation index is 0.77 to 0.88.18

A pulmonary artery catheter was inserted in 32 patients at the time of study for clinical management. We used the pulmonary artery wedge (PAW) pressure that was measured at end-expiration as an index of pulmonary microvascular pressure. We did not correct for the possible effects of PEEP on PAW.

Samples of ≥3 ml of edema fluid (EF) were suctioned from the endotracheal tube with a soft plastic catheter and collected in a Luca’s trap. We selected this volume to ensure that we sampled during alveolar flooding. Sampling was performed within ten minutes of the measurement of PAW. If EF was visibly contaminated with mucus or other debris, the sample was rejected. The EF was yellow and occasionally contained small fibrin clots in patients with permeability edema; whereas EF was pink in cases of hemodynamic edema (hematocrit value 0.5 to 2.0 percent). A sample of blood was obtained simultaneously. We measured the colloid osmotic pressure of EF and plasma (P).19 In some instances, we were able to obtain multiple samples of EF and P over time.

The EF/P COP ratio of the first set of samples was used to assess the pulmonary microvascular permeability to protein.5,4,15

We defined a clinically significant coagulation defect if two or more of the following were obtained: the prothrombin time was >3 sec from control;26 the activated partial thromboplastin time was >40 sec;21 fibrin split products of >1:5 (≥ 10g/mL) were identified by the latex fixation method;25 the platelet count was <100,000/cu mm; and/or there was a decrease of fibrinogen to < 200 mg/100 ml. The coagulation profile was obtained within four hours of EF sampling. Two groups of patients were identified; those with a coagulation defect (C) and those who did not present with these coagulation criteria (NC).

Data are reported as the mean ± SD. The Student’s t-test for unpaired data, the χ2 test, and linear regression by the least squares method were used for statistical calculations. Statistical significance was selected at the P = 0.05 level.

RESULTS

The diagnoses, EF/P COP ratios, oxygenation index, pulmonary compliance, survival, and the coagulation measurements are shown in Table 1. Patients are ranked by the EF/P COP ratio and are classified as C and NC and as ARDS or hemodynamic edema. All patients but five with ARDS met our criteria for coagulopathy. In contrast, in none of the 18 patients with clinical features of hemodynamic (high pressure) edema was a coagulopathy identified. The PAW at the time of edema fluid sampling for the ARDS group was less (X² = 14.5 ± 6.7 mm Hg) than that observed for the hemodynamic group (X² = 26.4 ± 11.4 mm Hg) (P = NS), although there was considerable variation in the level of PAW. As anticipated, the oxygenation index of the patients with ARDS was substantially less than that of patients with hemodynamic edema (X² = 0.16 ± 0.1 vs 0.4 ± 0.2; P < 0.001). For the 45 patients, there was a correlation between the oxygenation index and the EF/P COP ratio (Fig 1); oxygenation was more severely affected when the EF/P ratio was high.

For the 27 patients with ARDS, we corroborated an increase in pulmonary vascular permeability to protein by a high EF/P COP ratio (X² = 0.87 ± 0.2; range = 0.54 – 1.32). This included one patient with freshwater near-drowning (No. 26) in whom the initial ratio was 0.54. However, on subsequent measurements of EF and P, the ratio increased to 1.1, and florid edema with EF production persisted for hours. We therefore classified this patient among those with permeability pulmonary edema. For the 18 patients with clinical findings of hemodynamic edema, the mean EF/P COP ratio was low (X² = 0.54 ± 0.13; range 0.28 – 0.69) (P < 0.001).

Multiple systemic and/or pulmonary factors were implicated in the genesis of permeability pulmonary edema for patients with ARDS. Furthermore, several factors were identified in each patient. Aspira-
<table>
<thead>
<tr>
<th>No.</th>
<th>Age/sex</th>
<th>Diagnoses</th>
<th>EF/P COP (mm Hg)</th>
<th>PaO2/PaO2 (Index)</th>
<th>PAW (mm Hg)</th>
<th>S/D</th>
<th>Protime (sec)</th>
<th>APTT (sec)</th>
<th>Flign, mg/100 ml</th>
<th>PBP dil</th>
<th>WBC x10^9/mm</th>
<th>Platelet x10^9/mm</th>
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<tr>
<td>1</td>
<td>64F</td>
<td>Aspiration, GI bleed, shock</td>
<td>27.5/19.5 1.32</td>
<td>0.26 27</td>
<td>0</td>
<td>D</td>
<td>12/11</td>
<td>28/30</td>
<td>270</td>
<td>1:5</td>
<td>8.7</td>
<td>28</td>
</tr>
<tr>
<td>2</td>
<td>63F</td>
<td>Hypovolemia, GI bleed, renal failure, rheumatoid, chronic steroid use</td>
<td>25.1/19.1 1.31</td>
<td>0.15 17.5</td>
<td>9</td>
<td>D</td>
<td>17/11</td>
<td>30/20</td>
<td>150</td>
<td>(--)</td>
<td>18.4</td>
<td>150</td>
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<tr>
<td>3</td>
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<td>Heroin overdose, volume overload</td>
<td>10.4/8.6 1.21</td>
<td>0.10 27.5</td>
<td>30</td>
<td>D</td>
<td>17/11</td>
<td>73/33</td>
<td>62</td>
<td>1:5</td>
<td>10.3</td>
<td>120</td>
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<td>87M</td>
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<td>21.1/20.8 1.01</td>
<td>0.17 22</td>
<td>14</td>
<td>D</td>
<td>14/11</td>
<td>40/24</td>
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<td>Aspiration, (7) palm embolus, IHD</td>
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<td>15</td>
<td>D</td>
<td>17/11</td>
<td>50/25</td>
<td>60</td>
<td>1:20</td>
<td>3</td>
<td>90</td>
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<td>6</td>
<td>69F</td>
<td>Bacteroides bacteremia, shock, mesenteric ischemia</td>
<td>16.3/18.7 0.98</td>
<td>0.24 20</td>
<td>18</td>
<td>D</td>
<td>11/11</td>
<td>34/24</td>
<td>400</td>
<td>1:10</td>
<td>12.6</td>
<td>48</td>
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<td>20</td>
<td>D</td>
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<td>0.27 20</td>
<td>20</td>
<td>D</td>
<td>14/11</td>
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<td>55M</td>
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<td>14.1/15.5 0.91</td>
<td>0.10 23</td>
<td>9</td>
<td>D</td>
<td>12/11</td>
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<td>Pulmonary embolism, femoral artery thrombosis, shock, CPR</td>
<td>9.1/10 0.91</td>
<td>0.18 30</td>
<td>13</td>
<td>D</td>
<td>17/11</td>
<td>42/25</td>
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<td>1:40</td>
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<td>11</td>
<td>77F</td>
<td>E Coli bacteremia, shock, pericardial, renal failure</td>
<td>18.5/20.3 0.91</td>
<td>0.18 23</td>
<td>11</td>
<td>D</td>
<td>15/11</td>
<td>51/23</td>
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<td>Anaphylaxis to epinephrine-thiadoside, shock</td>
<td>17.0/18.7 0.91</td>
<td>0.16 34</td>
<td>...</td>
<td>S</td>
<td>14/12</td>
<td>33/29</td>
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<td>Aspiration, CPR, shock, IHD</td>
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<td>0.10 22</td>
<td>...</td>
<td>S</td>
<td>21/10</td>
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<td>Heroin overdose, shock, Bacteroides bacteremia, aspiration</td>
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<td>0.15 23</td>
<td>16</td>
<td>D</td>
<td>15/11</td>
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<td>0.11 28</td>
<td>20</td>
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<td>Fat embolus, aspiration</td>
<td>16.4/19.4 0.79</td>
<td>0.32 30</td>
<td>...</td>
<td>S</td>
<td>12/11</td>
<td>23/24</td>
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<td>13.0/18.8 0.70</td>
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<td>22</td>
<td>D</td>
<td>29/10 &gt;300/33</td>
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<tr>
<td>18</td>
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<td>Aspiration, CPR, circovirus</td>
<td>13.3/18.0 0.68</td>
<td>0.11 24</td>
<td>12</td>
<td>S</td>
<td>16/10</td>
<td>35/23</td>
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<td>19</td>
<td>72F</td>
<td>Alpha-Streptococcus bacteremia, cerebral embol, aspiration</td>
<td>15.0/21.9 0.68</td>
<td>0.27 16</td>
<td>18</td>
<td>D</td>
<td>16/11</td>
<td>41/25</td>
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<td>71F</td>
<td>Peritonitis, shock, renal failure, aspiration</td>
<td>13.9/22.0 0.63</td>
<td>0.10 23</td>
<td>20</td>
<td>D</td>
<td>16/11</td>
<td>53/25</td>
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<td>Colon carcinoma, shock, CPR</td>
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<td>0.15 16</td>
<td>15</td>
<td>D</td>
<td>17/11</td>
<td>64/24</td>
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<td>...</td>
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<td>12.3/20.0 0.61</td>
<td>0.11 20</td>
<td>D</td>
<td>5/11</td>
<td>68/25</td>
<td>155</td>
<td>...</td>
<td>...</td>
<td>19.5</td>
<td>46</td>
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</table>

**Table 1—Diagnoses, Hemodynamic, Pulmonary, Coagulation and Edema Fluid Measurements**

*EF indicates edema fluid; P: plasma; COP: colloid osmotic pressure; S: survived; D: died; protime, prothrombin time; APTT, activated partial thromboplastin time; Pt, patient; C, control; Dop, fibrinogen; FSP, fibrin split products; rheum, rheumatoid; CPR, cardipulmonary arrest and resuscitation; IHD, ischemic heart disease; AMI, acute myocardial infarction; PAW, pulmonary artery wedge pressure; and patients 28, 29, 31, 35, 36-38, and 40-43 had dynamic compliance.

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FULMINANT PULMONARY EDEMA 45

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tion of gastric contents, serious systemic infection, drug overdose, and perfusion failure were the most common factors implicated in patients with ARDS. Pulmonary embolization, near-drowning, and anaphylaxis were also contributing illnesses in some patients. In contrast, aside from cardiopulmonary arrest and hypertensive crisis, no other contributing factors accounted for edema in the patients with hemodynamic pulmonary edema. The mortality rate was high for patients with ARDS; only 8 patients (30 percent) with ARDS and permeability pulmonary edema survived, whereas 12 of 18 patients (67 percent) with hemodynamic edema were discharged from hospital ($P < 0.05$). The duration of EF production for patients with ARDS was prolonged ($\bar{X} = 212 \pm 410$ min); for patients with hemodynamic edema the duration was $43 \pm 40$ min ($P < 0.05$). For the ARDS group, there did not appear to be any correlation between the PAW and the EF/P COP ratio, or the PAW to any of the other factors except the duration of edema fluid production. When PAW was increased the interval of EF sampling was correspondingly protracted. In most instances, an elevated PAW for a patient in the ARDS group was related to volume loading in an attempt to reverse perfusion failure (shock). The interval between onset of florid pulmonary edema and death in fatal instances of ARDS ranged from one hour to more than 30 days. In the patients who died within 48 hours, shock (perfusion failure) was observed together with progressive pulmonary failure.

Although the plasma COP was slightly greater for patients with hemodynamic edema, than for the ARDS group ($\bar{X} = 22.8 \pm 3.1$ vs $19 \pm 4.5$ mm Hg), the difference was not statistically significant. No one coagulation factor appeared to correlate with the EF/P COP ratio or any of the other factors tested in the ARDS group (Fig 2). However, the most common coagulation defects were prolongation of the prothrombin and/or partial thromboplastin times. Thrombocytopenia was documented in 13 instances in the ARDS group, although the platelet count was always within the normal range for the patients with hemodynamic edema. Although there were substantial differences in the white blood cell count, leukopenia of $< 5,000$ cu mm was observed in four patients of the ARDS group. Three of these instances occurred in association with bacterial shock. Fibrinogen concentration was decreased in ten instances, and fibrin split products were identified in 17 patients of the ARDS group. No similar coagulation changes were observed for the hemodynamic group ($P < 0.001$).

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21186/ on 06/27/2017)
Only five patients with clinical features of ARDS failed to meet our criteria for a coagulation defect. For these patients, edema was due to fresh water near-drowning in two instances, and one each to aspirin overdose, bacteremia, and aspiration of gastric contents.

**DISCUSSION**

The pulmonary microvascular membrane serves to restrict the movement of fluid and protein into the interstitium and alveolus. If the integrity of the membrane is altered, a substantial increase in the escape of fluid and protein results, even in the absence of an increase in microvascular pressure. Accordingly, the fluid that accumulates in the lung will be similar to plasma and will contain a high protein content. In contrast, the protein content of edema fluid that stems from hemodynamic (high pressure) edema is low. Therefore, in fulminant edema in which edema fluid can be sampled the EF/P COP ratio can be used to assess the degree of microvascular permeability. The high EF/P ratios we observe for patients with ARDS substantiates the hypothesis that a hallmark of the physiologic defect of this syndrome is an increase in pulmonary microvascular permeability to protein. However, for the ARDS group, there was a wide spectrum of ratios, consistent with varying degrees of membrane damage. Multiple systemic and pulmonary disorders were implicated in the genesis of ARDS in each patient. In contrast, for patients with high pressure edema, the EF/P ratio was low; this supports the concept that in this form of edema, the integrity of the alveolar capillary membrane is maintained. However, the presence of red blood cells in EF that we observed in the high pressure group confirms the findings of previous studies and suggests that there may be focal microvascular damage.

Other factors that may affect the composition of fluid sampled from the airway include active transport or permeation of macromolecules across the airway mucosa. However, we believe that if precautions are taken to ensure that fluid is sampled during alveolar flooding, the fluid is likely to be representative of the alveolar-capillary flux.

We observed marked changes in the blood coagulation profile in most of the patients with clinical features of ARDS. In some instances, the findings were compatible with disseminated intravascular coagulation (DIC). Our findings are therefore analogous to those of Bone et al. However, in that study only 23 percent of patients with ARDS met criteria for DIC. Similarly, in the present study, the coagulation changes were often less specific. In contrast, in no patient with hemodynamic edema were comparable coagulation derangements observed. These data suggest that activation of the coagulation cascade and permeability pulmonary edema are interrelated; but that in severe hemodynamic edema no similar relationship pertains. Injury to the alveolar-capillary membrane appears to be a key factor in the interaction of permeability edema and coagulation derangements. The following two mechanisms may be operative: (1) damage to the pulmonary epithelial and endothelial surfaces with subsequent activation of the coagulation cascades; and (2) primary activation of the coagulation system via a systemic illness or injury that leads to secondary pulmonary damage. Both of these processes may act in concert and may be due in part to the release of vasoactive mediators that increase vascular permeability, and lead to endothelial and/or epithelial damage. In this regard, experimental acid aspiration with injury to the alveolar epithelium has been shown to increase permeability of the lung to macromolecules and to trigger the release of vasoactive substances such as histamine. In the present study, we frequently observed coagulation changes in association with gastric aspiration. However, comparable changes were not observed for the two patients with near-drowning. These data support the notion that direct injury to the airway by acid is required to initiate the release of vasoactive mediators and the induction of enzyme cascades. Similar processes are likely to be operative when the pulmonary vascular endothelium is subjected to the effects of pulmonary embolic processes. Microemboli by fibrin, fibrinopeptides or platelets and/or white blood cells have been looked to by some investigators as an attractive hypothesis to account for an increase in pulmonary vascular permeability and pulmonary vascular resistance. Nevertheless, the picture is emerging that permeability pulmonary edema, emboli, and hemostatic mechanisms are interrelated in the genesis of ARDS.

A variety of systemic disorders may primarily affect the coagulation system and lead to subsequent pulmonary injury. Bacterial shock and endotoxin have been shown to influence the kinin, complement, and coagulation systems, as well as to lead to platelet and fibrinogen destruction and consumption. Furthermore, these enzyme cascades are also interrelated and lead to the release of a variety of vasoactive mediators, including histamine. Complement-mediated leukostasis that may be caused by bacteremia or other insults with subse-
Abnormal Tests of Hemostasis

Pulmonary Edema

Coagulation

Complement

Kinin

Permeability

Abnormal

Tests of Hemostasis

...quent embolization to the lung may also lead to an increase in pulmonary vascular permeability. Therefore, we believe that in most instances multiple pathologic processes that may include primary as well as secondary lung injury and that are associated with activation of the coagulation and other enzyme systems are the causative factors of the edema that characterizes ARDS. Our findings confirm that a key feature of ARDS is permeability pulmonary edema. Although the precise mechanisms or sequence of mechanisms for these processes have not been clarified, a schema that illustrates likely pathways in the genesis of permeability pulmonary edema is shown in Figure 3. Based on the diversity of clinical features that were associated with ARDS in this and in previous studies, we believe that more than one of these features are usually required for the development of the clinical picture that typifies ARDS. In contrast, high pressure edema that is not associated with systemic or local toxic insults does not lead to coagulopathy.

Our findings point to the potentially ominous clinical implications when abnormal coagulation factors are found in association with permeability pulmonary edema. It is not clear, however, the extent to which the coagulation defects mirror the pulmonary dysfunction or are operative in the genesis and/or perpetuation of ARDS. Sampling of edema fluid and measurement of coagulation factors are helpful to classify and to assess the severity of the edema process.

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17th Annual Radiology Postgraduate Course

The 17th annual course on Chest Radiology, organized by the Department of Radiology, Medical College of Virginia, Virginia Commonwealth University, will be held March 1-5 at the Williamsburg Conference Center, Williamsburg, Virginia. Further information is available from the Department of Continuing Medical Education, Medical College of Virginia, Box 48, Richmond 23298.