MEDICAL GRAND ROUNDS
PARKLAND MEMORIAL HOSPITAL
November 4, 1965

PULMONARY EMBOLISM

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Case #1 [redacted]

This [redacted] woman was first seen at [redacted] in 1959 when she was 33 years old and five months pregnant with her fourth child. She was admitted at that time for marked hypertension which was controlled with reserpine and phenobarbital. She developed pre-eclampsia during that pregnancy; otherwise she had an unremarkable delivery in [redacted], 1960. She was not seen again until [redacted], 1961, when she presented eight months pregnant with pre-eclampsia again. At that time she delivered a macerated fetus, and following this she had a tubal ligation. Because of persistent hypertension following discharge, she was treated with reserpine, diuril, and potassium chloride. These medicines were started on an out patient clinic basis on [redacted]-61.

There was some confusion about medication dosage, and the patient took an extra amount of diuril with no potassium. She then developed a week's course of progressive malaise, weakness, and lethargy which was progressive until [redacted]-61 when she presented to the emergency room. While in the emergency room she was noticed to be tachypneic, tachycardic, and to have a blood pressure of 100/80. While in the emergency room she was observed to have a grand mal seizure. An electrocardiogram showed a vertical axis with non specific ST-T wave changes over the posterior myocardium. After admission it was considered that she most likely had an electrolyte disturbance due to the overdose of diuril. After volume replacement she became asymptomatic except for a continued tachycardia and tachypnea. She then developed a definitely accentuated pulmonary second sound that was widely split with a pulmonic systolic murmur. The chest x-ray was within normal limits, but pulmonary functions revealed a $PaCO_2$ of 29 millimeters of mercury, $PACO_2$ (end tidal) 23.4, $PACO_2$ (end expired) 24.5, and oxygen saturation of 89%. On 100% oxygen breathing she was shown to have a right to left shunt of approximately 23% of her cardiac output. A diagnosis of pulmonary embolism was made, and the patient was heparinized. She did well except for continued tachycardia and tachypnea until approximately [redacted]-61 at which time she suddenly became more tachypneic and developed a widely split P_2. EKG at that time showed ST elevation in lead III and lead AVF with T wave changes in III, AVF and V_5 and V_6. It was felt by some that these changes indicated posterior myocardial infarction, and although a vena cava ligation was considered desirable, it was decided to postpone this procedure for two to three weeks. A cardiac catheterization carried out on [redacted]-61 revealed pulmonary hypertension with a mean of 22 millimeters of mercury that did not decrease on 100% oxygen breathing. She was still desaturated to 89% on room air breathing. Following cardiac catheterization, ligation of the inferior vena cava was carried out on [redacted]61. The patient tolerated the procedure well, and except for a small amount of leg edema in the immediate postoperative period, there were no complications.

The patient had a repeat cardiac catheterization on [redacted]-61, and once again the PA mean pressure was elevated to 29 millimeters of mercury. On this occasion the patient's oxygen saturation was 93%. During catheterization the patient had a transient arrythmia which prevented complete catheterization. Bronchospirometry on [redacted]-61 revealed no oxygen uptake from the right lung. The arterial to alveolar CO_2 gradient on the right was 23 millimeters of mercury, while the gradient on the left was only 6 millimeters of mercury. Pulmonary angiogram at approximately the same time revealed complete occlusion of the artery to the right lung.

Case #1 [redacted] (continued)

It is interesting that the first definitely abnormal chest x-ray on this patient was on [redacted]-61, approximately six weeks after the inferior vena caval ligation. At that time she showed possible cavitation in the right middle lobe.

Since the initial admission for pulmonary embolus, the patient has been admitted several times for further study. Repeat cardiac catheterization, bronchospirometry, and pulmonary angiography has revealed that the blood flow to the right lung has not been re-established, and there is no oxygen uptake from that side. During the intervening years the patient has been virtually asymptomatic as far as dyspnea is concerned or as far as postphlebitic changes in the legs are concerned. She has had recurrent infiltrates in the avascular right lung indicating periodic tissue breakdown. Ultimately, she developed an infection in the right lung with atypical mycobacteria of the Group I type. Since these organisms were resistant to first line antituberculous drugs, right pneumonectomy was attempted, but during induction of anesthesia the patient became hypotensive. For this reason, the operation was abandoned, and the patient has been treated with second line antituberculous drugs.

Case #2: [redacted]

This 58 year old [redacted] man was entirely well and working as a [redacted] until [redacted], 1960, when he first noted onset of pain and swelling in his left calf. He came to the [redacted] emergency room where the diagnosis of thrombophlebitis was made and the patient was sent to the surgical clinics. There he was started on tetracycline and was advised to keep his leg elevated and at rest. Despite adherence to these instructions he noted no improvement over the next three weeks. He was seen again in clinic and started on oral varidase. Three days later he had the sudden onset of left anterior pleuritic chest pain and noted a cough productive of small amounts of whitish sputum. After five days of such symptoms the patient presented again to the hospital and was admitted to the medical service. At that time his physical exam was remarkable only in that his respiratory rate was 26 per minute, P_2 equal to A_2, and his left calf was warm, tender, and one centimeter larger than the right calf. Admission studies revealed a chest x-ray with moderate prominence of the hilar vascular shadows. There was strand-like infiltration at the left base with blunting of the left costophrenic angle. The electrocardiogram was interpreted as subendocardial infarction because of markedly inverted T waves over all of the early V leads. A left thorocentesis yielded only 1 cc of bloody fluid. A diagnosis of thrombophlebitis with pulmonary embolus with infarction was made, and the patient was started on 50 milligrams of subcutaneous heparin every six hours. This medication caused a prolongation of the Lee White clotting time to approximately three times normal. In addition, the patient was placed on bed rest with elastic stockings and the foot of his bed slightly elevated. On this regimen the patient became asymptomatic in approximately two days. Subsequently the electrocardiogram reverted to normal, and the x-ray revealed diminishing hilar structures and a disappearance of the left infiltrate. After four days the patient was started on coumadin, and when the prothrombin time was approximately 20% of control, the heparin was discontinued. The patient was ambulated after approximately one week and was discharged on oral anticoagulants after a total hospitalization of two weeks.

The patient was then followed on oral anticoagulants in the out patient department for approximately two months, and he was then lost to follow up, since he failed to keep clinic appointments.

The patient was then asymptomatic until approximately [redacted], 1964. At that time he developed a right pleuritic chest pain and cough productive of white sputum. His local physician treated him with injections for presumed pneumonia, and the patient became asymptomatic in about two weeks. At about the same time, however, the patient began to develop increasing dyspnea on exertion which forced him ultimately to discontinue working in about [redacted], 1964. During [redacted] he had recurrence of the right pleuritic chest pain which occurred intermittently over the course of the month. In addition, he had frequent cough, intermittent fever of 100 degrees, and chilly sensations. Dyspnea on exertion was progressive. Because of these symptoms, he was readmitted to [redacted] on [redacted]-64. At that time the physical examination revealed a pulse of 120, respirations of 30, and a temperature of 100 degrees. The patient obviously had splinting of his chest which was apparently due to pleuritic pain. There was dullness to percussion over the right lower lung field anteriorly, and there were decreased breath sounds in this area. P_2 was much louder than A_2, and there was fixed splitting of this sound. The left border of cardiac dullness was in the anterior axillary line. There was also 1+ pretibial pitting edema.

Case #2 [redacted] (continued)

The admission electrocardiogram showed right axis deviation, a Q_3 T_3 pattern, and clockwise rotation. Admission chest x-ray revealed the heart to be markedly enlarged, and the pulmonary arterial segment was quite prominent. The right hilar shadow was also quite prominent. Pulmonary vasculature was less prominent in the right upper lobe than elsewhere. There was a suggestion of pleural fluid at the right base. Fluoroscopy confirmed these impressions. A tentative diagnosis of pulmonary embolization was made, and the patient was started on subcutaneous heparin. On the fourth hospital day a pulmonary angiogram was performed through a venous cutdown. There was marked enlargement of the pulmonary arterial segment. The right pulmonary artery had an abrupt blockage of the contrast material and no definite pulmonary vasculature was seen in that lobe. There was also some tapering off of the visualized portions of the pulmonary arteries to the right middle and lower lobes. No abnormality was seen in the left lung. Vena caval ligation was planned, but was delayed for a few days because of the intercurrence of abdominal pain of unknown etiology. During this time, the electrocardiogram reverted towards normal, and there was some regression in the size of the pulmonary outflow tract. On the tenth hospital day the vena cava was ligated without incident. The patient subsequently made an uneventful recovery except for persistent pedal edema despite elastic stockings.

Since time of operation the patient has had no recurrence of chest pain. Dyspnea on exertion has continued at the preoperative level, but it has not become more severe. The pedal edema has slowly regressed over the intervening months. The patient was readmitted in June of 1965 for right heart catheterization and re-evaluation. The findings of this catheterization are listed below and indicate that the patient has a fixed pulmonary hypertension due to a high pulmonary vascular resistance. A repeat pulmonary angiogram performed at the time of right heart cath showed delayed emptying of contrast media in part of the left main pulmonary artery indicating some trapping by an old clot. The right lung showed no blood vessels in the right upper lobe, and the rest of the right lung showed decrease vascularization.

EVALUATION OF [redacted], [redacted], 1965

	PREDICTED	OBSERVED
Total Lung Capacity (cc)	5950	5260
Forced Vital Capacity (cc)	4000	2885
Forced Expiratory Volume 0.5 Seconds (cc)	1730	1379
Forced Expiratory Volume 1.0 Seconds (cc)	2164	2010
Single Breath Nitrogen	< 2.5%	2.1%
Membrane Diffusion Capacity (ml/min/mmHg)	74.2	36.6
Capillary Blood Volume (ml)	78.7	60.1
pH	7.35 - 7.45	7.34
$PaCO_2$ (mmHg)	40 ± 5	39.5
PA CO_2 (End Tidal)		27
PA CO_2 (End Expired)		31.5
Pa O_2 (mmHg)	> 85	57
PAO_2 - PaO_2	< 15	43
PA Pressure, Room Air Rest	20/10	79/35 (52)
PA Pressure, 100% O_2		90/40 (60)
Cardiac Output (L/min)		3.37
Pul. Vasc. Resistance (Dynes-Sec-Cm^{-5})	< 400	997

Case #3 [redacted]

This 28 year old [redacted] woman was entirely well until the [redacted] of [redacted], 1964, when she developed tenderness, redness, and swelling of the left calf which was so painful that it prevented walking. This subsided over a four day course. She then developed an episode of acute onset of left sided pleuritic chest pain, radiating to the back followed by dyspnea, sweating, and a dry, hacking nonproductive cough which lasted for approximately thirty minutes. During the next four days she had four similar episodes. This syndrome prompted her to seek medical aid on [redacted], 1964. Because of this history she was admitted to [redacted], and at the time of admission her physical examination was within normal limits except for modest obesity. While the patient was being tested for Homan's sign on the left the patient cried out in pain; on questioning she stated that she had chest pain similar to that that prompted hospitalization. The patient became acutely dyspneic. Her respiratory rate went from 16 to 28, and her pulse rose from 80 to 136. P_2 which had been less prominent than A_2 became much more pronounced. This entire episode lasted approximately five minutes. An electrocardiogram taken during the episode showed non specific ST changes which were not different from the changes observed in an electrocardiogram shortly before the episode. Chest x-ray was entirely within normal limits.

The patient was thought to have had a series of pulmonary emboli, and she was placed on 50 milligrams of heparin subcutaneously every six hours, strict bed rest, elastic stockings, and heat to the left leg. She was subsequently placed on coumadin so that her prothrombin time was kept at about 30 seconds. Her symptoms subsided with the institution of this treatment. She was ambulated on the seventh day, and was discharged after a total hospital course of two weeks.

The patient was continued on anticoagulation in the out patient department, and she continued to do well. Repeated chest x-rays and electrocardiograms were within normal limits. Because of troublesome gum bleeding while brushing her teeth, the anticoagulants were discontinued in mid[redacted], 1964. Her total course of anti-coagulation was, therefore, approximately two months.

On about the first of [redacted], 1964, the patient again noted pain, redness, and swelling in her left leg. Shortly thereafter she had the sudden onset of pleuritic chest pain, dyspnea, and a dry hacking cough. She returned to the hospital and she was admitted again on [redacted] 1964. At the time of admission the physical exam was entirely negative except for mild tenderness in the calf of the left leg with a tender cord in the left popliteal fossa. The electrocardiogram was unchanged, and the chest x-ray was again within normal limits. The pulmonary angiogram performed through a peripheral vein demonstrated no anatomical abnormality of the pulmonary vascular tree. The patient was nevertheless considered to have had recurrent pulmonary emboli, and she was restarted on coumadin therapy. Total hospitalization on that admission was for fifteen days, and the patient was discharged on anticoagulants. These have been continued to the present time, and the patient has not had recurrence of her previous symptoms.

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REVIEWS

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The review by Parker and Smith is excellent except for the section on treatment. The approach taken by Hickam and Sieker is somewhat more didactic, but it is nevertheless excellent. The latter has a good section on treatment also.

INCIDENCE

5. Breckenridge, R. T. and Ratnoff, O. D.: Pulmonary embolism and unexpected death in supposedly normal persons. N. Eng. J. Med. 270:298, 1964.

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It is extremely difficult to arrive at a meaningful figure for the incidence of thromboembolism. Reports based on clinical diagnosis are gross underestimates. Freiman reports that at Beth Israel Hospital in Boston only 3 of 39 autopsy proven cases were diagnosed clinically (7.5%). Coon (see below) reports a similar diagnostic rate (7.1%) at the University Hospitals of Michigan. Although these estimates are likely intentionally low, certainly the experience of such institutions can not be overlooked.

Pulmonary embolism may even be easily missed at routine postmortem examinations. In a beautiful article Korn describes the evolution of emboli and thrombi in pulmonary arteries into delicate threadlike structures of fibrous consistency which may be easily ignored. Freiman indicates that evidence of pulmonary embolism was found in 29% of routine postmortem examinations, but when these same tissues were studied more carefully, the actual incidence rose to 64%.

In patients autopsied in general hospitals the occurrence of embolization is reported between 10 and 40%. The incidence of pulmonary emboli believed to be the major cause of death is approximately 2%. Towbin, however, reasons that these data are on an extremely selected population. He therefore studied the autopsy incidence of embolism at the Columbus State Hospital which is a custodial institution where the age distribution is similar to the general population. He found that thromboembolic lesions were present in 25.7% of the cases, and that massive pulmonary embolism was the cause of death in 14.2%. In all of the data reported, the incidence rises dramatically over the age of 50.

Belding reports in a massive series of various operations the relationships of death due to pulmonary embolus to the total number of surgical deaths. He finds, for example, that the mortality rate due to cholecystectomy is 1.34%, and that of these deaths 62.5% are due to pulmonary embolism. Similarly of the 0.37% mortality rate following herniorrhaphy, 78.5% are due to pulmonary embolus, and of the 0.56% mortality rate following hysterectomy, 61.5% are due to emboli.

VENOUS THROMBOSIS

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In 1856 Virchow first proposed the factors that are still considered important in the genesis of venous thrombi: stasis, injury of the vascular wall, and hypercoaguability. Conditions predisposing to venous thrombosis, presumably by one of these mechanisms, include prolonged bed rest, heart failure, the postoperative state, hypotension, trauma to the lower extremities, parturition, polycythemia, carcinoma, dehydration, and possibly obesity. In the presence of any predisposing factors, increasing incidence of thrombosis is associated with increasing age.

There is some disagreement about the frequency of the site of origin of thrombi in the venous system. This is at least in part true because the meticulous dissection of a venous circulation necessary for finding the thrombosis is rarely carried out. Studies like the beautiful work by McLachlin (11) indicate, however, that almost all emboli originate in the pelvis or lower extremities. Pulmonary emboli from the right heart are unusual in the absence of frank cardiac disease, and emboli from the upper extremities are extremely rare. McLachlin's work also indicates that superficial femoral vein ligation would not have prevented pulmonary embolism in 21 of the 34 cases of venous thrombosis. In addition, it has been found that thrombi are bilateral at least 50% of the time.

Wright (12) has shown that in the supine position the flow rate in the leg veins is approximately 4.2 centimeters per second. On standing and sitting the flow falls to 2.6 centimeters per second, while in the head down (10^{o}) position flow increases to 9.4 centimeters per second. McLachlin (14) has shown by a dye technique that blood may remain in the venous valve cusps for over 25 minutes when the patient is in a horizontal position. The best way to augment flow was found to be elevation of the foot of the table.

Despite many suggestive reports (21, 22), the Federal Drug Administration Committee has concluded that there is no significant increase in the risk of embolic death from the use of Enovid. Winter (23) has extended the statistical review of that Committee, and he has reached similar conclusions. Specifically from 1958 through 1962 the total female population mortality rate from embolism was 9.66 per million per year. The mortality from pulmonary embolism among Enovid users in 1963 was 7.4 per million. Winter also finds on careful review that although pregnancy is usually included as a predisposing cause for thrombosis, there is evidence to show that delivery and the puerperium and not the pregnant state itself are conducive to an increased incidence of thromboembolic morbidity.

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Pulmonary embolism may be divided into three syndromes based on the size of the offending embolus. Although these syndromes may overlap greatly, their differentiation seems useful. The first syndrome is that of a large pulmonary embolus unaccompanied by pulmonary infarction. This syndrome is most frequently manifested by one or more of the following signs or symptoms: dyspnea, substernal chest pain suggesting angina or myocardial infarction, tachycardia, symptoms of cerebral ischemia, electrocardiographic changes, shock, evidence of right ventricular failure, fever, or sudden death. The second syndrome is that of pulmonary embolism accompanied by pulmonary infarction. The manifestations of this syndrome usually include one or more of the following: pleuritic chest pain, x-ray densities, fever, hemoptysis, cough, dyspnea, tachycardia, or an elevated leukocyte count. The third syndrome is that of recurrent, small pulmonary emboli with or without infarction. This syndrome is the most difficult to diagnose, since the signs and symptoms are usually minimal until late in the course of disease. Although any of the previous signs and symptoms may be present, the only manifestations may be those of progressive dyspnea on exertion, episodic dyspnea unassociated with physical findings, or the unexplained occurrence of pulmonary hypertension with cor pulmonale. The three cases at the beginning of this protocol illustrate an example of each syndrome.

PHYSIOLOGICAL ALTERATIONS

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Pulmonary embolism of any extent is accompanied by an increase in the pulmonary vascular resistance and pulmonary artery pressure, and by a fall in the cardiac output. It is still debated whether the increase in the pulmonary vascular resistance is due to mechanical blockage of the vascular bed or by a reflex vasoconstriction. It is quite likely that vasoconstriction plays some part in the increased resistance due to small emboli, and current views favor serotonin as the mediating factor. There is also an increase in airway resistance, and it is also thought that this is mediated by serotonin. It has been demonstrated that the increase in airway resistance can at least in part be blocked by heparin. The increased airway resistance is greatest in the area of embolization, and this tends to decrease ventilation in the area of absent blood flow. Despite this, however, some ventilation in the embolized area continues, and hence a greater proportion of the total ventilation than normal is expended in deadspace ventilation. The changes in airway resistance causing partial shutdown in small lung units also result in a decreased compliance.

Embolization is almost universally associated with tachypnea and an increase in total minute ventilation. It has been stressed by some investigators that the minute ventilation is disproportionately greatly increased during mild exercise.

Embolization is frequently associated with arterial hypoxia. The major cause of hypoxia is the occurrence of right to left shunting. Although the exact site of the shunts has not been definitively proven, it is likely that these shunts are in anatomically present but normally shut blood vessels between small pulmonary arteries and pulmonary veins bypassing the pulmonary capillary network. In addition, mild desaturation may be caused by ventilation perfusion disturbances and a mild diffusion defect caused by obliteration of part of the pulmonary capillary bed. These disturbances regress over the course of a few days, and the patient's oxygen saturation again returns to normal.

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Although the LDH level usually rises after pulmonary embolism with infarction and SGOT levels are usually normal in this circumstance, this pattern of change is frequently seen in other types of cardiopulmonary disorders. Moreover, it is not invariably seen in patients with pulmonary embolism. Initial enthusiasm toward the use of enzymes in the diagnosis of pulmonary embolism has therefore waned.

A reported incidence of positive electrocardiographic changes varies widely. The method of case selection probably is the reason for the disparate findings. In massive pulmonary embolism the electrocardiogram usually shows some sort of change, but in small multiple emboli the electrocardiogram usually remains normal until cor pulmonale develops.

The classic EKG pattern of acute cor pulmonale described by McGinn and White in 1935 occurs in only about 10% of patients; this pattern is a large S_1, Q_3, with an inverted T_3 with inverted T waves in the right precordial leads. Other changes include ST segment depression in leads 1 and 2, appearance of right axis deviation, development of clockwise rotation of the heart, appearance of right bundle branch block or right ventricular enlargement, peaked P waves in leads 2, 3 and AVF, or a variety of nonspecific ST - T wave changes.

The classic article defining x-ray and postmortem changes with pulmonary emboli is by Hampton and Castleman (53). It is emphasized by these and other authors that pulmonary embolism may cause no changes on x-ray except the disappearance of normal vascular shadows which may be very difficult to detect. Pulmonary infarctions are more likely to cause x-ray changes, but these are rarely diagnostic. The most common changes are pleural effusions, homogeneous densities, elevation of the diaphragm on the affected side, and linear densities presumably representing areas of atelectasis.

Abnormal findings occur within 24 hours of the onset, and they may clear rather slowly requiring longer than a month in many cases.

Pulmonary angiography is the most important new diagnostic procedure in many years. Even angiograms are of no benefit, however, in medium size to small emboli. Angiography performed through peripheral veins is satisfactory for gross emboli, but to be really detailed, the dye must be injected through a right heart catheter. The newer procedure of lung scan after injection of radioactive macroaggregated albumin does not seem to offer any great advantage. The technique can pick up only rather large emboli, it can not differentiate pulmonary infarcts from pneumonia, and it can not be used in severely ill patients since the patient must lie still for approximately 20 minutes during the lung scan.

TREATMENT

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Anticoagulants are the mainstay of treatment of thromboembolism. Despite the lack of well controlled studies during the 1940's and '50's, the beneficial effects of anticoagulants have been convincing, so that the controversy that surrounds their use in myocardial infarction has been avoided. The study by Barritt (71) is a well constructed series with parallel control cases, and it indicates that anticoagulant therapy after the patient has had pulmonary embolism reduces the risk of death from that embolism and the liklihood of recurrent embolism. The estimate by Crane (65) of approximately 10 to 12% failures in patients treated with heparin seems reasonable at least in the clinically diagnosable case of embolism. Despite animal experimentation which indicates that heparin but not coumadin is effective in preventing venous thrombi or extension of already existent thrombi (66, 67, 70, 77), the excellent study by Sevitt (69) and subsequently others (80, 83) indicate that oral anticoagulants are effective prophylactic agents in the prevention of thromboembolism.

Fibrinolysins must still be considered experimental agents. When reports of results with this type of therapy are evaluated, it must be borne in mind that even quite large pulmonary emboli may undergo spontaneous lysis. This has been well demonstrated by angiography at this institution by Dr. Robert L. Johnson, and this has recently been reported in abstract form by Dr. Herbie Fred at Baylor.

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Venous ligation is usually reserved for those patients who have continued emboli while on anticoagulants. Although some surgeons still perform ligation at the femoral level, most ligations are now of the vena cava, since many thrombi originate proximally to the site of femoral ligation. The incidence of postphlebitic complications reported varies considerably from series to series. It would appear, however, that with meticulous postoperative care, vena cava ligation is not associated with the particularly high incidence of severe symptoms. There is little indication that vena cava plication is associated with fewer symptoms than ligation.

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There is some indication that thrombectomy will reduce the incidence of postphlebitic symptoms, but this procedure is still in the experimental stage.

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There is no doubt that pulmonary embolectomy is now technically possible. Since approximately 25% of patients who sustain a massive embolus live greater than one hour, the operation would be feasible in at least this group. At least some of these patients, however, will subsequently recanalize the pulmonary artery, and hence these patients would not logically be candidates for embolectomy. Furthermore, other patients may do relatively well without embolectomy, and the operation itself carries a considerable mortality. For these reasons, the exact indications for embolectomy have not as yet been determined.

MISCELLANEOUS

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There is an interesting relationship between pulmonary infarction and hemoglobinopathies. It is thought most likely that the infarctions are caused by thrombosis in situ, but this point is not absolutely proven. Since this is thought to be true, however, venous ligation is usually not carried out in such patients.

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This is one of several articles indicating that serotonin is present in high concentration in blood clot, and this causes many of the physiological disturbances noted in experimental animals during pulmonary embolization.

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This author feels that by relating the patient's age, per cent over weight, preanesthetic immobilization, number of hours of anesthesia, postoperative immobilization, occurrence of previous operations, and existence of factors known to produce venous thrombosis he can predict the liklihood of postoperative thromboembolism.

103. Engelberg, Hyman: Cigarette smoking and the in vitro thrombosis of human blood. J.A.M.A. 193:1033, 1965.

Cigarette smoking damned again.

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This article gives a nice description of the time of appearance of EKG changes after embolization. The most consistent and persistent change is the T wave inversion especially over the right ventricle leads.