CONGESTIVE ATELECTASIS — A COMPLICATION OF THE INTRAVENOUS INFUSION OF FLUIDS*

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PAUL BERT[4] described congestive atelectasis as a phenomenon attending rapid decompression. Hurtado[14] first described the microscopic appearance thereof in the lungs of guinea pigs. Fegler and Banister have performed well-controlled experimental studies upon its etiology relative to decompression. Their description of the gross and microscopic appearance of the lungs is as follows:

"Congestive atelectasis is the term chosen to describe a further lung change in which the microscopical picture showed maximal dilatation of the lung capillaries and complete exclusion of air from the alveolar spaces. Macroscopically the condition suggested gross hemorrhage. The areas affected were sharply limited and stood out from the surrounding tissue because of their liver-like, uniform, dark red coloration; thus they were readily distinguishable even when they co-existed with severe hyperemia in the same part of the lung. Closing the trachea before opening the chest intensified the contrast between hyperemia and congestive atelectasis. Inflation of the lung caused the congestive airless parts of the lung to disappear and a restoration of the normal pink coloration, which showed the condition to be congestive and not hemorrhagic. The areas affected by congestive atelectasis were not confined to a particular part of the lung surface nor to any one lobe. However, they seemed to develop more frequently around the hilum or on the dorsal surfaces."[10]

During the past nine years the gross and microscopic characteristics of congestive atelectasis in patients have been seen in more than 20 instances; and rapid decompression was not an etiologic factor in any case. The following case summaries present the clinical experience with the phenomenon during the last three years.

CASE SUMMARIES

Case 1.—A. G. (Pkd. 38434), a 26-year-old male, was admitted August 8, 1946, with acute appendicitis and rupture of the appendix. Circulatory and respiratory status were satisfactory. Temperature, 99.6° F., pulse 100, blood pressure 120/70.

Appendectomy was performed, following anesthesia with pentothal sodium (0.5 Gm.), followed by cyclopropane and oxygen, and 160 units of "Intocostrin." Anesthesia was uncomplicated except for profuse sweating. No fluids were given intravenously before or during operation. Immediately postoperatively, a solution of 5 per cent dextrose in

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distilled water containing 3 Gm. sodium sulfadiazine per liter was started intravenously, but was discontinued shortly thereafter when the patient suffered a chill.

Postoperative course. Three hours after the chill began he was sweating profusely, and was moderately cyanotic; pulse rate 140, blood pressure 80/30, temperature 99.8° by mouth. One liter of 5 per cent dextrose in distilled water was given slowly intravenously.

Six hours after the chill his rectal temperature reached 106°, pulse rate more than 180, and blood pressure was 69/30. Respirations were 38 per minute and labored. He was cyanotic and sweating profusely.

Physical examination of chest, on August 9, revealed: right inspiratory lag; diminished expansion of right chest, with an expiratory position of the right hemithorax; distant breath sounds with patches of dullness over the right chest. On the same date, roentgen examination revealed: upper two thirds of right lung clear; base of right lung somewhat hazy, probably the result of technical variation rather than intrapulmonary disease. Bronchoscopic examination at this time yielded moderate amount of thick secretion in both main bronchi, more especially in the right one. Following this examination a loose, rattling cough developed, productive of only small amounts of thick, clear sputum.

Oxygen was administered through an oro-nasal mask, but cyanosis persisted. One and a half liters of 5 per cent glucose in normal saline were given slowly, intravenously. Cold packs and rectally administered aspirin were employed to lower his fever. Five hours after bronchoscopy his temperature was still 103° F., pulse rate was 140, respiration 26, and blood pressure 80/40. Cyanosis had disappeared and he was breathing easily.

Twelve hours later the oxygen was discontinued, and cyanosis did not reappear. He had no fever, pulse rate had fallen to 112, respiratory rate was normal, but blood pressure was still only 84/50. The right chest still lagged during inspiration, and dullness and bronchial sounds were found over large areas of the whole right hemithorax. No evidence of tracheal deviation or of mediastinal shift was found.

One day later only the chest lag remained and he felt well. Blood pressure was then 100/60.

Roentgenogram of the chest on August 12 indicated no evidence of atelectasis, tuberculosis or pneumonia. Haziness at the base of the right lung had disappeared.

Case 2.—J. V. (pkd. W51872), a 59-year-old woman, was admitted October 6, 1947, suffering from diabetes mellitus and meningococcal infection. There was a 3-week history of weakness, dizziness, polydipsia, polyuria, and anorexia, progressing to coma 16 hours before admission, with disorientation for 12 hours preceding the coma.

Physical examination. Her rectal temperature was 104.4°. The skin was dry and cool and without turgor; breathing was shallow, rapid (48 per minute), and grunting. The lungs were clear to auscultation; chest expansion was free and equal. Blood pressure was imperceptible to auscultation; peripheral pulse was 120 per minute and difficult to palpate. The point of maximum intensity of the cardiac beat was diffuse and outside the mid-clavicular line. The abdomen was distended and tympanitic. There was no roentgen ray evidence of parenchymatous infiltration in either lung field. There was some haziness of both costophrenic sinuses. Petechiae covered most of the body, and there were many large purplish indurated areas over the lower extremities.

During the first 12 hours the urine output was 400 ml., and the fluid administered totaled 10,450 ml., of which 4450 ml. were of a balanced salt solution, and the remainder was of 5 per cent dextrose in distilled water. Antibiotics and insulin were also given. During this period the blood pressure reached 120/90, and the patient became rational. The carbon dioxide combining power reached 50.4 volumes per 100 of plasma, and the blood sugar fell to 484 mg. per 100 cc.

During the next 24 hours she became comatose again, and her blood pressure was not obtainable. She received 2750 ml. plasma, and 4000 ml. glucose in distilled water.

Urine output during this time was 660 ml. Respiratory rate rose to 36 per minute, and breathing was abdominal in character and labored. Breath sounds were absent over much of both lung fields. Tidal air was very small. Cyanosis was definite. Roentgen ray examination of the chest showed elevations of both hemidiaphragms without evidence of pneumonia, pleural effusion or other abnormalities.

On the third hospital day she again became lucid, but later while being bathed, she suddenly ceased breathing, and a peripheral pulse was not obtainable. Following an intracardiac injection of 0.5 mg. epinephrine and artificial respiration by intermittent positive pressure oxygen by mask, she began breathing again, and the pulse was palpable. She remained comatose throughout the remainder of her hospital course. Fluid therapy during the third hospital day was 800 ml. 5 per cent dextrose in distilled water, intravenously, and 1000 ml. of 5 per cent dextrose in distilled water by clysis. She excreted 100 ml. of urine.

On the fourth hospital day she was given 500 ml. of blood intra-arterially, and the blood pressure reached 105/70 and remained elevated while an additional 1000 ml. of blood were given intravenously. Desoxycorticosterone was also given. Urine output for the fourth day was 375 ml.

Digitalization was carried out on the fifth hospital day, using "Purodigin," 1.2 mg. intravenously. The systolic blood pressure remained between 85 and 116, while the diastolic was 60 to 80 mm. of mercury. Respirations were rapid and shallow, and the pattern was not changed by caffeine sodium benzoate, intravenously, or by carbon dioxide inhalations. Fluids for the fifth day consisted of 2000 ml. of 5 per cent dextrose in distilled water intravenously, and 2400 ml. of 5 per cent dextrose in distilled water by clysis. She excreted 300 ml. of urine.

Her physiologic status was apparently unchanged on the sixth hospital day, and her fluid intake consisted of 1000 ml. of 5 per cent dextrose in distilled water by clysis, and 1000 ml. of a balanced salt solution by clysis. Her urine output was 300 ml.

On the seventh hospital day there was an almost continuous flow of brown, liquid feces. Persistent cyanosis was present. Breathing was very rapid and labored. Distant bronchial breathing was present over both lower lung fields. There was dullness to percussion over both lower lung fields, and coarse, fine, persistent râles throughout the lungs. Respiration was assisted by positive pressure oxygen. Roentgen ray examination demonstrated atelectasis in the right lung base, posteriorly, and probable pleural effusion on the right. Respiratory arrest took place, followed shortly by cardiac standstill. On this day she had received 1000 ml. of a balanced salt solution by clysis, and an uncharted total of feedings by stomach tube.

Postmortem findings. Grossly bilateral massive atelectasis was present. Mucopurulent material was found in the trachea and in the primary bronchi. This material was soft and easily detached from the air passages. The right lung weighed 400 Gm. and was considerably collapsed. The parenchyma of the right lower and right upper lobes was firm in consistency and of a dark purplish color. The cut surfaces were deep reddish purple in color and of homogeneous quality throughout. The parenchyma was firm, rubbery in consistency, and fluid was not expressible. The middle lobe was crepitant and of a salmon-pink color. The left lung weighed 400 Gm. and showed the same general appearance as the right lobes. Extensive atelectasis with moderate intracapillary congestion was discovered histologically. No intra-alveolar hemorrhages, no pulmonary edema, no emphysema, and no bronchiolar plugging could be found.

Case 3.—H. J. (Pkd. 63178), a 36-year-old man was admitted October 1, 1948, suffering from a shotgun wound of the right buttock with perforation of the rectum and sigmoid colon, retroperitoneal hematoma, and primary syphilis. He was in profound peripheral circulatory failure. The blood pressure was unobtainable; 750 ml. of plasma and 500 ml. of blood were administered intravenously. Following this, his blood pressure rose to 110/60. However, he experienced a chill during the administration of the

second bottle of plasma after 250 ml. were injected. When anesthesia was begun his blood pressure was 115/50, his pulse was 88 per minute, and he was fully conscious.

Pentothal sodium with nitrous oxide-oxygen induction was used for anesthesia. This was followed by endotracheal ether-oxygen maintenance. The laceration of the sigmoid was closed, and proximal sigmoid colostomy, debridement of the wound of the buttocks, and a posterior incision to drain the perirectal space were accomplished. During the operation 1400 ml. of blood were given intravenously. Blood pressure at this time was 90/45, pulse 100, and respirations 36.

Postoperative fluid therapy was as follows:

During first 12 hours, 1000 ml. blood and 1000 ml. 5 per cent glucose in water were given. Blood pressure was 60/40, pulse 120, and respiration accelerated up to 52 per minute.

At 15 hours, 500 ml. blood were given. Blood pressure was 16/0, pulse 132.
At 18 hours, 500 ml. plasma were given. Blood pressure was 68/40.
At 20 hours, 500 ml. blood were given. Blood pressure was 68/40.
At 27 hours, 500 ml. blood were given. Blood pressure was 60/40.
At 28 hours, 500 ml. blood were given. Blood pressure was 76/50.

On October 3 he showed resistant cyanosis. A roentgen ray film of the chest showed an area of infiltration behind the cardiac silhouette on the left. This had the appearance of pneumonia, with some atelectasis. The right base, the site of the clinical findings, was clear. The following excerpt from the resident's note describes his subsequent course, approximately 36 hours postoperatively.

"October 3, 7 P.M. Pulse 120, temperature 98² axillary. Venous distention is prominent in neck veins. Marked expiratory effort is present. Paradoxical pulse = 10 mm. mercury. P_2 is louder than A_2, and P_2 has a slapping quality. There are crepitant râles in right lower lung field. Median basilic venous pressure = 170 mm. saline. P.M.I. is in anterior axillary line and left cardiac border has advanced 2-3 cm. laterally to percussion. Four ml. Cedilanid given intravenously. This dose to be repeated in 4 hours. Patient has passed no urine since operation, so an indwelling catheter was inserted and about 200 ml. dark brown urine was obtained.

"October 3, 12:00 M. Blood pressure 110/60, pulse 72. Respiration of normal depth and no expiratory effort. No Cedilanid to be given tonight. He has improved remarkably.

"October 4, 1:05 A.M. Death occurred suddenly and without any unusual event. Respirations ceased first, then the pulse and heart beat ceased in about two minutes. Ten minutes prior to death, the nurse checked blood pressure = 104/50, and pulse = 72."

Postmortem findings. Large gunshot wound of right buttocks, extending into the pelvis; elevation of the hemidiaphragms to the fifth interspaces. Weight of right lung 900 Gm. (weight of left lung not recorded). Diffusely red pulmonary parenchyma with slate-blue color of dependant portions; the intrapulmonary and the right main bronchi were obstructed with clotted blood. A very hemorrhagic mass of pulmonary tissue was found in the lower posterior lobular segments of the right lung. This looked like an infarct, but obstruction to the respective pulmonary arterial segments could not be found. The left lung was grossly similar to the right. Only the anterior medial segments of both lungs contained any air. Edema fluid could be expressed from scattered areas on the cut surfaces. (See Figure 9A.)

Microscopically all sections of the lungs showed intense capillary congestion, massive intra-alveolar hemorrhage, patchy areas of pulmonary edema superimposed upon the congestion and hemorrhage, and incomplete expansion of the pulmonary parenchyma.

Case 4.—J. G. (Pkd. W43543), a 45-year-old man was admitted January 5, 1947, with a diagnosis of perforation of a jejunal ulcer seven years after the performance of a posterior gastro-enterostomy. His respiratory rate was 20, and no signs of

peripheral circulatory failure were present. All pulmonary and cardiac auscultatory signs were normal; 1000 ml. of 5 per cent dextrose in water were administered intravenously.

Omental closure of the perforation was done, using pentothal sodium (0.5 Gm.) anesthesia induction, followed by cyclopropane-oxygen and 80 units of "curare." During the operation 400 ml. of 5 per cent dextrose in water were given intravenously.

For the first two postoperative days he felt well and had no fever. Two liters of 5 per cent dextrose in water and one liter of 5 per cent dextrose in saline were given intravenously each day.

At 1:25 A.M. on the third postoperative day, while he was receiving a rapid intravenous infusion of 5 per cent dextrose in saline (600 ml. in 40 minutes) he complained of nausea and suffered unproductive retching. The infusion was stopped and he quieted and slept. One hour later he was found pulseless. Oxygen was started, Coramine and adrenalin were injected intramuscularly, and 1000 ml. of plasma and 500 ml. of blood were infused intravenously. His mouth temperature then rose to 105°, his pulse to 120-140 per minute, and his respirations to 32. An ashen cyanosis appeared, and he became disoriented. His blood pressure was recorded at $\frac{40\text{-}60}{30\text{-}40}$ for the next 48 hours. Oxygen was administered through a mask and later by a tent. Examination of the chest 15 hours after the onset of trouble showed right inspiratory lag, forceful, grunting expiratory movements, multiple areas of dullness and distant bronchial breathing over the right middle and lower lobes. The heart sounds were distant. No râles could be heard. No signs of a mediastinal shift were elicited, and the tracheal position was normal. The presence of a "massive collapse" of the right middle and lower lobes was suspected by one examiner but not by others. There were no peripheral signs of congestive cardiac failure. No roentgenographic evidence of atelectasis or other significant abnormality of the chest was observed.

The following day, 2 ml. of "Cedilanid" were injected intravenously, and his blood pressure rose. On January 11, respiratory rate was 24, and no cyanosis was present. Examination of chest was as on January 9. However, the signs of pulmonary trouble spread gradually. The areas of dullness and damped bronchial breathing over the right lower lobe coalesced, and dullness appeared over the right upper and the left lower lobes during the following days. Persistent basilar and right axillary râles were noted. Cyanosis returned on January 14 and persisted, and the patient continued to be disoriented. Respiratory rate was 30 per minute. He also became oliguric and azotemic. Five days after the onset of trouble, a bronchoscope was passed and small amounts of a thin, watery, blood-stained fluid were seen in and removed from the major bronchi. The bronchi were unobstructed. The bronchial mucosa was boggy and red. Considerable expiratory bulging of the bronchi and trachea were noted. After removal of the fluid, a foamy fluid flowed into the main air passages. He died an asphyxial death 6 days after the "infusion reaction." A film of the chest taken on the day of death with a mobile unit showed an extensive infiltration involving practically the entire right lung field, which was slightly less dense around the periphery. On the left a dense infiltration extending from the hilum which does not extend to the periphery, was seen. These changes were considered to be due chiefly to pulmonary edema, although the possibility of some bronchopneumonia existed.

Postmortem findings. The right lung weighed 730 Gm.; it was reddish-grey in color. The upper lobe was firmer than the remainder of the lung; it was not crepitant. The cut section of this lobe was reddish-grey in color and had a homogeneous fleshy appearance. A moderate amount of thin reddish fluid was expressible from the cut surface. The middle and lower lobes were pinkish-grey and were moderately crepitant. The intrapulmonary bronchi of all lobes were hyperemic and contained some thick mucinous yellow material. The pulmonary arteries were patent. The left lung weighed

590 Gm., and the major portion of the upper lobe and all of the lower lobe were non-crepitant. The non-crepitant areas were reddish-grey, and after sectioning a small amount of thick reddish fluid could be expressed from the cut surface. The intrapulmonary bronchi were hyperemic and contained a thick yellow mucinous material. The pulmonary arterial branches were normal. The heart was normal except for a number of fine fibrinous strands which connected the visceral and parietal pericardial surfaces. The epicardial surface was reddish and had a granular appearance.

Microscopically the sections of the lungs showed two types of abnormality. One was characterized by complete atelectasis with congestion of pulmonary capillaries, small, widely separated areas of intra-alveolar hemorrhage, no edema, and filling of the bronchioles with disintegrating and disintegrated (ghosts) red blood cells. The other was marked by incomplete collapse of alveoli, widespread intra-alveolar hemorrhages, innumerable heart-failure cells, intense capillary congestion, small patches of edema, and filling of the bronchioles with red cells and polymorphonuclear leukocytes. No bacterial colonies could be found and the alveoli were free of leukocytes.

Case 5.—R. F. (Pkd. 64224), a 62-year-old man, was admitted November 8, 1948, suffering from a perforated carcinomatous gastric ulcer. Blood pressure was 150/74, pulse 120, and respiration 18. There were no signs of peripheral circulatory failure, or of congestive heart failure. A_2 was equal to P_2. There was moderate emphysema, and no râles. Lung fields were clear to percussion and auscultation. Roentgen ray film of the chest together with AP and transverse films of the abdomen showed air under the right diaphragmatic leaf with an underlying fluid level. Infiltration, considered to represent atelectasis, in the adjacent portion of the right lung was detected. No preoperative fluid was given.

Omental closure of the perforation was performed under nitrous oxide-oxygen-ether anesthesia given by intratracheal tube. During the operation 500 ml. of blood were given intravenously.

During the anesthetic recovery period signs of peripheral circulatory failure appeared and 500 ml. of blood and 1000 ml. of lactated Ringer's solution were given in 3 hours. Subsequently, one liter of 10 per cent glucose in saline and one liter of 10 per cent glucose in water were given intravenously. Total intravenous fluid input for the day of operation was 5 liters, and no urine was passed.

The next day his temperature and pulse had fallen toward normal levels, and all signs of peripheral circulatory failure had disappeared. Following the administration of one liter of Hartmann's solution and 2 liters of 5 per cent glucose in water, subcutaneously, he developed labored breathing and tachypnea. Visible, forceful expiratory efforts were being made. Venous pressure was elevated and expiratory wheezes and râles were heard over the lower pulmonary fields bilaterally. No peripheral edema was present. He was placed in a sitting position and the fluid administration stopped. Breathing became less labored. Digitoxin (0.3 mg.) was given intravenously. He passed no urine this day.

The respiratory difficulties persisted and progressed slowly. Two liters of fluid were given intravenously for the 3 subsequent days, and 225 ml. of urine were passed. On November 11 respirations were labored, 20 per minute, and were marked by bilateral basilar expiratory wheezes. Numerous fine crackles and posttussive râles up to the fifth dorsal spine were heard. Dullness to percussion was present over both lower lung fields. The pulmonary arterial second sound was louder than the aortic sound. Roentgen ray showed persistence of the atelectasis on the right and a rather homogeneous area involving both lower lung fields. The latter was considered to be due to hypostasis.

An hyperemic and edematous bronchial mucosa was all that was seen at the time of bronchoscopy. Because of the failure to find bronchial obstruction while he showed signs of a rising right diaphragm, tachycardia, and rising temperature, the right sub-

phrenic spaces were explored under local anesthesia, and found normal. Death occurred 4 hours later.

Postmortem findings. The lungs filled 80 to 85 per cent of the opened chest. The right lung weighed 775 Gm. The right upper and lower lobes were deep reddish brown and firm in entirety. The middle lobe was normal. No bronchial obstruction was found. The entire left lung simulated the right upper and lower lobes, and contained little air; considerable hemorrhagic material was expressible therefrom after section. The pulmonary arteries were all patent. An intense capillary congestion with massive intra-alveolar hemorrhage, minimal pulmonary edema, and incomplete expansion of the alveoli were observed microscopically in the section taken from the right lower lobe. The right upper lobe section was similar to that taken from the right lower, except that more edema was present. The section taken from the right middle lobe showed only capillary congestion.

Case 6.—A. M. (Pkd. 65115), a 52-year-old man, was admitted December 16, 1948, suffering from gunshot wounds of the pharynx, mandible, colon, and liver. He had mild peripheral circulatory failure and was conscious. Blood pressure was 100/72, pulse 120, respiration 28. Peripheral pulses were of good volume. Respiration was somewhat labored. No râles were heard. Point of maximum cardiac impulse was at the midclavicular line in the fifth left intercostal space. Five hundred ml. of plasma and 250 ml. of blood were given intravenously.

At operation, a colostomy followed by tracheotomy were performed. The anesthetic consisted of pentothal sodium, 0.4 Gm., and 80 units of d-Tubocurarine followed by tracheal intubation and endotracheal semi-closed ether vapor anesthesia—120 ml. of ether were used. Bronchoscopic examination was performed just before the tracheotomy. The bronchial tree was free of blood and secretions. Blood pressure was well maintained until the termination of the operative procedures, and then it fell precipitously to 80/60 following bronchoscopy; 750 ml. of blood were given intravenously during the operation.

Because of the acute hypotension, 500 ml. of blood were given immediately after the termination of the operation. The blood pressure rose to 120/80, and the pulse rate fell to 120, but the minute respiratory rate increased to 34-40, with intervals of Biot and Cheyne-Stokes breathing. Ten hours postoperatively râles were heard over the whole right thoracic cage, and the point of maximum cardiac impulse was found to have shifted to the mid-axillary line without shift of the trachea or mediastinal structures. Breath sounds were transmitted well through the thoracic cage. Digitalization was begun with "Crystodigin," 0.4 mg., intravenously. His respiration remained shallow, labored and rapid. Periods of apnea lasting 30 seconds or more began, and his blood pressure began to fall. One-half liter of blood was then given and his blood pressure rose to 150/100. Five hundred ml. of plasma and a liter of 10 per cent glucose in water were then given (500 blood, 500 plasma, 1000 water in four hours) and the blood pressure fell steadily, the pulse rate increased, and the respiratory rate remained about 40. More "Crystodigin" (0.4 mg.) was administered intravenously and the blood pressure rose to 170/110. Later the blood pressure again fell precipitously and after the injection of another one-half liter of blood it disappeared and could not be determined before his death 8 hours later. Breathing became very labored, deep, and remained rapid. Repeated tracheal aspiration throughout his postoperative life removed very little. Consciousness did not return at any time after the anesthesia was begun. Oxygen was administered via an oro-nasal Boothby-Lovelace mask throughout the postoperative period.

Postmortem Findings. The lungs weighed 1755 Gm. (his liver weighed 1825 Gm.). Only the anterior superior surfaces of the upper lobes contained air. The remaining portions were dark red and contained practically no air. A small amount of bloody-frothy fluid was expressible from the cut surface. The smaller bronchi were hyperemic. The major bronchi were not obstructed. Microscopically, all sections of the lungs showed intense intra-alveolar hemorrhage and capillary congestion, patchy pulmonary

edema, and a few areas of intense capillary congestion without intra-alveolar hemorrhages or edema. Expansion was incomplete in all segments. No bronchial plugs were seen. (See Figure 8A.)

Case 7.—W. C. L. (Pkd. W51671), a 52-year-old man, was admitted September 17, 1947, suffering from extrahepatic obstructive jaundice due to stone, and chronic cholecystitis with cholelithiasis. His blood pressure was 134/77, and pulse 89. There was mild emphysema. No preoperative fluids were administered.

Operation consisted of cholecystectomy and choledochostomy. Anesthesia was induced by sodium pentothal followed by cyclopropane, followed by ethylene, followed by ether in a closed circuit. Blood pressure and pulse were normal throughout the operative procedure. Post-anesthetic reaction was rapid and accompanied by excitement, and 500 ml. of 5 per cent glucose in water and 500 ml. of blood were given intravenously during the operation.

All went well for 4 days postoperatively, and, except for a mild nausea, progress was satisfactory. Because of the nausea intravenous fluids were given daily as follows:

Day of operation: 1300 ml. of 10 per cent dextrose in water; first postoperative day: 2000 ml. of 10 per cent dextrose in water; second postoperative day: 3000 ml. of 10 per cent dextrose in water; third postoperative day: 3000 ml. of 10 per cent dextrose in water; fourth postoperative day: 200 ml. of 10 per cent dextrose in water.

At 9:30 A.M. on the fourth postoperative day, while receiving the first 200 ml. of a projected dose of 2.5 liters of 10 per cent glucose in distilled water, he developed a chill. The infusion was discontinued, and the chill stopped soon thereafter. By 11:15 A.M. his mouth temperature had risen from 99° to 100.6°, his pulse rate was 150, and his respiratory rate was 40 per minute. At this time there was an episode of projectile vomiting, following which he complained of a feeling of substernal pressure. A peripheral pulse was not palpable. He became extremely dyspneic and his skin was ashen-grey. One and one-half hours later his skin was cool and dry, while his rectal temperature stood at 106.6° F. No blood pressure reading was obtainable. The right lung transmitted breath sounds poorly, but no mediastinal or tracheal shift could be detected. There was slight dullness over the right hemithorax. Roentgenogram revealed no evidence of atelectasis or other significant abnormality involving the chest. Oxygen was given by mask and one liter of blood was infused. No relief from the dyspnea and cyanosis was provided by the oxygen. His pulse had re-appeared before the blood transfusion, but thereafter he became pulseless and remained so for 5 hours, when he died.

Postmortem Findings. The lungs filled 70 per cent of the opened chest. They weighed 1135 Gm. and were red and firm throughout. The cut surface did not bleed readily. The anterior portions of the right lung were somewhat crepitant. The left lung was similar to the right lung but was non-crepitant. No pulmonary arterial thrombi or emboli were found. The bronchi were open.

Intra-aleolar hemorrhage, intra-alveolar edema, and intense capillary congestion were the microscopic characteristics of the posterior parts of the right lung. All segments of the left lung showed intense capillary congestion coupled with atelectasis without intra-alveolar hemorrhage or edema. Congestion of the hepatic sinusoids and the adrenal and renal capillaries were also found. A few old subendocardial microscopic infarcts were noted. The large coronary vessels were completely patent.

Case 8.—T. L., H-157-49, a 25-year-old man, was admitted January, 1950, with traumatic rupture of the third portion of the duodenum, pancreatic fat necrosis, necrosis of the inferior portion of the right rectus muscle, and retroperitoneal hemorrhage. His apical pulse was 180, radial pulse 90, and blood pressure 60/0. One liter of Hartmann's solution, 750 ml. of irradiated plasma, and 1000 ml. of blood were given intravenously.

At operation closure of the duodenum, and drainage of the left duodenal fossa and the space of Retzius were done and 500 ml. of blood were given during the operative procedure.

A continuous intravenous drip of blood was begun with the operation and continued for 11 hours. At 3:30 A.M., 5 hours postoperatively, his blood pressure was 96/70, his pulse was 132, and his respiratory rate was 42. By 8:00 A.M. (11 hours after the operation) he had received an additional two liters of blood. At 8:00 A.M. his blood pressure was unobtainable, and the rate of injection of the blood was increased. One liter of blood flowed in during the following hour, and his blood pressure rose to 85/0. The infusion was continued with 750 ml. of Hartmann's solution and 750 ml. of 10 per cent dextrose in water, and by 3:00 P.M. his blood pressure had risen to 125/80. His pulse rate had increased to 136 and his respiratory rate to 54 per minute and a deep resistant cyanosis had appeared. The blood pressure again began to fall and another liter of blood was injected intravenously, and the blood pressure became unobtainable while his respiratory rate climbed to 62 per minute. He died one-half hour later. Breathing was labored throughout the postoperative period. No râles were heard. With the repeated transfusions the red blood ecll count rose from 5.79 million to 8.82 million and the hemoglobin climbed from 18.5 Gm./100 ml. to 24 Gm./100 ml.

Postmortem Findings. (1) The right lung weighed 550 Gm. and the left lung 600 Gm. Both lungs were dark blue and airless. They felt solid and abundant blood could be expressed from their cut surfaces. The bronchi were filled with a hemorrhagic, fluid, frothy material. The pulmonary arteries were patent. One and one-half liters of a hemorrhagic fluid were present in each thoracic cavity. (2) The heart was normal. (3) There was extensive retroperitoneal edema with some hemorrhage. Microscopic findings consisted of an intense capillary congestion, incomplete alveolar expansion, and diffuse intra-alveolar hemorrhage characterized the pulmonary microscopic picture. No pulmonary edema existed.

The signs associated with the development of the congestion, intra-alveolar hemorrhage, and incomplete expansion of the lungs are listed in Table I. Some of the possible causes of the trouble may be adjudged from clinical correlations (see Table II). Physical injury, excessive fluid administration, anaphylactoid reactions, pre-existing shock, and rapidity of intravenous infusion appear to be contributory factors. More will be said about them in the discussion.

Thus far the attempts that have been made to reverse the process have been unsuccessful. Only one young, strong man recovered. Because of the discovery of cardio-pulmonary signs suggestive of congestive cardiac failure, preparations of digitalis acceptable for rapid digitalization were given to five of the eight; all of them died. Humidified oxygen was administered through an oro-nasal mask to everyone, but it did not clear the cyanosis and it did not save them. Phlebotomy was not attempted because it had been tried five years before and failed. Bronchoscopic aspiration was performed on five of the eight, and one of them recovered. However, the recovery was not dramatic, and a bronchial obstruction was not found; therefore it is possible that the bronchoscopy had little to do with his recovery. Blind tracheal aspiration was employed in two of the three others. Little fluid was removed, and they died. The breathing of oxygen under positive pressure was used in one instance (J.V.); it served to prolong her life but did not effect reversal of the process. Frequent turning, coughing, encouragement of deep breathing, and the inhalation of five per cent carbon dioxide in oxygen were tried.

DISCUSSION

Congestive atelectasis was suggested by E. E. Muirhead as the descriptive term best fitting the macroscopic and microscopic appearance of the lungs of these people. At the time he made the suggestion, he was not aware of the fact that Fegler and Banister had employed it earlier[10] (see introduction). It is remarkable that the diagnostic criteria adopted by Fegler and Banister, and Muirhead are practically identical.

TABLE I

			Respiratory Signs					
Case	A.G.	J.V.	H.J.	J.G.	R.F.	A.M.	W.C.L.	T.L.
Labored breathing	+	+	+	+	+	+	+	−
Tachypnea	+	+	+	+	+	+	+	+
Accentuation of expiratory effort	+	−	+	+	+	−	−	−
			Pulmonary Signs					
Case	A.G.	J.V.	H.J.	J.G.	R.F.	A.M.	W.C.L.	T.L.
Dullness	+	+	−	+	+	O	O	−
Diminished transmission of breath sounds	+	+	−	+	O	O	+	−
Râles	O early	+	+	O	+	+	O	O
Mediastinal cardiac and tracheal shift	O	O	O	O	O	O	O	−
O_2 Therapy used	+	+	+	+	+	+	+	+
Cyanosis not alleviated by the administration of oxygen	+	+	−	+	−	+	+	+
			Cardiovascular Signs					
Case	A.G.	J.V.	H.J.	J.G.	R.F.	A.M.	W.C.L.	T.L.
Hypotension	+	+	+	+	O	+	+	+
Tachycardia	+	+	+	+	+	+	+	+
Increased venous pressure	−	−	+	−	+	−	−	−
Enlargement of cardiac areas	−	+	+	−	−	+	−	−
Roentgenographic abnormality at time of earliest significant signs of pulmonary trouble	Haziness of right base	Elevation of hemidiaphragms only	No signs on the side of maximum trouble	Normal chest	Atelectasis lower lobe right homogeneous density over lower lung fields		Normal chest	
Temperature response	106° F (R)	−	O	103° F. (O)	102.6° F. (O)	105° F. (R)	106.6° F. (R)	102° F.
Outcome	Recovery	death	death	death	death	death	death	death

Legend: + = present; O = absent, and − = indeterminate from the record at the time of notation of the other listed signs.

Gross and microscopic descriptions of the phenomenon are numerous. Some are to be found in descriptions of "massive collapse of the lung."[2, 5, 17] The pathologic report of an instance of acute bilateral (non-obstructive) atelectasis by Symmers, Miles and McGrath, quoted by Bergamini and Shepard[2] is a good example:

"At autopsy—left lung collapsed; right lung partially collapsed, moderately engorged; no food in bronchi; tongue natural. Fairly well-marked laryngeal oedema.

The epiglottis was peculiar in shape; no obstruction by food. Heart and abdominal organs normal.

"Histological findings reported as follows by Doctors Symmers, Miles and McGrath: 'Examination of microscopic preparations from the collapsed pulmonary lobes reveals a tissue which it is difficult to recognize as lung, resembling rather a solid organ. This appearance is found to be due to complete atelectasis of the pulmonary alveoli, the epithelical cells of which lie closely packed together, having lost entirely their normal alveolar arrangement. The individual cells are swollen, certain of them being obviously hydropic, and the cell outlines are rather indistinct. The bronchioles are also collapsed for the most part, many of them being represented merely by circular clumps of cuboidal cells. The capillaries, arterioles and venules, on the other hand, are all uniformly dilated and filled with blood, producing almost an angiomatous appearance in certain areas. This constitutes the most characteristic feature of the histology of the condition.'"

TABLE II

Case	A.G.	J.V.	H.J.	J.G.	R.F.	A.M.	W.C.L.	T.L.
Pre-existing hypotension or shock...	no	yes	yes	no	no	slight	no	yes
Physical injury (operative included)	yes	no	yes	yes	yes	yes	yes	yes
Rapid intravenous injection of fluid	no	yes	no	yes	no	yes	no	yes
"Excessive" fluid given								
a. Blood	no	no	yes	no	no	yes	no	yes
b. Plasma	no	no	no	no	no	no	no	no
c. Saline	no	yes	no	no	yes	no	yes	no
d. Glucose in water	no	yes	no	no	yes	no	yes	no
Pre-existing extracellular fluid deficit	no	yes	no	no	no	no	no	yes
Anaphylactoid* reactions to the parenteral fluids	yes (glucose) Na-sulfadiazine	no	yes to plasma	yes to glucose in saline	no	no	yes to glucose in water	no
Pre-existing heart disease	no	no	no	no	no	no	old microscopic myocardial infarcts	no
Pre-existing pulmonary disease	no	no	no	no	yes	no	no	no
Hemo-concentration	no	no	no	no	no	—	—	yes
Hemo-dilution	no	yes	yes	yes	yes	—	—	no

* No living bacteria were found in any of the fluids producing the reaction.

Other descriptions of congestive atelectasis are to be found in discussions of the pathologic changes associated with rapid decompression,[4] and in the organ—descriptive literature of shock,[16] blast injury,[21] burns,[15] and the excessive transfusion of blood.[13]

The symptoms and signs of congestive atelectasis differ from those associated with obstructive atelectasis only in a few respects. The congestive type is attended by a cyanosis that is generally deeper and does not clear as readily when oxygen is breathed. The mediastinal structures do not shift appreciably with congestive atelectasis, but do with the obstructive. However, one cannot be sure about this because the congestive process was bilateral in everyone examined after death and therefore a mediastinal shift would not be as likely to occur as it would had the involvement been unilateral. The presence, location, and extent of obstructive atelectasis are fairly determinable with roentgen rays[1, 19, 20] but the determination of the presence and extent of congestive

atelectasis cannot be made early with them. In other words, obstructive atelectasis produces organic pulmonary changes detectable with roentgen rays, and early congestive atelectasis does not. In this series there is no correlation between the roentgenologic, the physical, and the postmortem examinations. The proved extent of pulmonary involvement was greater than the physical signs indicated, and the physical signs were more indicative of the extent of the process than the roentgen signs were.

The congestive and the obstructive types of atelectasis differ also in respect to the effectiveness of bronchoscopic and tracheal aspirations in their treatment; bronchoscopic and tracheal aspirations are very effective therapeutic measures for the latter type[12, 18] but apparently have little therapeutic effectiveness for the former.

The physical signs and symptoms of congestive atelectasis are sufficiently similar to those of congestive cardiac failure[19] to suggest that left heart failure might be its cause. If this were true, digitalization should have saved some of the five individuals to whom preparations of digitalis were given. One (H.J.) showed a dramatic, immediate response to Cedilanid. His blood pressure rose and his pulse and respiratory rates fell to normal within five hours —but he died soon thereafter with his alveoli and bronchi filled with blood. Digitalization was attended in two others (J.G. and A.M.) by a temporary return of blood pressure toward normal, but it had no appreciable effect upon the respiratory difficulties or the pulse rate, and they soon died.

It might also be said that congestive atelectasis is only one of the organic manifestations of shock.[16] As far as available experimental evidence is concerned that is not any more likely than the possibilities that it might be an attendant physiologic response or a cause of shock under certain circumstances.

These questions cannot be solved without recourse to active experimentation. Two exploratory approaches have been made. The first entailed the study of the effects of the infusion of fluids at rapid rates into normal dogs lightly anesthetized with sodium pentobarbital and into some suffering from "tourniquet shock." The physiologic variables recorded were: tidal air, intrapleural pressure and the jugular venous, pulmonary arterial, left auricular, and systemic arterial pressures.

Tidal air was recorded with a 40 Gm. counter-weighted Hutchinson spirometer of 1000 ml. capacity. The jugular and left atrial pressures were measured with saline manometers of 2 mm. bore that had been calibrated with a mercury manometer. The mean pulmonary and systemic arterial pressures were measured with damped mercury manometers. The chest and pericardium were opened for the insertion of the left atrial cannula and then they were closed. Intrapleural pressure was recorded with a water manometer connected to a special T cannula inserted through an intercostal space. The appearance of the lung was watched through a plexiglass window inserted into the right chest.

Figure 1 illustrates some of the effects of a rapid intravenous infusion of 0.9 per cent saline solution into a lightly anesthetized dog. Soon after the beginning of the infusion, the systemic blood pressure fell 20 mm., and the pulmonary arterial pressure rose eight. At the same time, the jugular venous and left atrial pressures rose two and one millimeters respectively. The infusion was stopped for 15 minutes and the systemic arterial pressure rose and the pulmonary arterial fell and continued to fall for a time after the infusion was started again. Peripheral venous pressure rose steadily, but the left atrial pressure fell and continued to fall even after the pulmonary arterial pressure began its second rise.

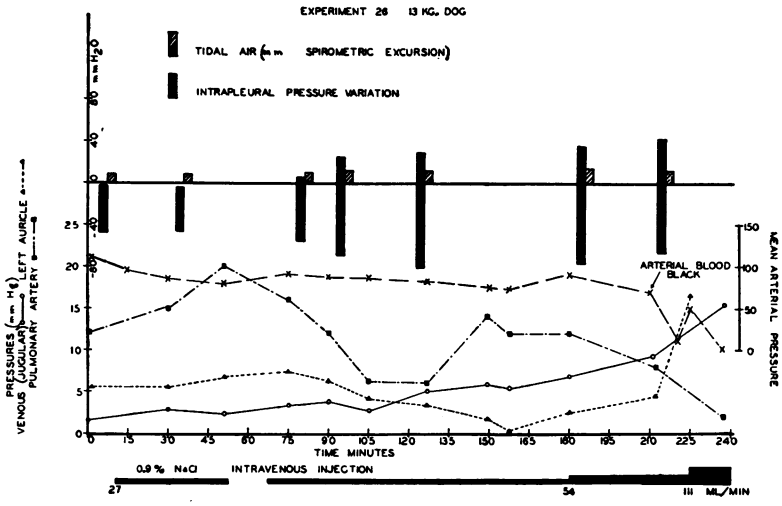

FIG. 1.—The effects of the intravenous infusion of 0.9 per cent solution of sodium chloride upon jugular venous, pulmonary arterial, left atrial, and femoral arterial pressures, tidal air, and intrapleural pressure variations in the anesthetized dog.

Coincident with the decline of the pulmonary arterial and left atrial pressures to levels below those of the pre-injection period, the expiratory intrapleural pressures became positive and the inspiratory became much more negative. At the same time tidal air increased very little. The association of a large increase in respiratory effort, as evidenced by the increase in the change of intrapleural pressure with each breath, with only a slight increment of tidal exchange, denotes an increase in the resistance of the lungs to deformation. In other words, the lungs were becoming stiff.

Thirty minutes before the animal died the rate of consumption of oxygen dropped precipitously (not shown in Figure 1), cyanosis appeared, the systemic and pulmonary arterial pressures fell sharply and the peripheral venous and left atrial pressures rose rapidly. The elevation of venous and left atrial pressures to a level higher than the pulmonary arterial is inexplicable. It is

only seen just before death and after death all pressures quickly reach identical levels. However, before the systemic arterial pressure fell, the lung beneath the observation window became deeply congested and remained so until the animal died.

The lungs were removed after closing the trachea and they barely floated in water. Over 90 per cent of them looked like liver. A cannula was inserted into the trachea and air was introduced through it, and the carnification disappeared. The microscopic examination of a small section of lung removed before the inflation showed intense capillary congestion, intra-alveolar hemorrhage, large areas of incomplete expansion of alveoli, and a little edema (see Figure 2).

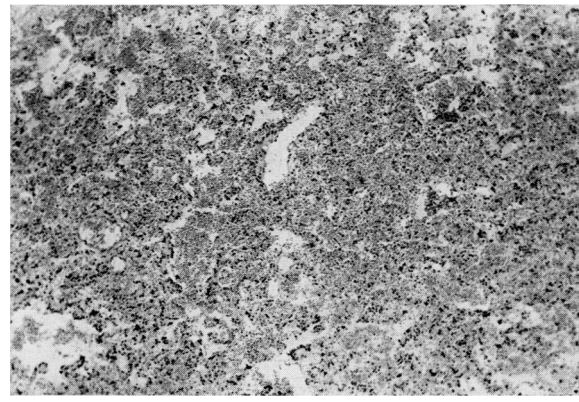

FIG. 2.—Congestive atelectasis produced by the intravenous infusion into an old anesthetized dog of 0.9 per cent sodium chloride at the rate of 45 ml. per minute for 20 minutes.

Other animals given saline rapidly developed fulminant pulmonary edema with practically no congestion. The question "Why do some animals when given saline rapidly die with pulmonary congestion, atelectasis and little edema and others with pulmonary edema without appreciable pulmonary congestion?" cannot be answered. The most significant observation in experiment 26 was the continuous development of pulmonary stiffness *while left atrial pressure was falling*. Therefore, the usual explanation for stiffening of the lungs while saline solution is injected rapidly intravenously, namely, that the left ventricle has been overloaded and fails, is untenable. How can one believe that back-pressure caused by failure of the left heart produces the stiff lung in experiment 26 when the left atrial pressure falls as the lung stiffens?

When the circulation to the hind legs of the animals was cut off for an hour and a half by the application and removal of tourniquets, the responses to the rapid intravenous injection of saline were somewhat different from that shown in Figure 1 (see Figures 3 and 4).

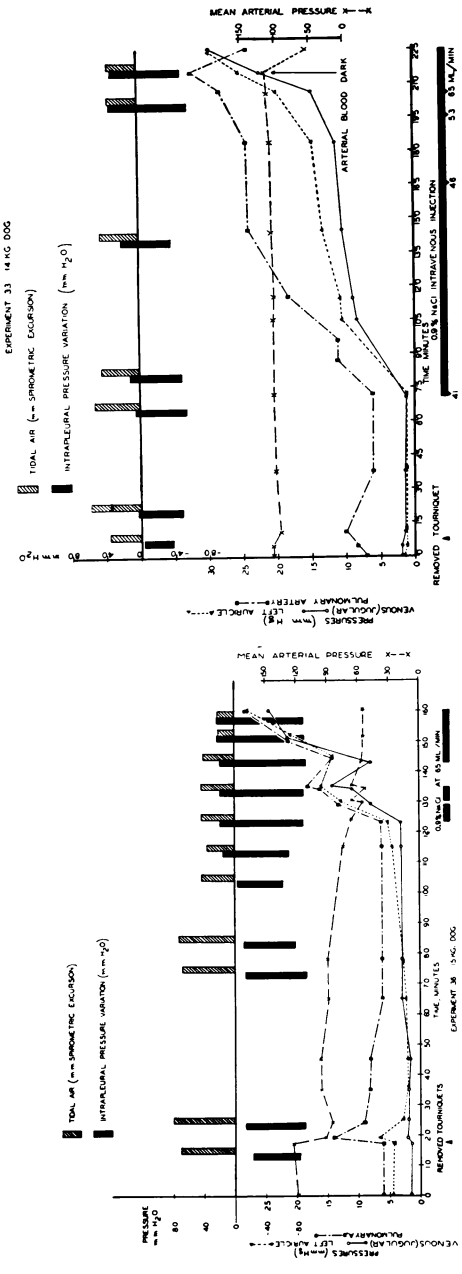

FIG. 3

FIG. 4

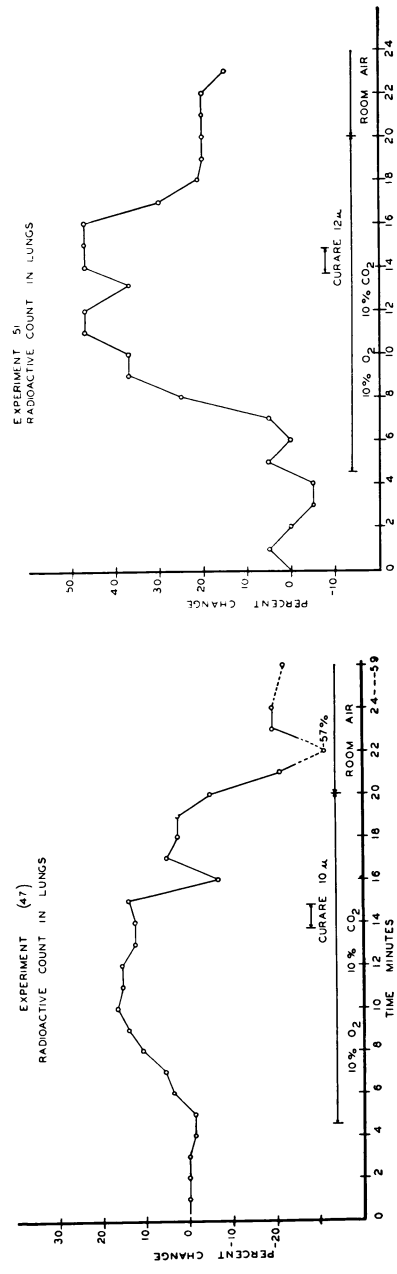

FIG. 5

FIG. 6

FIGS. 3 and 4.—The effect of tourniquet shock and the intravenous infusion of saline solution upon various vascular pressures, tidal air, and intrapleural pressure variations in the anesthetized dog.

FIGS. 5 and 6.—The effect of combined hypercarbia and hypoxia upon pulmonary blood volume of the anesthetized dog, using red blood cells tagged with P^{32}.

The volume of air moved per breath relative to the effort expended in moving it fell and the expiratory intrapleural pressure became positive before the saline was injected. In addition, the lung beneath the window became visibly hyperemic before the infusion was started. After the infusion was started, breathing effort increased quickly and the pulmonary arterial, peripheral venous and left atrial pressures rose rapidly with similar rates of change while systemic arterial pressure which previously had been falling slowly, fell fast during the infusion and rose during the two short periods that the injection was stopped. These pressure changes associated with infusion after tourniquet shock has developed are entirely compatible with backward heart failure.

The sequestration of extracellular fluid in the legs after the removal of the tourniquets is in part the cause of tourniquet shock, and saline solution administered at moderate rates of speed is known to be effective in treating it. However, when it is given very rapidly during tourniquet shock it aggravates the hypotension and presumably induces heart failure.

The observance of hyperemia and a progressive stiffening of the lung as the shock developed before fluid was injected in experiment 36 is indicative, at first thought, that shock is the cause of the pulmonary hyperemia and stiffening. However, the expiratory intrapleural pressure rose quickly toward atmospheric level within six minutes after the removal of the tourniquets, and during experiment 33 (Fig. 4) it was actually positive five minutes after their removal. These observations indicate that at least the lung stiffening is an attendant phenomenon and not the resultant of tourniquet shock. After death, the lungs of dogs 33 and 36 were found to contain air only in anterior segments; the middle and posterior segments showed the macro- and microscopic appearance of congestive atelectasis. Microscopically, the anterior segments were congested and edematous but not atelectatic.

After a year of work, it was realized that the answers which were sought could not quickly be found employing the technics illustrated in Figures 1, 3 and 4 and a more direct approach had to be made.

The changes in the pulmonary blood volume had to be measured in the intact animal. In collaboration with A. Reid, Professor of Biophysics, a fairly direct method was worked out using red blood cells tagged with P^{32}. An aluminum channel, the inner orifice of which was covered with an airtight layer of plastic, was inserted through the chest wall and so adjusted that after the withdrawal of the pneumothorax the inner surface of the channel was constantly in contact with the lung.

A shielded counting tube was attached to the outer end of the channel, making possible a continuous observation of the number of red blood cells within a small segment of lung. Because the emission of beta particles is a function of time, only variation in the volume of blood within the lungs is detectable; changes in the rate of flow alone do not affect the count,

Figures 5, 6 and 7 are examples of consistent changes in the volume of blood in the lung attendant upon controlled asphyxia and hemorrhage. The rapidity with which and the extent to which blood cells collect in the lung following hemorrhage is remarkable. The hemorrhage illustrated in experiment 8 amounted to 175 ml. and was sufficient to lower the mean arterial pressure to 70 mm. of mercury. The rise in pulmonary blood volume attendant upon hemorrhage takes place too quickly to ascribe it to shock.

Controlled asphyxia (10 per cent O_2, and 10 per cent CO_2 in nitrogen) is also attended by speedy and significant increments of blood cells in the lung. Earlier studies of Drinker, Churchill and Ferry[9] failed to demonstrate any significant change in the volume of blood in the lungs with hypercarbia and hypoxia; however the peripheral blood circuit was excluded in their animals.

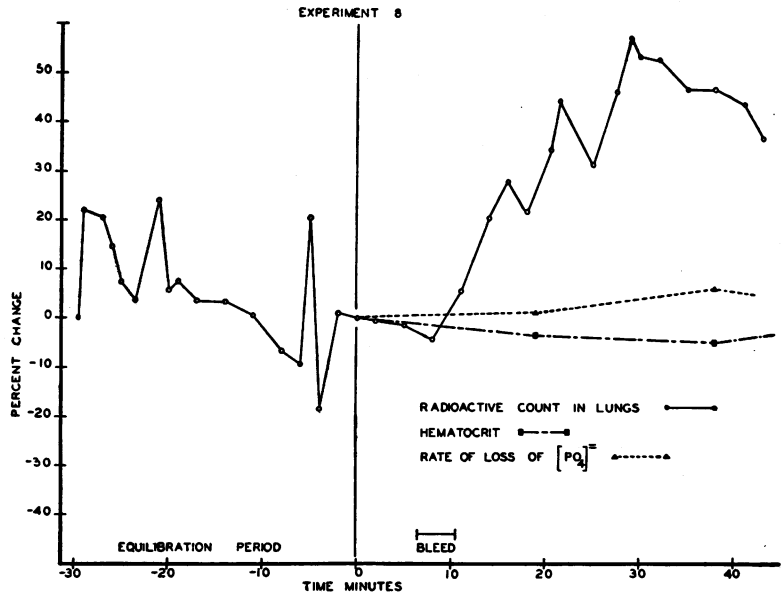

FIG. 7.—Change in the volume of blood in the lungs of the anesthetized dog following a hemorrhage of 175 ml.

The use of radioactive red cells alone does not permit the determination of the changes in the volume of blood in the lungs associated with the intravenous infusion of various fluids with sufficient certainty to permit us to report them now. Random changes in the distribution of P^{32} have been found during the infusion of saline solutions, and because of them methods for the simultaneous determination of the volumes of fluid and the volume of red cells must be developed before one can make accurate determinations of changes in the volume of blood in the lungs associated with the infusion of fluids. The development of such methods now appears to be possible.

It is apparent that the experimental approach has not clarified the etiology of non-decompressive congestive atelectasis. However it has contributed

FIG. 8.—(A) Magnification X 140. Congestive atelectasis. Man (A.M.), case summary in text. (B) Magnification X 225. Congestive atelectasis. Dog (2) anesthetized; saline given intravenously.

FIG. 9.—(A) Magnification X 35. Congestive atelectasis. Man (H.J.), case summary in text. (B) Magnification X 38. Obstructive atelectasis, aspiration; a child (N.O.).

something to our understanding of the phenomenon. It has shown: (1) that the volume of blood in the lungs increases rapidly following hemorrhage and asphyxia; (2) that the relationship of pulmonary arterial pressure to left atrial pressure is remarkably altered immediately following the release of arterial occlusive tourniquets (see Figures 3 and 4); (3) that stiffening of the lung begins soon after the injection of saline solution, and occasionally

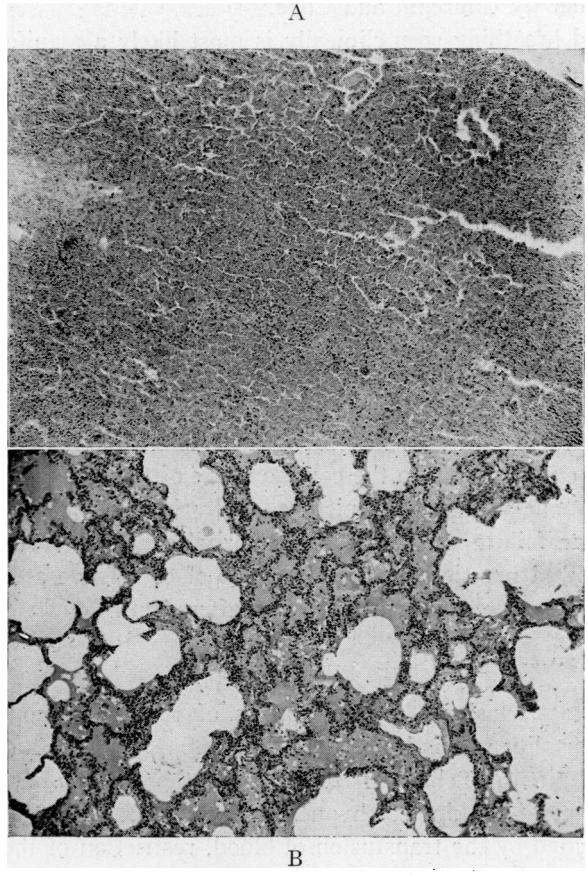

FIG. 10.—(A) Magnification X 45. Pulmonary infarction, embolism; a man (P.B.). (B) Magnification X 38. Pulmonary edema, cardio-renal failure; a woman (N.P.).

becomes very pronounced in the non-shocked animal while left atrial pressure is falling (this rules out failure of the left ventricle as the universal cause of pulmonary-stiffening); (4) that stiffening of the lung appears soon after the release of arterial occlusive tourniquets and becomes pronounced by the time shock is well established; and (5) that the rapid injection of saline solutions during tourniquet shock, when the lungs have become stiff, quickly causes changes in vascular pressures that are compatible with acute heart failure, and the severity of the shock is increased.

The correlation of clinical experience with experimental experience has served to clarify partially the signs of congestive atelectasis. The very rapid breathing which attends the onset of congestive atelectasis is due evidently to the congestion, and is of reflex origin. Blocking the vagus nerves during congestive tachypnea abolishes it (this observation is not illustrated). The dependence of congestive tachypnea upon pulmonary proprioceptive reflexes was discovered earlier by Churchill and Cope.[7]

The labored breathing seen clinically is most likely a manifestation of the stiffening of the lung. The bronchoscopically visible inward expiratory bulging of the trachea and bronchi associated with congestive atelectasis is likely attendant upon the development of positive intrapleural pressure during expiration, coupled with sufficient congestion of the lung so as to render inward deformation of the larger air passages easier than the deflation of the extrabronchial pulmonary parenchyma.

The resistant cyanosis is manifestly caused by the continuation of a fairly rapid flow of blood through areas rendered airless by the positive increment of blood in the pulmonary capillaries and alveoli.[3] In this respect, congestive atelectasis differs from obstructive atelectasis.[3, 8, 11]

Finally, attention should be called to the almost complete correlation of the clinical pictures of congestive atelectasis and pulmonocardiac failure.[6] The similarity extends even to the common lack of responsiveness to digitalis and to the pulmonary pathologic picture. A kyphotic woman suffering from cardiopulmonary failure whom I had the privilege of observing before and after death showed practically all of the signs of congestive atelectasis before death, and the complete pulmonary picture after death.

SUMMARY

Congestive atelectasis is a complication of the intravenous infusion of fluids. The mortality rate is high.

The clinical picture is characterized by the rather sudden onset of dyspnea, labored expiratory breathing, tachypnea, tachycardia, fever, hypotension that is often aggravated by the transfusion of blood, restriction of thoracic motion, dullness over the involved portions of the lung, the practical absence of roentgen ray signs early, and resistant cyanosis.

Organically, the involved parts of the lung are carnified, but when inflated with air under pressure they inflate and appear grossly normal. Microscopically the process is characterized by capillary congestion, intra-alveolar hemorrhage with little edema, incomplete expansion of alveoli, and compression closure of bronchioles.

The clinical and pathologic pictures and the clinical course of congestive atelectasis is strikingly comparable to pulmonocardiac failure. Even the ineffectiveness of digitalis as a therapeutic agent is common to both syndromes.

Two experimental approaches to the problem have been developed. A preliminary report on some of these studies is made.

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