Correlations Between the Chest Film and Hemodynamics in Acute Myocardial Infarction

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SUMMARY
A series of 169 standardized chest films was analyzed relative to hemodynamic parameters obtained within 2 hr of the roentgenogram in 86 patients with acute myocardial infarction. The films were evaluated for cardiomegaly using the cardiothoracic ratio and the standardized, external left heart dimension. The radiographic appearance of the pulmonary vasculature was divided into levels of increasing severity: normal, pulmonary venous congestion, interstitial pulmonary edema, alveolar pulmonary edema. These were compared with wedge pressure levels of <12, 13-18, 19-25, and >25 mm Hg, respectively. Correlations with the admission films of the 86 patients showed: 1) The appearance of the pulmonary vasculature accurately predicted the patient's wedge pressure on admission in 43% of the cases, overestimated it in 33% and underestimated it in 24%. 2) The presence of cardiomegaly and/or pleural effusion indicated an elevated wedge pressure with a high degree of certainty; however the absence of these signs did not exclude an elevated wedge pressure. 3) In only 62% of the studies was the chest film able to detect or exclude the presence of pulmonary venous hypertension. However, when wedge pressures were elevated to 19-25 mm Hg and over 25 mm Hg, 74% and 100% respectively of roentgenograms had some evidence of pulmonary venous hypertension. 4) There was a high degree of correlation between the patient's admission physical findings, as evidenced by clinical classification, and the radiologic assessment of pulmonary venous hypertension.

There was no statistical variation in the accuracy of the estimate of wedge pressure in relation to the time elapsed from onset of symptoms of myocardial infarction. However, a therapeutic phase lag was encountered in 21 patients, the chest films remaining abnormal for a period of 1 to 4 days following return of the wedge pressure to normal. A diagnostic phase lag was encountered in 6 patients in whom the chest film did not correlate with elevation of wedge pressure for up to 12 hr. Furthermore, in 17 patients the films were normal in spite of a persistently elevated wedge pressure for 6 to 24 hr.

These studies document the limitations of the chest roentgenogram in predicting the hemodynamic status of patients with acute myocardial infarction.

Additional Indexing Words:
Pulmonary artery wedge pressure Pulmonary hypertension Pulmonary edema
Hilar angle Kerley's "B" lines Kerley's "C" lines

The CHEST roentgenogram has been used effectively to assess hemodynamic status and to follow the clinical course of patients with longstanding cardiac disease. Changes in the appearance of the pulmonary vascular bed on both sides of the pulmonary capillaries have been evaluated in attempts to quantify the severity of pulmonary

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Received March 22, 1973; revision accepted for publication May 4, 1973.

Circulation, Volume XLVIII, September 1973
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arterial and pulmonary venous hypertension.1–6 That the presence of altered hemodynamic patterns can be recognized roentgenographically is undisputed, but the accuracy with which their severity can be estimated is controversial, and most studies have been concerned with correlations between hemodynamic parameters and changes in the chest film in patients with chronic mitral valve disease. Several authors have extrapolated these data and methods to the acute setting and have proposed methods for radiologic evaluation of the hemodynamic status of patients with complications of acute myocardial infarction.7–8

The availability of flow-directed cardiac catheters has now made it possible to obtain considerable hemodynamic information in patients with acute myocardial infarction.9,10 Several studies of relatively small groups of patients have appeared in which correlations were attempted between the acute hemodynamic status of the patient and a concurrent chest roentgenogram.11–13 However, the lack of conclusive and consistent findings has prompted us to make a detailed analysis of chest roentgenograms in a group of patients with acute myocardial infarction and to correlate them with concurrently obtained hemodynamic data.

Methods

Admission and follow-up chest roentgenograms were obtained in 86 patients with acute myocardial infarction admitted to the Myocardial Infarction Research Unit. The diagnosis was established on the basis of at least two of the following criteria: 1) a history of prolonged, typical chest pain; 2) electrocardiographic changes indicative of acute myocardial injury; 3) characteristic serial elevations of serum enzymes (creatinine phosphokinase, serum glutamic oxaloacetic transaminase and/or lactic dehydrogenase). Patients were classified clinically by the Myocardial Infarction Research Unit (MIRU) as follows: Class I: (uncomplicated) no clinical signs of heart failure. Class II: (borderline) orthopnea, dyspnea, sinus tachycardia between 110 and 140 beats/min, other signs of mild failure, or evidence of significant prior cardiac disease. Class III: (complicated) pulmonary edema or a third heart sound as well as signs of low cardiac output. Class IV: shock.

Pulmonary arterial (PA) systolic, diastolic and mean, and wedge pressures were obtained simultaneously or within two hr of the chest films, utilizing a flow-directed pulmonary artery balloon catheter.9 (In several patients, when PA wedge pressure [PAW] was not obtained, PA end diastolic pressure was used.) The pulmonary pressures were recorded via Microdot transducer on a Honeywell recorder; zero reference level was at the midchest.

Postero-anterior chest films were exposed at end-diastole with the patient supine or in a 45° reclining position and with inspiration to one liter from functional residual capacity, using a ceiling mounted C-arm radiographic system with fixed tube film distances of 36 inches.14 The films were taken on admission to the unit and at 12 to 24 hr intervals thereafter for two to four days.

A total of 169 films were interpreted independently by two observers without knowledge of the patients' clinical or hemodynamic status. The admission chest roentgenogram of each patient was evaluated independently without resort to any other films. For evaluation of subsequent films during the patient's course, the earlier films were used as a basis of comparison.

Radiological Parameters

Patients were classified as having radiologic signs of pulmonary venous hypertension when they had one or more of the following changes: 1) Pulmonary venous congestion. This was defined as a disparity between the caliber of the upper and lower lobe pulmonary vessels, with distention of the upper lobe vessels.15 Without a previous “normal” film for comparison, diffuse pulmonary engorgement could not always be identified with certainty. 2) Interstitial pulmonary edema was diagnosed if one of the following radiological signs was present: a) septal (Kerley’s “B”) lines; b) perivascular edema (manifested by “hilar clouding” and loss of definition of pulmonary vascular markings),16 c) poorly defined diffuse reticular pattern (Kerley’s “C” lines).5 3) Alveolar edema was defined as confluent shadows of no specific distribution, more or less uniform in density with ill-defined margins.17 The “bat’s-wing” pattern of edema was rarely seen. Serially these shadows frequently changed their appearance and distribution. When more than one of these patterns presented simultaneously, the patient was designated as having the most severe pattern.

The pulmonary artery pressure was estimated indirectly as follows: 1) MPA/chest ratio (MPA = main pulmonary artery); this ratio was defined as 100 times the distance from the body midline to the most lateral aspect of the pulmonary outflow segment divided by the transverse diameter.12 2) The width of the descending right pulmonary artery; this vessel was measured at its widest point near the bifurcation of the artery to the lateral segment of the right middle lobe and above the branching of the middle basilar artery.18 Often the lateral wall of the air-filled right main stem bronchus was used as the medial boundary of this vessel (fig. 1).

The presence or absence of a pleural effusion was recorded.

Cardiomegaly was evaluated on the basis of: 1) The cardio-thoracic (CT) ratio. A ratio greater than .50 was abnormal. 2) The left heart dimension (LHD); this value has been defined as the distance from the midline of the anterior chest to the apex of the left ventricle corrected for magnification and normalized for body surface area.14 An LHD of 53 mm/M² or greater was considered abnormal.

The appearance of the hilar angle19 was noted on each study. The hilar angle, formed by the intersection of the right upper lobe pulmonary vein and the right
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Figure 1
This illustrates the methods by which radiologic measurements were made on the chest film. The anterior mid line was designated by connecting the images of lead markers placed on the skin (the superior marked midline between the clavicles and the inferior marker at the zyphoid). MPA = main pulmonary artery; LHD = left heart dimension; \( \frac{1}{2} \) TTD = half transsthoracic diameter; HA = hilar angle; DPA = descending pulmonary artery.

descending pulmonary artery, is normally acute. Elevation of pulmonary venous pressure and concomitant dilatation of the pulmonary vessels alters or obliterates this angle. Its appearance was recorded as being acute, obtuse or straight, reflecting increasing pulmonary venous pressure.

Hemodynamic Parameters
PA wedge pressure values were divided arbitrarily into four categories as follows: 12 mm Hg or less was normal, 13 to 18 mm Hg was mild venous hypertension, 19 to 24 mm Hg moderate, and greater than 25 mm Hg marked venous hypertension.

A given interpretation of a chest film was designated as an accurate assessment of the category of the pulmonary wedge pressure provided: 1) alveolar pulmonary edema was present and the PAW was more than 25 mm Hg; 2) interstitial edema was present and the PAW was 19-25 mm Hg; 3) pulmonary venous congestion was present and the PAW was 13-18 mm Hg; or 4) both were normal. Systolic, diastolic, and mean pulmonary arterial pressures also were obtained and were compared with measurements made on the concurrent film by placing the data points on an x-y plot, with measured values on the X axis and estimations from the roentgenograms on the Y axis.

The roentgenographic signs were correlated with hemodynamic data as follows: 1) The value of the initial chest film for estimation of cardiovascular function was analyzed by correlating it with the initial hemodynamic data. 2) The ability of the chest film to reflect a rapidly changing hemodynamic situation was evaluated by comparing all films with the hemodynamic values obtained concurrently, regardless of the temporal relation to admission or the onset of symptoms. 3) The chest film-hemodynamic correlation was evaluated in relation to the period of time which had elapsed since the onset of symptoms, to determine if there was a point in the clinical course when the reliability of the chest film was maximum.

Results
Admission Radiologic Findings versus Wedge Pressure
In 37 patients (45%) the pulmonary vessels and lungs were normal. In 49 patients (55%) the lungs showed abnormalities attributed to venous hypertension. One patient showed congestion of the pulmonary veins, 32 had interstitial pulmonary edema (38% of the total patients), and 16 had alveolar edema (19%). Measured wedge pressure was normal in 32 patients and elevated in 54.

The initial chest roentgenogram accurately detected the presence or absence of an elevated pulmonary wedge pressure in only 62% of cases (53

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patients) (fig. 2). Of the 32 patients with normal wedge pressure, there was radiographic evidence of pulmonary venous hypertension in 14 (44%). Conversely, of the 54 patients with elevated wedge pressure, chest films were normal in 19 (35%). However, the reliability of the initial chest roentgenogram for detection of venous hypertension was found to increase as the degree of wedge pressure elevation increased. Twenty-three patients were admitted with wedge pressures from 13 to 18 mm Hg, and ten of these patients (43%) had an abnormal chest film. Of 23 patients with wedge pressures from 19 to 25 mm Hg, 17 (74%) exhibited radiographic evidence of venous hypertension. All films of the 8 patients with wedge pressures greater than 25 were abnormal.

The reliability of the chest film for predicting the degree of wedge pressure elevation was also evaluated. Interstitial edema was present on the initial film of 32 patients, 18 of whom had a wedge pressure less than or equal to 18 mm Hg (56% false positive). Conversely, in 6 patients the film was normal in spite of a wedge pressure greater than 18 mm Hg. Alveolar edema was identified in 16 patients, 10 of whom exhibited a wedge pressure of less than 25 mm Hg (63% false positive). Moreover, 2 of the 8 patients with a wedge pressure greater than 25 did not show alveolar edema (fig. 3). There was a statistically significant relationship between the height of the wedge pressure and the presence or absence of a pleural effusion ($P < 0.03$). Eleven of the 13 patients with an effusion on admission had a wedge pressure greater than 12 mm Hg, 9 of the effusions being present in patients with pressures $> 18$ mm Hg.

There was no statistical correlation between the relative size of the cardiac silhouette and the wedge pressure on admission. However, of the 28 patients with a CT ratio $> 0.5$, 27 had a wedge $> 12$ mm Hg on admission. Similarly, 30 of the 33 patients with an LHD of 53 had wedge pressures over 12 mm Hg.

There was no correlation between the configuration of the hilar angle and the admission wedge pressure ($P > 0.2$).

**Clinical Classification and the Chest Film**

There was a statistical relationship between the MIRU classification on admission and the severity of pulmonary venous hypertension: venous congestion correlated quite closely with MIRU Class II ($P < 0.001$); interstitial edema correlated as closely with MIRU Class III ($P < 0.001$); alveolar edema correlated equally closely with Class IV patients ($P < 0.001$) (fig. 4).

**Correlation of Hemodynamic Data and Chest Films in All Studies**

Of 194 films taken at all times during the initial admission, 169 were judged adequate for detection or exclusion of pulmonary venous hypertension and correlation with the wedge pressure.

**Wedge Pressure vs Radiologic Findings**

The accuracy of the film for detection of the presence or absence of elevated wedge pressure in the entire series was similar to that for the

**Figure 2**

A graph plotting radiographic appearance of the vascularity on the initial film vs pulmonary wedge pressure.

**Figure 3**

Relationship between radiological assessment of venous hypertension and measured wedge pressures. The diagonal where there is good correlation between the two parameters is highlighted.
admission films. Of the 169 observations, 102 were either normal when the wedge pressure was normal or abnormal when the wedge pressure was elevated. Thus, the overall accuracy of the chest roentgenogram in reflecting the presence or absence of an elevated pulmonary wedge pressure was 60%. Of the 79 determinations of normal wedge pressure, there was radiographic evidence of pulmonary venous hypertension in 32 (41%). Of the 90 instances where wedge pressure was elevated, films were normal in 35 (39%). As in the evaluation of the accuracy of the initial chest film, the accuracy of the entire series increased with increasing measured wedge pressure. Twenty-six of the 35 films taken at the time when the wedge pressure was determined to be between 19 and 25 mm Hg exhibited interstitial and/or alveolar edema and 10 of the 14 films taken at the time that the wedge pressure was found to be greater than 25 mm Hg indicated alveolar edema.

In the entire series, the appearance of the pulmonary vasculature correctly indicated the degree of pulmonary venous hypertension in 43% of the studies. The degree of pulmonary venous hypertension was overestimated in 34% and underestimated in 23% of the studies.

There was a statistical correlation between the MIRU clinical classification and the radiologic estimate of venous hypertension in the entire series, as was encountered on analysis of admission films ($P < .001$).

**Wedge Pressure vs Radiologic Findings in Relation to Time Elapsed from Onset of Symptoms**

The total series was separated into groups according to the time from onset of symptoms as follows: Less than 6 hr, 6 to 12 hr, 12 hr to 24 hr, 24 hr to 2 days, 2 to 3 days, and 3 to 4 days (fig. 5). Few observations were made within the first 12 hours after onset of symptoms. This is in keeping with the well known lag between onset of symptoms and hospitalization of patients with myocardial infarction. The accuracy of the film as a reflection of wedge pressure varied from 40% to 50%. The percentage overestimation and underestimation also remained within the range encountered in the previous analyses.

**Changes in Hemodynamics vs Radiographs—Time Course Followed in Individual Patients**

In all patients in whom a series of films and concurrently obtained hemodynamics were available, the change in each parameter was plotted against time. Six patients demonstrated a 12 hr lag before the radiographic findings became abnormal in spite of an elevated wedge pressure (fig. 6). In 17 other patients, however, the film remained normal in spite of a wedge pressure which remained persistently elevated for 6 to 24 hr (fig. 7). In 21 patients, the fall in wedge pressure was not reflected immediately on the film, but required from 1 to 4 days for complete clearing (fig. 8).

**Estimation of Pulmonary Artery Pressure**

Of the 169 films, 119 were satisfactory for evaluation of the size of the main pulmonary artery and calculation of an MPA/chest ratio, and/or measurement of the diameter of the right descending pulmonary artery. There was no significant correlation between the MPA/chest ratio and the mean pulmonary artery pressure measured on the
admission film or in the entire series. (The correlation coefficient of the MPA pressure and MPA/chest ratio of all films was −0.109). Similarly, a poor correlation was present between the mean pulmonary pressure and the diameter of the right descending pulmonary artery (correlation coefficient 0.37).

**Discussion**

The chest roentgenogram has been helpful in evaluating the degree of pulmonary venous hypertension in patients with mitral valve disease. A sequence of changes in the pulmonary vasculature has been observed, namely pulmonary venous engorgement, interstitial edema, and alveolar pulmonary edema. The appearance of the pulmonary vasculature has been thought to reflect the severity of the elevated pulmonary capillary wedge pressure in these patients.

The early changes in the pulmonary circulation in pulmonary venous hypertension have previously been documented. A redistribution of blood flow
occurs in this early phase with an increased blood flow through the upper lobe vessels and a diminution in the blood flow through the lower lobes. The chest roentgenographic changes which reflect this altered physiology are said to be generalized pulmonary venous engorgement and a disparity between the dilated upper zone vessels and constricted lower zone vessels. The level of wedge pressure at which lower zone venous constriction occurs has been placed somewhere between 12 and 18 mm Hg. A similar correlation has not been found concerning dilatation of the upper lobe vessels.

Transudation of plasma from the capillaries into the interstitial space occurs when capillary pressure exceeds the colloid osmotic pressure of blood (25 mm Hg). One manifestation of this is Kerley “B” lines, which are usually said to appear with a minimal mean left atrial pressure of 18 mm Hg. Grainger found them to be present when the wedge pressure exceeded 20 mm Hg. In contrast, Melham, Dunbar and Booth found “B” lines in patients with pressures as low as 14 mm Hg.

Other signs of interstitial edema, including the loss of definition of the hilar and peripheral pulmonary vessels, perivascular and peribronchial cuffing, and peripheral clouding have not been correlated with hemodynamic data. The range of 18 to 25 mm Hg has been suggested by Simon for the appearance of these early signs of interstitial pulmonary edema.

Alveolar pulmonary edema occurs when pulmonary wedge pressure greatly exceeds the colloid osmotic pressure of plasma. Although interstitial fluid is present at this time, the great majority of edema is in the alveoli. The critical wedge pressure necessary for the appearance of alveolar pulmonary edema has been postulated as 22 to 25 mm Hg by McHugh et al. and by others as 25 to 35 mm Hg. The appearance and resolution rates of alveolar edema are dependent upon several factors: the thickness and permeability of the alveolar wall and adjacent connective tissue, the rapidity of onset of the venous hypertension, the patency and absorptive capacity of the pulmonary lymphatics, and the position of the patient.

Chest roentgenograms have frequently been used as a method of evaluating congestive heart failure in patients with acute myocardial infarction. Several series have been published in which there is good correlation between the hemodynamics and the chest film following acute myocardial infarction. All of the patients reported by Fluck et al. manifested pulmonary edema and elevated pulmonary pressure on admission. The pressure fell to normal, followed by resolution of the pulmonary
edema. Kirby reported good correlation in two patients with acute myocardial infarction, and Nixon in one, between chest radiographs and left ventricular end diastolic pressure.

Recently McHugh et al. found excellent correlation between the radiologic signs of pulmonary venous hypertension and the hemodynamics. They found a 90% correlation between the level of pulmonary wedge pressure and the radiologic degree of left ventricular failure in 30 patients. They did, however, report serious discrepancies in 10% of the cases. Although a good correlation between the radiographic signs and mean pulmonary wedge pressures was found by Lassers et al., there was considerable overlap in their series and they cited three patients with pulmonary edema and pressures of 15 mm Hg or less. Nixon and Durh, reporting on a single patient with acute myocardial infarction, found severe pulmonary edema with normal left ventricular end diastolic pressure.

It is somewhat difficult to compare the data of the present series with previous reports because of the subjective nature of film interpretation. We found, as did Harrison, Conte and Heitzman, that pulmonary venous distention could not be used as a reliable index of acute cardiac failure. It is difficult to obtain a film in a patient with acute myocardial infarction of quality sufficient to evaluate the major pulmonary vessels because of the poor degree of inspiration, respiratory symptoms, and the general clinical situation. Furthermore, we experienced some difficulty in the detection of interstitial edema, due in part to the suboptimal film quality. However, interstitial fibrosis was found in several patients to masquerade as interstitial edema. Only after evaluation of the series of films over several days was it possible to detect this error.

The major cause for the lack of correlation between the chest roentgenogram and hemodynamic state in acute myocardial infarction probably lies in the rapidity with which the hemodynamic changes take place, and the inability of chest radiographic changes to reflect this faithfully. A "post therapeutic phase lag" has been demonstrated by McHugh et al. This refers to the time interval required for the chest film to become normal after return of an elevated wedge pressure to normal. Examination of the serial films in our series reveals 21 patients who exhibited this phenomenon. In this group, the fall in wedge pressure was not reflected immediately radiographically, but up to four days was required to demonstrate the clearing. This time interval is greater than the 1/2 to 21 hr mentioned by McHugh et al., but corresponds to the findings of Fluck. This posttherapeutic phase lag undoubtedly occurs because of the time required for the edema fluid to be resorbed after return to normal wedge pressure. It is conceivable that if one were able to follow more closely and accurately changes in the pulmonary vascularity, better results might be obtained.

In this series we encountered a second type of lag, namely a "diagnostic lag" which was responsible for further lack of hemodynamic-roentgenographic correlation. In this group of patients, a clearly documented elevation in wedge pressure was not reflected immediately on the radiograph; instead there was 12 hr lag before the film reflected the abnormality. This phenomenon may also be explained by the finite period of time required for pulmonary edema to accumulate, either in the interstices or the alveoli, after elevation of the wedge pressure secondary to left ventricular failure. Furthermore, a substantial quantity of edema fluid is most likely required for radiographic visualization.

During the acute phase of myocardial infarction with rapidly changing hemodynamics, one would expect the chest film to be relatively inaccurate because of the above-mentioned mechanisms. Given a lag of up to 12 hr for the radiographic manifestations of moderate left ventricular failure to occur and a lag of up to 4 days for these signs to clear, it is quite possible for a patient to be at an unknown point on two diverging curves in his clinical course, which can lead to serious misinterpretations. Specifically, the left ventricular decompensation which occurs with the acute myocardial infarction may require up to 12 hr to manifest itself radiographically. If the patient receives immediate successful therapy of this event, the wedge pressure could then return to normal at about the time the pulmonary edema appears radiographically, although signs of the edema could then persist on the film for up to four days.

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Circulation. 1973;48:624-632
doi: 10.1161/01.CIR.48.3.624

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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