The Mechanism of Arterial Hypoxemia in Acute Myocardial Infarction

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SUMMARY
Arterial blood gases were measured before and during oxygen breathing in 34 patients with acute myocardial infarction of whom 12 exhibited the shock syndrome, 14 showed evidence of left ventricular failure, and eight had no complications. Initial mean values for Po2 of 58, 60, and 72 mm Hg, respectively, were found for the three groups. When the same patients breathed oxygen via a nasal cannula or face mask at flow rates of 8 to 12 L/min, values were 106, 128, and 160 mm Hg, respectively. In all instances hemoglobin saturation values in excess of 95% were obtained when the patient breathed oxygen.

In 15 patients measurements of the oxygen saturation of right atrial blood permitted calculation of the degree of venous admixture. Saturation of mixed venous blood was found to be 34, 56, and 73% for the groups of patients, while the degree of venous admixture was calculated at 18, 27, and 23%, respectively. Furthermore, from the data obtained while the patients breathed oxygen, the magnitude of true right-to-left shunt was identical for all three groups at 11%. It was concluded that the initial pulmonary defect is similar for all patients with acute myocardial infarction, and possibly is due to elevated pulmonary venous pressure in all patients. As time progresses, however, the pulmonary defect changes according to the general progress and circulatory response of the subject. Thus, in those patients in whom general deterioration occurred, pulmonary function also deteriorated; whereas, in those who demonstrated clinical and hemodynamic improvement, pulmonary function improved progressively.

Additional Indexing Words:
Left ventricular failure
Pulmonary defect
Oxygen saturation of mixed venous blood
Pulmonary function
Right-to-left shunt

Arterial hypoxemia occurs frequently in patients with acute myocardial infarction.1-7 Moreover, this defect in pulmonary gas exchange is more evident when congestive heart failure or shock develop. Such observations pose two important questions: (1) What is the mechanism of arterial hypoxemia? (2) What are the physiologic importance and the therapeutic implications of the defect in pulmonary gas exchange? The purpose of this study was to gain data relevant to these two questions.

Arterial hypoxemia may be due to any of the following mechanisms alone or in combination: (1) alveolar hypoventilation, (2) right-to-left shunt, (3) ventilation-perfusion inequalities with areas of lung having low Va/Qc ratios, or (4) a reduction in diffusing capacity. In a previous study,7 we noted

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alveolar hypoventilation to be an uncommon finding in hypoxic patients with acute myocardial infarction, occurring only in patients who had severe chronic obstructive pulmonary disease, or who had received large doses of narcotics. Thus, arterial hypoxemia in the patient with acute myocardial infarction is more likely to be due to one or more of the last three mechanisms. Right-to-left shunt may be due to anatomic shunting of venous blood via the pulmonary artery or bronchial veins to pulmonary veins, or physiologic shunting via pulmonary capillaries traversing atelectatic or fluid-filled lung segments. In the normal subject, only a small fraction of total pulmonary blood flow (cardiac output) is shunted anatomically, and thus effects a minimal 1 to 2% decrease in arterial oxygen saturation. With respect to the latter two mechanisms, there is little practical value in separating the contribution of $V_A/Q_c$ in equalities from diffusion impairment as causes of arterial hypoxemia, since the hypoxemia due to either cause is easily corrected by inhalation of oxygen at an inspired concentration of 40% or more. Furthermore, the arterial hypoxemia occurring in patients with alveolar-capillary block syndrome can be explained on the basis of ventilation-perfusion inequalities and is not due to impaired diffusion. For our purposes, the term "$V/Q$ abnormality" will be used to represent the shunt-like effect of both $V_A/Q_c$ inequalities and diffusion impairment. The term "venous admixture" will represent the fraction of the cardiac output that is behaving functionally as right-to-left shunt. During the breathing of room air, the venous admixture is denoted "total venous admixture" or $Q_{VA}/Q_T$ and includes both the shunt-like effect of the $V/A$ abnormality and the shunting effect of the right-to-left shunt, denoted as "true venous admixture" or $Q_v/Q_T$. During the breathing of 40% or 100% oxygen the venous admixture is due solely to right-to-left shunt, that is, true venous admixture. Thus, by assessing venous admixture while breathing room air and 40 to 100% oxygen, it is possible to separate the $V/Q$ abnormality and right-to-left shunt and to define the mechanisms of arterial hypoxemia in patients with acute myocardial infarction.

Methods

Thirty-four patients with proved acute myocardial infarction who were admitted to the Coronary Care Unit at Cedars of Lebanon Hospital between November 1967 and March 1969 were selected for this study. Acute myocardial infarction was documented on the basis of clinical findings, and classical electrocardiographic and serum enzyme changes (SGOT, SGPT, LDH, and CPK). Eight patients were classified as having uncomplicated acute myocardial infarction, with no clinical evidence of left ventricular failure or shock. Fourteen patients were classified as having left ventricular failure manifested by a protodiastolic ventricular gallop ($S_2$), moist inspiratory rales at both lung bases on auscultation, and pulmonary vascular congestion or pulmonary edema on roentgenograms of the thorax. Twelve patients exhibited the shock syndrome manifested by cold, clammy skin; hypotension; or progressive fall of arterial blood pressure, or both; oliguria (urinary output less than 25 ml/hour), and alteration of sensorium. Central venous pressure (CVP) was monitored in all patients. A polyethylene catheter was introduced into an antecubital vein by the percutaneous or cutdown technic and then advanced into the superior vena cava. In 15 of the 34 patients, the tip of the CVP catheter was advanced into the right atrium. Catheter position was verified by chest roentgenogram after injecting 3 to 5 ml of Hypaque (diatrizoate) into the catheter. An 18-gauge Teflon needle catheter was placed percutaneously in either the brachial or the femoral artery. At least 15 minutes were allowed to elapse after cannulation of the artery before any blood samples were taken.

All studies were initiated within 24 hours of admission or after the onset of shock. Arterial blood samples were collected anaerobically in all 34 patients while the patient was breathing room air and analyzed immediately for pH, $P_{CO_2}$, and $P_O_2$. The patients then received oxygen either through a nasal cannula or a plastic face mask without a reservoir bag, at high flow rates (8 to 12 L/min) for 15 to 45 minutes, at which time arterial blood samples were again obtained and analyzed. The inspired oxygen concentration during use of this system and the flow rate of oxygen have been reported to be approximately 50%. Serial blood gas studies were obtained in nine of the 34 patients. Five of these nine patients (two without complications and three in left ventricular failure) subsequently improved and were discharged from the hospital. The other
four patients were in shock and died in the hospital.

Blood samples were analyzed for pH, Pco2 and Po2 by a pH/gas analyzer.* The oxygen electrode of the analyzer was calibrated with 10.6% carbon dioxide in nitrogen (0% oxygen) and 11.6% oxygen for the normal range readings (between 20 and 150 mm Hg) and with 11.6% and 100% oxygen for high oxygen tension readings (between 150 and 700 mm Hg). The reproducibility for Po2 determinations in our laboratory is ± 1 mm Hg in the low range (20 to 60 mm Hg), ± 2 mm Hg for the medium range (60 to 150 mm Hg) and ± 10 mm Hg in the high range (above 150 mm Hg). Oxygen saturation in the arterial and venous blood was determined from the oxygen dissociation curve with appropriate corrections for pH and temperature as described by Severinghaus.11

Determinations of venous admixture were made within 36 hours after admission or within 24 hours after the onset of shock in the 15 patients in whom the tip of the catheter was in the right atrium. Arterial and right atrial blood samples (mixed venous) were obtained simultaneously with the patient breathing room air. Patients then breathed 40% oxygen (nine patients) or 100% oxygen (six patients) for 12 to 30 minutes through a tight-fitting mouthpiece and 1-w resistance non-rebreathing valve after which arterial and venous blood samples were again drawn simultaneously.

Venous admixture was calculated using the following formulae:

\[
\frac{Q_{VA}}{Q_T} = \frac{C_{CO2} - C_{AO2}}{C_{CO2} - C_{VO2}} \times 100; \quad (1)
\]

\[
\frac{Q_{VA}}{Q_T} = \frac{C_{CO2} - C_{AO2}}{C_{CO2} - C_{VO2}} \times 100; \quad (2)
\]

that is total venous admixture \(Q_{VA}\) measured while the patient was breathing room air, expressed as percentage of cardiac output \(Q_T\).

Also \(\frac{Q_S}{Q_T} = \frac{C_{CO2} - C_{AO2}}{C_{CO2} - C_{VO2}} \times 100\)

that is true venous admixture \(Q_S\) was measured while patient was breathing oxygen expressed as percentage of cardiac output. \(C_{CO2}, C_{AO2},\) and \(C_{VO2}\) in milliliters of \(O_2\) per 100 ml of blood represent end-pulmonary capillary, arterial, and mixed venous oxygen contents, respectively.

Oxygen contents were calculated as:

\[S_o2 \times Hgb \times 1.34 + 0.003 \times P_o2\]

where \(S_o2\) was the oxygen saturation, \(P_o2\), the oxygen tension, and \(Hgb\), the hemoglobin content in grams per 100 ml of blood.

\(C_{Ao2}\) was calculated by assuming alveolar oxygen tension \(P_{Ao2}\) to be equal to end-pulmonary capillary oxygen tension.

\[P_{AO2} = P_{to2} - \frac{PaCO2}{RQ} \times (1 - FiO2 [1 - RQ]).\]

Where \(P_{to2}\) (mm Hg) = inspired oxygen tension; \(P_{ACO2}\) (mm Hg) = arterial carbon-dioxide tension; \(FiO2\) (%) = concentration of inspired oxygen; and \(RQ\) = respiratory exchange ratio.

It was assumed (1) that the venous sample from the right atrium was representative of mixed venous blood, and (2) that the RQ was equal to 0.85. The former of these assumptions will be considered in the discussion section of this paper.

Venous admixture \(\frac{Q_{VA}}{Q_T}\) and \(\frac{Q_S}{Q_T}\) was studied on a second occasion in eight of the 15 patients.

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Table 1

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>pH (unit)</th>
<th>Po2 (mm Hg)</th>
<th>So2 (%)</th>
<th>pH (unit)</th>
<th>Po2 (mm Hg)</th>
<th>So2 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>57 ± 12</td>
<td>7.45 ± 0.05</td>
<td>37 ± 5</td>
<td>72 ± 7</td>
<td>94 ± 2</td>
<td>7.43 ± 0.06</td>
<td>39 ± 7</td>
</tr>
<tr>
<td>58 ± 13</td>
<td>7.45 ± 0.05</td>
<td>35 ± 5</td>
<td>60 ± 10</td>
<td>90 ± 6</td>
<td>7.46 ± 0.06</td>
<td>35 ± 7</td>
</tr>
<tr>
<td>71 ± 15</td>
<td>7.46 ± 0.06</td>
<td>33 ± 5</td>
<td>58 ± 4</td>
<td>89 ± 4</td>
<td>7.46 ± 0.06</td>
<td>33 ± 5</td>
</tr>
</tbody>
</table>

*8-12 L/min via nasal cannula or face mask, \(FiO2\) = 50%.
†Mean values ±1 SD are shown.
Results

Values for arterial blood gases while the patients breathed air and the response to nasal or face mask oxygen inhalation in the 34 patients with acute myocardial infarction are summarized in Table 1. The patients in the shock group were significantly older (71 ± 15 years) than the patients with left ventricular failure (58 ± 13) or without complications (57 ± 12) (P < 0.05). There was no significant difference in mean arterial pH or Pco2 in the three groups on breathing room air. However, mean arterial Po2 was significantly lower in the left ventricular failure and shock groups than in the group without complications (P < 0.05). There were no significant changes in arterial pH or Pco2 during oxygen breathing compared to room air breathing in any group. The response to administration of oxygen via nasal cannula or face mask during the initial study is shown in Figure 1. The increase in arterial Po2 was the greatest in the uncomplicated group and least in the shock group.

The response of blood gases to nasal or face mask oxygen inhalation was determined serially for nine of the 34 patients. In five patients who recovered and left the hospital alive (Fig. 2), oxygen administration effected an average increase in arterial Po2 at the time of the initial study of 54 mm Hg, and of 92 mm Hg 4 to 8 days later. In one patient, although the arterial Po2 while breathing room air was lower during convalescence than during the initial study, the increase in arterial Po2 with oxygen breathing was greater than during the initial study. The four patients (Fig. 3) who died showed a comparable initial response to

![Figure 1](image1.png)

**Figure 1**

Effect of oxygen administration via nasal cannula during initial study on 02 pressure in 34 patients with acute myocardial infarction. Note that an increment on the vertical scale is equivalent to two and one-half times the magnitude indicated on the horizontal scale. A general relationship exists between the initial 02 pressure and the response to breathing nasal oxygen.

![Figure 2](image2.png)

**Figure 2**

The effect of oxygen administration on arterial 02 pressure during the initial and recovery periods in five patients with acute myocardial infarction. In the initial study (A) the average increase in Po2 was 54 mm Hg. Four to eight days later the response to oxygen administration in the same manner was 92 mm Hg.

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200

r-

H20

I2

23

#31

BREATHING ROOM AIR OXYGEN

SHOCK { INITIAL STUDY

LAST STUDY

Figure 3

Effect of oxygen administration on arterial Po2 at the onset of the shock state and during the last trial administration prior to death. The average initial increase was 48 mm of mercury. One to three days later the response was 28 mm Hg.

oxygen breathing averaging 48 mm Hg. However, 1 to 3 days later, the average increase during oxygen breathing had fallen to 28 mm Hg. Even though two patients had a slightly higher arterial Po2 while breathing room air during the final study, the increase in

Table 2

Venous Admixture Studies in 15 Patients with Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Breathing room air</th>
<th>Breathing oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>SaO2 (%)</td>
<td>SVO2 (%)</td>
</tr>
<tr>
<td>Mean</td>
<td>94.4</td>
</tr>
<tr>
<td>± SD</td>
<td>± 1.8</td>
</tr>
<tr>
<td><strong>No complications (6 patients)</strong></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>88.6</td>
</tr>
<tr>
<td>± SD</td>
<td>± 9.4</td>
</tr>
<tr>
<td><strong>Left ventricular failure (5 patients)</strong></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>88.7</td>
</tr>
<tr>
<td>± SD</td>
<td>± 5.8</td>
</tr>
</tbody>
</table>

**Abbreviations:** A-VO2 diff. = arteriovenous oxygen difference.

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arterial Po2 while breathing oxygen was less than during the initial determination.

Venous admixture calculations obtained for 15 of the 34 patients are shown in table 2. Arterial hypoxemia was due to combined \( \dot{V}/Q \) abnormality and right-to-left shunt in all patients. The contribution of \( \dot{V}/Q \) abnormality to arterial hypoxemia was equivalent to a shunt averaging 12.6% of cardiac output in the six patients without complications, 16.7% in the five patients with left ventricular failure, and 6.4% in the four shock patients. Right-to-left shunt averaged 11.1% of cardiac output in the six patients without complications, 11.4% in the five patients with left ventricular failure, and 11.6% in the four shock patients. The average true venous admixture to total venous admixture ratio was 0.51 in the patients without complications, 0.45 in patients with left ventricular failure, and 0.67 in patients with shock. Individual patients with severe hypoxemia had moderate degrees of right-to-left shunt, but higher \( \dot{V}/Q \) abnormalities.

Serial studies of venous admixture were obtained in eight of the 15 patients (fig. 4). Four patients (three without complications and one with left ventricular failure) showed reduction of both right-to-left shunt and \( \dot{V}/Q \) abnormality during convalescence and an increase in arterial Po2 indicating improvement of lung function. All four showed widening of the arteriovenous (A-V) oxygen difference which was slight in the three patients without complications. One patient in left ventricular failure showed a marked reduction of \( \dot{V}/Q \) abnormality after clinical improvement. \( \mathrm{CvO}_2 \) remained low, thus the \( \mathrm{CaO}_2 - \mathrm{CvO}_2 \) difference increased.* A fifth patient with left ventricular failure, who remained in left ventricular failure during the second study, showed an increase in \( \dot{V}/Q \) abnormality, indicating a worsening of lung function. The remaining three patients who were in shock had an increase in both \( \dot{V}/Q \) abnormality and right-to-left shunt.

Discussion

\( \dot{V}/Q \) abnormality and right-to-left shunting have been implicated in several studies of the mechanism of arterial hypoxemia in patients with acute myocardial infarction,\(^2,4,5,12\) and it has been suggested that pulmonary function is impaired to a greater degree when left ventricular failure or shock are present. Our data demonstrate that arterial hypoxemia in acute myocardial infarction is due to both \( \dot{V}/Q \) abnormality and right-to-left shunt, each mechanism contributing to a similar degree to the increase in venous admixture and to the decrease in arterial oxygen saturation. The patients in left ventricular failure and shock did have a greater degree of arterial hypoxemia. However, the severity of pulmonary dysfunction in the initial stage of the illness, as defined by the degree of venous admixture on breathing air, was closely similar in the groups of patients with uncomplicated myocardial infarction, the patients in left ventricular failure, and the patients in shock and was approximately one quarter of the cardiac output. This was an unexpected and a surprising finding. One would have assumed a priori that with the pulmonary congestion and edema of overt heart failure, pulmonary function would be impaired to a greater extent. How then can the differences in the degree of arterial hypoxemia in the three groups of patients be explained? Venous oxygen content is a function of cardiac output, oxygen consumption, and arterial oxygen content as defined by the Fick equation. In a steady metabolic state, oxygen consumption is constant, and the arteriovenous oxygen difference and the venous oxygen content will be a function of cardiac output. The effect of a change in cardiac output on arterial and mixed venous oxygen saturation with a fixed degree of right-to-left shunt (\( Q_s/Q_t = 20\% \)), a normal hemoglobin concentration, and an oxygen consumption of 250 ml/min is shown in figure 5. For a cardiac output of 5.0 L/min the arterial oxygen saturation will be 93.8% and mixed venous oxygen saturation 68.8%. If the cardiac output is reduced to 2.5 L/min, arterial oxygen saturation will fall to 87.5%.

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and mixed venous oxygen saturation will fall to 37.5%. The more severe arterial hypoxemia in the low output state is therefore primarily due to the lower mixed venous oxygen saturation, which results from a greater tissue extraction of oxygen because of the decrease in tissue perfusion. Thus, no fundamental change in pulmonary function is necessary to account for the decrease in arterial oxygen saturation in the sicker patients. Rather, a hemodynamic event is responsible. If the shunt were smaller, for example, 10%, arterial oxygen saturation would only fall from 97 to 94%.

Cardiac output is frequently reduced in acute myocardial infarction, particularly in the shock state,13-15 with a lower than normal mixed venous oxygen saturation resulting. Therefore, one cannot assume a normal A-V oxygen difference when calculating the percentage of venous admixture in patients with acute myocardial infarction. We have used mid-right or high-right atrial blood as representative of mixed venous blood, recognizing that it may differ from pulmonary artery blood of the patients studied. The most probable differences pertain to the patients in shock and some of the patients in failure in whom the oxygen content of inferior caval blood may be significantly less than superior caval (and high-right atrial) blood reversing the usual relationship. If this were so, then the true mixed venous blood would have a still lower oxygen saturation than the values of 31 to 41% (air) and 33 to 72% (oxygen) reported. This would allow only for the calculation of a shunt slightly smaller in magnitude than the values presented, further supporting the proposition that shunting is not greater in the patients with shock and left ventricular failure. The important issue, however is not the precise magnitude of the increase in venous admixture, but the observation that it is increased in all patients and increased to a comparable degree between groups of patients. Valentine and

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and associates measured mixed venous admixture in patients with acute myocardial infarction. The patients without radiologic pulmonary edema had a total venous admixture of 19.6% and those with pulmonary edema, 35.5%. However, the published data included only the highest value for total venous admixture, irrespective of the day when it occurred, whereas for this purpose we analyzed only the data obtained during the first 36 hours after hospitalization. When the highest values in our data irrespective of the time of collection were analyzed, the patients without complications had an average total venous admixture of 20.6%, and the patients in left ventricular failure and shock, 30.0%, a similar set of values. The mechanism of the widened A-V oxygen difference and low mixed venous oxygen saturation may differ in patients with left ventricular failure and shock. Marked depression of cardiac output is probably principally responsible. However, cardiac output has been found to be within normal limits in a significant number of patients with left ventricular failure. Hence, if comparisons between similar categories of patients are valid, a lowered mixed venous oxygen saturation may indicate an increase in total oxygen consumption, possibly due to the increased work of breathing caused by a reduction of lung compliance due to pulmonary congestion or edema, or both.

Our limited serial studies in patients with acute myocardial infarction demonstrated changes in accord with the course of the illness. Those who were convalescing satisfactorily showed improvement in pulmonary function with a reduction in V/Q abnormality and right-to-left shunt, while in those patients whose clinical status deteriorated, the abnormalities of pulmonary function as measured by the degree of venous admixture progressed. This suggests that the natural history of the pulmonary defect parallels the course of circulatory and other organ function. Furthermore, whatever the primary biochemical defect in the lung might be, it was not affected by the preservation of a normal arterial oxygen saturation during the interval between the serial studies, since it was possible to attain normal levels of oxygenation in all the patients studied.

It is possible to speculate on the structural alterations in the lung which cause pulmonary dysfunction in these patients. Depression of myocardial function results in reduction of cardiac output, an increase in left ventricular end-diastolic pressure, and an increase in left atrial pressure. Measurements of pulmonary artery diastolic pressures by Fluck and associates indicate that significant elevation of left ventricular end-diastolic pressure occurred in the great majority of patients hospitalized for acute myocardial infarction. In our experience, shock in acute myocardial infarction is almost always associated with radiologic evidence of left ventricular failure. Thus, clinical and arterial oxygen tension in the 34 patients. Mean values for each of the groups are shown. The magnitude of the response was greatest in the uncomplicated group but appeared to be in part related to the initial arterial O₂ pressure. Note, however, that in all instances arterial O₂ pressures appropriate to the attainment of arterial oxygen saturation in excess of 94% were attained under conditions of oxygen administration as they apply in conventional clinical practice.
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subclinical left ventricular failure with elevated pulmonary venous pressure occurs in the great majority of patients suffering from acute transmural myocardial infarction. In dogs subjected to increasing diastolic overload of the left ventricle by volume expansion, interstitial edema of the alveolar septa, intra-alveolar edema, and patchy atelectasis rapidly occur. Lung compliance falls and arterial hypoxemia develops. These structural alterations would result in some alveoli being underventilated, thus causing ventilation-perfusion inequalities, and cessation of ventilation to fluid-filled or atelectatic alveoli, that is physiologic right-to-left shunting. The observation that initially the patients without complications had a degree of pulmonary dysfunction similar to that of the more ill patients suggests that degrees of left ventricular failure that are subclinical can cause serious derangement of lung function.

The observations made in this study have a clinical implication. Because of the major contribution of V/Q abnormality to the venous admixture, arterial hypoxemia in acute myocardial infarction can be satisfactorily corrected by oxygen administration. Also, increased cardiac output has been noted with correction of arterial hypoxemia in patients with acute myocardial infarction and arterial oxygen saturations less than 90 per cent. The response of arterial Po2 to oxygen administration is closely related to the arterial Po2 while breathing room air (fig. 6) as observed by McKenzie and associates. Inability to achieve an adequate level of arterial oxygenation occurred almost exclusively during the later stages of this illness in patients with progressive shock or persistent left ventricular failure. The physiologic significance of these studies is not so much that the lung does not function normally, but that the course of the defect in pulmonary function appears to parallel the overall course of the patient. As the patients improved the response to oxygen administration increased. The reverse was true in the patients whose condition deteriorated. Hence, monitoring the blood gas response to oxygen appears to be a reasonably accurate indicator of the overall course of the patient.

References


Advice in Times of Adversity
Milton 1644

... that then, the people, or the greater part, more than at other times, wholly taken-up with the study of highest and most important matters to be reformed, should be disputing, reasoning, reading, inventing, discoursing, even to a rarity and admiration, things not before discoursed or written of, argues, first, a singular good-will, contentedness, and confidence in your prudent foresight, and safe government, ... as in a body, when the blood is fresh, the spirits pure and vigorous, not only to vital, but to rational, faculties, and those in the acutest, and the pertest operations of wit and subtlety, it argues in what good plight and constitution the body is; so when the cheerfulness of the people is so sprightly up, as that it has not only wherewith to guard well its own freedom and safety, but to spare, and to bestow upon the solidest and sublimest points of controversy and new invention....—MILTON, JOHN: Areopagitica: A speech for the Liberty of Unlicensed Printing, to the Parliament of England (1644). In Occasional Essays on Various Subjects, Chiefly Political and Historical; Extracted Partly from the Publick Newspapers. London, 1809, p. 236.
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