Precordial ST-segment Mapping

2. Effects of Oxygen Inhalation on Ischemic Injury in Patients with Acute Myocardial Infarction

JOHN E. MADIAS, M.D., NICOLAOS E. MADIAS, M.D., AND WILLIAM B. HOOD, JR., M.D.

SUMMARY Precordial ST-segment mapping was serially applied in the Coronary Care Unit for the study of the effect of oxygen inhalation on the ischemic injury in 17 patients with acute anterior transmural myocardial infarction. A 49-lead ECG system was used. The sum of all ST elevations ($\Sigma$ST) recorded was taken as an index of magnitude of ischemic injury and the number of recording sites showing ST elevation (NST) was taken as an index of extent of ischemic damage. Stability of the precardial maps was observed over a period of one hour while the patients were on ambient air. Oxygen inhalation for a mean of 66 min resulted in a fourfold increase of PaO$_2$ and a mean of 16% reduction of both $\Sigma$ST and NST. When the patients were returned to ambient air breathing, a mean of 13% increase of $\Sigma$ST and 19% of NST from the levels recorded during oxygen inhalation were observed. Levels of $\Sigma$ST and NST on ambient air following discontinuation of oxygen inhalation were not significantly different from the corresponding values from maps recorded before onset of oxygen breathing. Blood pressure and heart rate remained unchanged throughout the study. Clinical status of the patients was unchanged during the study period save for two patients who showed changes in intensity of their chest pain.

ARTHETRAL HYPOXEMIA is common in patients with acute myocardial infarction. Although diminished arterial partial pressures of oxygen (PaO$_2$) are most frequently noted in patients with congestive heart failure, hypoxemia is often present in patients without clinical evidence of pulmonary congestion. The independent effect of diminished PaO$_2$ on the hemodynamics of patients with myocardial infarction has not been determined, although in anesthetized and conscious dogs subjected to coronary occlusion, short periods of hypoxemia have not been found to depress further over-all left ventricular function.

Another major consideration is the effect of hypoxemia on the extent and magnitude of ischemic damage. Infarct size increased in dogs with coronary occlusion when periods of breathing low oxygen concentrations were applied. Conversely, in experimental studies with acute coronary occlusion, oxygen availability to border zones of myocardial infarction (as assessed by intramyocardial platinum electrodes) rose significantly during pure oxygen inhalation. Maroko et al. have found significant reduction of ischemic damage in dogs with acute coronary occlusion subjected to inhalation of increased oxygen concentrations.

Precordial ST-segment mapping reflects epicardial electrophysiology and has been applied in clinical studies for serial monitoring of the intensity and distribution of ischemic injury and for assessment of interventions directed at decreasing myocardial ischemic damage. The present study was undertaken to determine the effects of oxygen inhalation on the ischemic injury in patients with acute anterior myocardial infarction. By serially monitoring precardial ST-segment maps, a beneficial effect of oxygen inhalation was found. Both magnitude and extent of myocardial ischemic injury were favorably influenced.

Material and Methods

Seventeen consecutive patients with the preliminary diagnosis of acute anterior transmural myocardial infarction admitted to the Coronary Care Unit (CCU) of Boston City Hospital were studied. The diagnosis of myocardial infarction was based on the development of pathologic Q waves and evolution of ST-segment and T wave changes. Enzyme curves with rise and fall typical of myocardial necrosis were observed. The highest creatine phosphokinase was 767 ± 178 (range 180 to 2,700) with normal values up to 50 I.U./L. The highest serum glutamic oxaloacetic transaminase was 151 ± 18 (range 60 to 302) with normal values up to 20 I.U./L. The highest lactic dehydrogenase value was 569 ± 57 (range 244 to 1,000) with normal values up to 110 I.U./L. Fifteen patients were male and two female. Their mean age was 54.6 ± 2.3 (SEM) with a range of 36 to 70 years. Patients with bundle branch blocks, pacemakers, cardiogenic shock (Class IV of Killip), and those with onset of pain more than 12 hours prior to the time they were first examined were excluded. Based on the initial clinical assessment 11 patients were in Killip Class I, five in Class II and one in Class III. Management of patients followed routine principles and was not altered by the study protocol except for manipulation of oxygen therapy.

All patients underwent measurement of arterial blood gases while on ambient air, and after inhalation of pure oxygen for a mean of 66 min. Arterial blood samples were obtained from the radial artery, cuffed immediately after being drawn and iced. Measurements were made immediately in duplicate and the average of the two values was used in the analysis. A third measurement was done if PaO$_2$ or PaCO$_2$ varied more than 1 mm Hg and pH more than 0.01 units in the two initial measurements and the average was reported. An IL Model 313 Automatic Digital pH/Blood Gas Analyzer (Instrumentation Laboratory, Inc., Lexington, Mass.) was used. Calibration of the instrument with known standards using two points for pH and each gas is done routinely three times a day. A Kenwood disposable plastic oxygen mask (No. 76-0840, Will Ross, Inc., Milwaukee, Wisconsin) was taped on the patient’s face covering the nose and mouth. Oxygen supply tubing (Hudson No. 1115, formerly Model No. 0T536, 84" length) con-
Table 1. Clinical and Precordial Mapping Data before, during, and after Oxygen Inhalation (17 Patients)

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age/ Sex</th>
<th>Class</th>
<th>Age of MI (b)</th>
<th>O2 exposure (minutes)</th>
<th>PaO2 (mm Hg)</th>
<th>% O2 saturation</th>
<th>Heart rate (beats/min)</th>
<th>BP systolic (mm Hg)</th>
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</thead>
<tbody>
<tr>
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<td>Before O2</td>
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<tr>
<td>1</td>
<td>46 M</td>
<td>I</td>
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<td>48</td>
<td>71</td>
<td>305</td>
<td>93.0</td>
<td>99.5</td>
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<tr>
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<td>47 M</td>
<td>II</td>
<td>2.0</td>
<td>55</td>
<td>68</td>
<td>245</td>
<td>94.0</td>
<td>99.3</td>
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<tr>
<td>3</td>
<td>66 M</td>
<td>I</td>
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<td>55</td>
<td>85</td>
<td>253</td>
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<td>73</td>
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<td>90.3</td>
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<td>77</td>
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<td>280</td>
<td>93.3</td>
<td>99.3</td>
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</table>

Mean 54.6 5.7 66.1 70 278 91.7 98.4 89.7 90.3 89.6 90.7 139.2 138.2 137.5 135.3

P Value <0.001 <0.001 NS NS NS NS NS NS NS

Abbreviations: MI = myocardial infarction; SST = sum of ST elevations; Δ = change; Class = Killip Class; BP = blood pressure; SST = sites showing ST elevation ≥ 1 mm; a = first control map; b = second control map; c = map during O2 inhalation; d = map after discontinuation of O2 inhalation; b-c = change from map b to c; c-d = change from map c to d; h = hours.

Arterial blood gas analysis followed. A second control precordial map (b) and measurement of blood pressure was done 57.5 ± 3.3 (range 36 to 87) min following the initial control map (a). Following this baseline assessment the patients were given 66.1 ± 2.3 (range 48 to 80) min of oxygen inhalation. At the end of this period a third precordial map (c) and measurements of blood pressure and arterial blood gases were carried out. At the completion of this phase of the study the patients were returned to ambient air breathing, and 49.5 ± 1.9 (range 38 to 70) min after discontinuation of oxygen inhalation a final precordial map (d) and measurement of blood pressure were carried out. After the termination of the study the patients were placed on oxygen as done routinely. During the total duration of the study one of the authors remained at the bedside for the purpose of frequently examining the patients and monitoring their symptomatology.

The sum of ST elevation (SST) in mm (1 mV = 10 mm), measured at 0.06 sec after the nadir of S wave, using the TP segment as an isoelectric line was taken as an index of the...
magnitude of ischemic injury. The number of recording sites showing 1.0 or more mm ST elevation (NST) was taken as an index of the extent of the ischemic injury. Heart rate measurements were taken from the average of 20 R-R intervals of the last lead of precordial maps recorded (maps a to d).

Statistical evaluation of the results of the four study periods (two controls, one during oxygen inhalation, and one after oxygen breathing) was carried out by means of the Hotelling T^2 test. The results are reported as mean ± SEM. P values above 0.05 are reported as not significant (NS).

Results

Arterial Blood Gases

Oxygen inhalation resulted in a rise of PaO₂ from 70 ± 4.3 mm Hg measured during ambient air breathing to 278 ± 27.8 mm Hg (P < 0.001). Oxygen saturation improved from 91.7 ± 1.4% to 98.4 ± 0.8% (P < 0.001) as a result of oxygen inhalation. An increase of PaCO₂ from 31 ± 1.7 (range 14 to 47) mm Hg to 36 ± 1.6 (range 18 to 46) mm Hg (P < 0.02) was noted. pH remained stable, with values 7.44 ± 0.019 (range 7.34 to 7.52) units before and 7.41 ± 0.019 (range 7.30 to 7.57) units during oxygen inhalation. Details on the results of PaO₂ and oxygen saturation during the four phases of the study are shown in table 1. A weak correlation of the values of PaO₂ during ambient air breathing with the corresponding values obtained during pure oxygen inhalation was found (r = 0.669).

Heart Rate and Blood Pressure

Heart rates during the initial control periods (89.6 ± 2.8 beats/min in a and 90.2 ± 3.1 in b) did not change significantly throughout the study. Systolic blood pressure remained unaltered during the four study periods with values 139.2 ± 5.5, 138.2 ± 5.2 mm Hg at a and b, respectively. Changes in diastolic blood pressure from control values of 95.0 ± 3.2, 94.2 ± 3.1 mm Hg for a and b, respectively, were insignificant. Details on heart rate and blood pressure data are shown in table 1 and figure 1.

Clinical Status

Data on age, sex, clinical classification, and time of initial control study in relation to the onset of chest pain (age of
myocardial infarction) are shown in table 1. The patients’ clinical status remained unchanged throughout the study, except for two patients (Nos. 2 and 16) who showed alterations in the intensity of their chest pain (table 1).

**Precordial ST-Segment Maps**

The time interval between the two control precordial maps (a and b) was 57.5 ± 3.3 (range 36 to 87) min and the time between discontinuation of oxygen inhalation and the final precordial map (d) was 49.5 ± 1.9 (range 38 to 70) min. Precordial maps remained unchanged during the control period (a and b). \( \Sigma \text{ST} \) on ambient air breathing was 71.4 ± 10.8 mm at a and 70.1 ± 11.1 mm at b (NS). NST at a was 25.0 ± 2.2 and 23.8 ± 2.4 at b (N.S.). During oxygen inhalation a 16.2 ± 4.1% reduction of \( \Sigma \text{ST} \) and a 16.2 ± 4.3% decrease of NST from the preceding second control map (b) were noted. \( \Sigma \text{ST} \) and NST were significantly lower than both control maps (a and b) \( (P < 0.001) \). Reduction of the ST elevation in a patient under the influence of oxygen inhalation is shown in figure 2. Following discontinuation of oxygen inhalation a 13.1 ± 3.8% rise of \( \Sigma \text{ST} \) and a 19.6 ± 6.1% increase of NST were noted from the levels measured during oxygen inhalation (table 1, fig. 1). \( \Sigma \text{ST} \) after discontinuation of oxygen was 66.8 ± 11.1 mm and NST was 23.1 ± 2.4. These values were not statistically significantly different from the two control values (a and b). Percentage changes of \( \Sigma \text{ST} \) as a result of oxygen inhalation correlated well with those of NST \( (r = 0.722) \). There was no correlation between 1) age of the infarct, 2) duration of exposure to oxygen inhalation, 3) levels of initial PaO\(_2\), or 4) initial \( \Sigma \text{ST} \) or NST and the percentage changes of \( \Sigma \text{ST} \) and NST observed with oxygen inhalation. There was no correlation between the absolute values of PaO\(_2\) (during control and oxygen inhalation) as a function of the values of \( \Sigma \text{ST} \) or NST (at b and c). Also changes of PaO\(_2\) as a result of oxygen inhalation did not correlate with percentage changes of \( \Sigma \text{ST} \) or NST (map b and c). The percentage of increase of \( \Sigma \text{ST} \) and NST observed after discontinuation of oxygen breathing did not correlate with the time interval between the oxygen map (c) and the final study (map d). Details on data of \( \Sigma \text{ST} \) and NST and their percent changes during the four phases of the study are shown in table 1.

**Discussion**

Oxygen has been given routinely to patients with acute myocardial infarction, although the rationale of its use has not been clearly defined. Claims have been made in the past of oxygen’s effect in cardiac failure and alleviating chest pain in patients with angina or myocardial infarction.

Administration of oxygen raises oxygen saturation and may result in increased oxygen transport to the peripheral tissues and possibly also to the injured cardiac muscle. This latter mechanism may be of particular importance to the cardiac tissue in the periphery of an infarct, the “twilight zone.” Such zones surrounding the central areas of irreversible injury may be only partially damaged. Reduced oxygen tensions have been recorded from such zones using platinum intramyocardial electrodes in dogs subjected to coronary occlusion. Oxygen inhalation increased oxygen availability of these peripheral areas with marginal hypoxia although there was no systemic undersaturation following coronary occlusion and while the animals breathed ambient air. Sukumalchantra et al. when administering oxygen found a significant increase of oxygen transport to tissues (calculated as oxygen content \( \times \) cardiac output) only in patients with moderate to severe hypoxemia (oxygen saturation less than 90%).

Recently Angell et al. using intramyocardial polarographic assessment of local PO\(_2\) found that the epicardial ST elevation of various areas of myocardium of dogs subjected to coronary occlusions reflect local underlying intramyocardial PO\(_2\). Epicardial mapping in dogs with coronary occlusion revealed a significant increase of both \( \Sigma \text{ST} \) and NST during breathing of 10% oxygen, when compared to data from mapping during breathing of ambient air of the same animals. In addition the same workers found that breathing of 10% oxygen concentrations for eight hours following sustained coronary occlusion produced significant extension of necrosis at 24 hours when compared to control animals breathing ambient air. Extent and magnitude of necrosis was assessed histologically in these studies.

In recent experiments Maroko et al. using epicardial mapping found that both \( \Sigma \text{ST} \) and NST were significantly lower with 40% oxygen inhalation as compared to breathing of ambient air by the same animals during two periods of
occlusion. Mean PaO₂ of the control occlusion was 99 ± 5 mm Hg and during oxygen inhalation rose to 185 ± 5 mm Hg. This definite beneficial effect of increased oxygen concentrations on the ischemic damage was not accompanied by any changes in the blood pressure or heart rate of the animals.

In our study oxygen inhalation produced a significant reduction of both ΣST and NST and the changes of these parameters were similar. The duration of exposure, although not correlated with changes in the maps, was apparently sufficient to produce effects. Although it is possible that therapeutic interventions may be particularly effective in the first few hours after the inception of a coronary attack, we did not find any correlation between age of myocardial infarction and magnitude of changes in the map during oxygen inhalation. Pelides et al. found practolol to be effective in decreasing the magnitude of ischemic damage even in patients studied 72 hours after the inception of myocardial infarction, although the effect of the drug was attenuated when administered late in the course of the infarct. Improvement of PaO₂ with oxygen inhalation correlated with the value of PaO₂ on ambient air as previously described. However there was no correlation between the PaO₂ on ambient air or initial mapping data (map a) and improvement of the map during oxygen inhalation.

Blood pressure and heart rate remained unchanged throughout the study. Maroko et al. also did not find changes of these parameters during oxygen inhalation in dogs with coronary occlusion. Mackenzie et al. found an increase in blood pressure and a fall in heart rate during oxygen inhalation. In a study by Sukumalantra et al. of patients with mild hypoxemia (oxygen saturation above 90%), inhalation of oxygen did not result in significant changes in heart rate and arterial blood pressure, although both these parameters increased during oxygen breathing in patients with moderate to severe hypoxemia (oxygen saturation below 90%). Increase in blood pressure (not observed in our study) may cause an adverse effect of oxygen therapy by increasing ventricular afterload.

After discontinuation of oxygen inhalation in our study both the magnitude and the extent of ischemic damage were increased. Although there was no correlation between the time interval from discontinuation of oxygen breathing to the recording of the final map with the corresponding changes in the maps, the time on ambient air proved sufficient to reverse beneficial effects of oxygen inhalation. Reduction of PaO₂ with 10% oxygen breathing produced significant worsening of the map in the dog experiments of Radvany et al. within 30 min of inhalation of low oxygen concentrations.

There are presently no data with regard to the mechanism through which oxygen inhalation exerts its beneficial effect.

![Figure 2](https://example.com/f2.png)

**Figure 2.** Selected tracing from precordial maps during control periods (a and b), oxygen inhalation (c), and after discontinuation of oxygen breathing (d) of patient No. 15; note reduction of ST-segment elevations during oxygen inhalation. This patient showed stable control precordial maps and significant reduction of ST-segment elevation with oxygen; however ΣST after discontinuation of oxygen did not return to control levels in this example (table 1). Capital letters denote transverse rows and numbers vertical columns in the mapping protocol.
on cardiac muscle damaged by ischemia. Improvement of oxygen saturation can significantly increase the oxygen content of the blood but moderate to severe hypoxemia with saturation less than 90% is rarely seen in the uncomplicated myocardial infarction, though it is present in 50% of patients with significant congestive heart failure or cardiogenic shock. Only four of our 17 patients had oxygen saturation below 90% and only one showed significant changes of the precordial map with the improvement in the oxygen saturation. It was hypothesized that increase of the arterial-tissue partial pressure of oxygen gradient may have a salutary effect on the marginally ischemic cardiac tissue although there is no direct evidence for this. It has been suggested, but not proven, that the flow of hyperoxygenated blood through collaterals may be the reason for improvement. Observed oxygen availability was increased during oxygen inhalation to a greater extent when ischemia was produced by constriction of vessels than with total occlusion. Although extrapolation from the dog experiments to the clinical population cannot be carried out directly, it is tempting to attribute the improvement with oxygen inhalation to increased availability of oxygen through a severely narrowed but not occluded vessel. Such a speculation is in keeping with current beliefs by some workers that thrombosis is a consequence rather than a cause of myocardial infarction and is not an early event in the process of necrosis.

Another mechanism for the beneficial effect of oxygen inhalation in patients with coronary artery disease has been implicated by Ishikawa et al who found significant reduction of left ventricular contractility indices during 100% oxygen breathing. Such reduction in left ventricular contractility and consequent decrease of myocardial oxygen demand might be also operating in patients with myocardial infarction and might lead to improvement of marginally ischemic tissue. Even small changes of the oxygen content with oxygen inhalation, through the improved saturation of hemoglobin coupled with the increase of arterial-tissue oxygen gradient, may be sufficient to alleviate the imbalance of oxygen supply and demand of some marginally ischemic cardiac tissue.

Beneficial effects of high oxygen concentrations in patients with myocardial infarction should be weighed against potentially harmful effects of pure oxygen breathing on the lung. Although normal subjects breathing 100% oxygen did not suffer any untoward effects with oxygen inhalation of six to 12 hours' duration, a consideration should be given to possible vulnerability of the congested lungs of patients with myocardial infarction to toxic effects of pure oxygen. It may be that inhalation of pure oxygen for two to three hours does not have any harmful effect on the lung and probably could be further prolonged when brief periods of breathing ambient air or lower oxygen concentrations are interspersed with periods of high oxygen concentration breathing.

The best way to administer oxygen was not explored in this study. Although the current beneficial effects on the severity of ischemic damage resulted from high oxygen concentrations, this does not mean that 100% oxygen should be given to all patients with myocardial infarction. It is possible that oxygen at lower concentrations exerts its therapeutic effects on the ischemic injury. It is interesting that in a recent study in dogs subjected to coronary occlusions the effect of 100% oxygen was no more beneficial in reducing ischemic injury than 40% oxygen concentration. In the above study, 40% oxygen inhalation resulted in a PaO2 of 185 mm Hg. Such a level of PaO2 can be reached in patients with uncomplicated myocardial infarction with relatively low oxygen concentration but 100% oxygen may be required in patients in pulmonary edema and/or cardiogenic shock. Perhaps the PaO2 should be monitored so that in patients with uncomplicated myocardial infarction appropriate oxygen concentrations are used to achieve levels of PaO2 shown in the experimental studies to result in salvage of ischemic myocardium: higher concentrations might be more appropriate for patients with congestive heart failure or cardiogenic shock. Therapeutic interventions with 40% oxygen can safely be applied to patients with myocardial infarction. Studies with serial precordial maps and close clinical monitoring of patients will be of great significance to explore the effects of lower, more desirable oxygen concentrations on the size of the myocardial infarct.

References
Absolute Determination of Cardiac Output in Intra-aortic Balloon Pumped Patients Using the Radial Arterial Pressure Trace

GEORGE A. HERZLINGER, PH.D.

SUMMARY We describe a new method for the absolute determination of cardiac output in intra-aortic balloon pumped (IABP) patients. The method uses the known pumping volume of the IABP balloon and the radial arterial pressure trace, which is commonly used to monitor IABP patients, to determine the cardiac output. Two pressure excursions denoted by \( P_1 - P_0 \) and \( P_2 - P_0 \), characterizing IABP balloon deflation, and ventricular ejection, respectively, are extracted from the radial trace. The cardiac output (CO) is then determined by the simple relation: CO = \( (BV) \times \frac{(P_1 - P_0)}{(P_2 - P_0)} \times \frac{1}{HR} \), where HR is the heart rate, and the value for pumped balloon volume (BV) is corrected for the effect of the pressure in the patient's aorta. Comparison with dye dilution and thermal dilution procedures as carried out on a routine basis in a clinical setting produced a good correlation \( r = .928 \). When fit to a straight line through zero output, the data yielded a constant of proportionality of 0.973 between the above formula and the clinical procedures.

The procedure does not disturb the patient in any way, and enables continuous monitoring of cardiac output. This has been implemented using a real-time, miniaturized computer and allows much more information to be obtained than in usual single measurements.

THE RADIAL ARTERIAL PRESSURE CONTOUR which is commonly used to monitor intra-aortic balloon pumped (IABP) patients consists of several pulse segments representing discrete events in the cardiac cycle: ventricle ejection of blood into the aorta, balloon inflation accompanied by runoff from the aorta into the arterial tree, and balloon deflation, with the inflation transient separated from the balloon deflation by a short, relatively flat “plateau” region (fig. 1). The signal thus contains well defined cardiac and balloon events. Since one knows, and can control the pumping volume of the intra-aortic balloon, analysis of the radial pressure contour provides an opportunity to determine in absolute terms the stroke volume of the heart. This concept was enunciated by Arthur Kantrowitz (unpublished) in 1969. In this paper we present such an analysis, and describe a simple method for determining stroke volume and cardiac output on a continuous, beat by beat basis in IABP patients. The distortion of the arterial waveform as the pulse propagates from the aorta to the radial artery does not affect the features of the waveform relevant to the analysis, and the pressure is measured using the same radial cannula commonly used in IABP patients. The method described here gives the cardiac output (CO) directly in absolute terms and thus differs from the various empirical formulas which have been used in the past \(^4\) to determine CO within a constant of proportionality from the central aortic pressure contour.

The basic idea of the method is that two pressure excursions denoted by \( P_1 - P_0 \) and \( P_2 - P_0 \) can be extracted from the radial artery trace, one characteristic of balloon deflation within the descending thoracic aorta, the other of the change in aortic size due to ventricular ejection into the aortic root. As illustrated in figure 1, the pressure change \( P_1 - P_0 \) extends from the end of the plateau-like region of the pulse (just before the point \( F \) in fig. 1) to the pressure minimum at the end of diastole (point \( G \) in fig. 1). The pressure change \( P_2 - P_0 \) represents the excursion from the minimum at \( A \) to the systolic maximum at \( B \). The cardiac output is then given by:

\[
CO = \frac{BV \times (P_1 - P_0)}{(P_2 - P_0) \times HR}
\]

where HR is the heart rate, and BV the balloon pumping volume. The definition of \( P_1 \), \( P_2 \), and \( P_0 \) for several other types of radial traces with varying plateau characteristics is shown in figure 2. We have found that the very simple for-
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