Detection of Residual Myocardial Function in Acute Transmural Infarction Using Postextrasystolic Potentiation
A Computerized Angiographic Study

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and Y. Bouvrain, M.D.

SUMMARY Twelve subjects without clinical or hemodynamic heart failure, admitted for a first untreated anterior transmural myocardial infarction, were evaluated within the first 24 hours after the onset of symptoms. Pulmonary angiography was performed while a right ventricular extrastimulus was delivered every fourth beat at 50% of the RR interval to systematically analyze the basal and the postextrasystolic left ventricular frames. Left ventriculograms were quantitatively processed to determine the ejection fraction (EF) and the percentage of the end-diastolic circumference showing hypokinetic (%HK) or akinetic (%AK) areas. Left ventricular angiography was performed 1 month later in all cases at the same paced atrial heart rate to compare this final angiogram to the basal and the electrically induced postextrasystolic initial beats. During the 1-month period of the study none of these subjects had complications such as recurrent chest pain, heart failure or rhythm disturbances, and no drug administration was necessary.

Comparing the basal cycle of the initial angiogram and the final cycle, a poor correlation was found between the corresponding values of EF (r = 0.34), %HK (r = 0.38) and %AK (r = 0.48). The correlations were much better when a comparison was made between the postextrasystolic cycle of the initial angiogram and the final cycle (EF, r = 0.84; %HK, r = 0.96; %AK, r = 0.95).

These results indicate that, from the first day after a TMI, the analysis of the postextrasystolic frame allows accurate estimation of the final left ventricular function and regional wall motion abnormalities. Postextrasystolic potentiation may be useful in the acute state of transmural infarction to discriminate potentially reversible ischemic from definitely jeopardized areas.

HEMODYNAMIC PATTERNS of transmural myocardial infarction (TMI) have been extensively evaluated. Most studies only take into account cardiac output and capillary wedge pressure. This approach is useful for recognizing high-risk patients, especially using multivariate analysis. Although overall and regional wall motion in acute TMI have been evaluated, the information is far from complete.

Most studies define the acute state of TMI too broadly, i.e., from the first days to the first month after the clinical onset of symptoms. The early evaluation of ejection fraction and extent of akinetic areas do not give reliable information about the possible early detection of residual myocardial function. Postextrasystolic potentiation (PEP) is effective in detecting residual potential contractile function in stable coronary artery disease, but this has not been evaluated in man during the acute state of TMI.

The aim of our study was to quantitate, using computerized angiographic data, overall and regional left ventricular (LV) function and wall motion in the very acute (< 24 hours) state of TMI. During this early angiogram, the right ventricle is paced every fourth beat to systematically analyze the postextrasystolic
beats. We selected a homogeneous sample of subjects showing a first untreated anterior TMI without signs of cardiac failure in the first 24 hours after the beginning of the chest pain. LV angiography was performed again 1 month later to evaluate the natural evolution of wall motion abnormalities and the prognostic value of the PEP. The results of our work indicate that PEP, on the first day after the occurrence of a TMI, is effective in detecting potentially reversible hypokinetic areas.

Materials and Methods

Criteria of Admission

Twelve patients, ages 29–61 years (mean 52 years) with ECG evidence of acute anterior or anterolateral TMI were selected for this study. In all cases, the chest pain started less than 16 hours before admission to the coronary care unit. When the time of the clinical onset of pain was in doubt, the patient was rejected from the study. No subject showed clinical or hemodynamic signs of heart failure. (See the protocol section for hemodynamic definition of pump failure.) No patient had mechanical complications, such as acute mitral insufficiency or ventricular septal rupture. No patient had any rhythm disturbance, so no antiarrhythmic drug administration was necessary.

Only patients not taking chronic drug therapy (such as β-blocking agents, digitalis, lidocaine or other antiarrhythmic drugs) were admitted. If the chest pain persisted for more than 18 hours after the clinical onset of the TMI, despite a morphine injection administered at least in the twelfth hour, the patient was excluded from the study.

Criteria of Exclusion

Any one of the following criteria was sufficient to reject a patient from the study: a clinical history or ECG signs of myocardial infarction; chronic visceral disease with possible cardiovascular repercussion (chronic renal, hepatic or respiratory failure, diabetes or arterial hypertension); associated valvular or myocardial disease; or clinical signs of coronary insufficiency present more than 3 months before the current TMI. These admission and exclusion conditions were carefully controlled for each patient so that we could study a homogeneous sample of untreated subjects without previous cardiovascular problems, having a first anterior TMI in the first 24 hours, without LV pump failure, and without persistent pain. Each patient gave signed, informed consent. The protocol was examined, approved and supported by the General Delegation for Scientific and Technical Research.

Protocol

Right-heart catheterization was performed using a #7F Swan-Ganz thermodilution catheter connected to a P23Db Statham transducer. Right-heart pressures, capillary wedge pressure (CWP), and cardiac output (thermodilution 9520 Edwards cardiac output calculator) were measured.

When CWP was 15 mm Hg or more, or when the cardiac index was 2.5 l/min/m² or less, the patient was rejected from the study. If the patient had no hemodynamic evidence of LV heart failure, a bipolar pacing catheter was inserted in the right ventricle using the Seldinger technique; femoral arterial pressure was recorded using a #5F cannula; the Swan-Ganz catheter was then removed and a #7F NIH catheter was positioned in the trunk of the pulmonary artery. Pulmonary angiography was then performed in the 30° right anterior oblique (RAO) position, injecting 60 ml of 10% sodium amidotrizoate, 66% meglumine amidotrizoate (RadioSelectan) over 3 seconds. During angiography, a right ventricular extrastimulus was delivered through the pacing catheter every four sinus beats using an orthorhythmic coupled stimulator (SAVITA). The extrastimulus coupling interval was 50% of the RR interval in every case. Cine event markers (50 frames/sec) were recorded simultaneously with the ECC and the femoral pressure during angiography using a CGR 1000 photographic recorder. The angiographic system included an AB CGR Continental II generator and a Teleject volumetric injector. Each patient was then removed from the table and, as described in the method of Kasser and Kennedy,11 a calibration grid was filmed at the midthoracic level, with the same arcus and table positions to correct the x-ray magnification and lateral distortion. This initial hemodynamic investigation was performed in all the cases after the sixth but before the twenty-fourth hour after the clinical onset of the TMI (range 8–18 hours, mean 12 hours). During the first 72 hours after the onset of chest pain, all patients had an i.v. catheter inserted percutaneously into an antecubital vein so that blood could be withdrawn every 3 hours for measurement of MB-CPK. MB-CPK serum level was determined by the electrophoretic method.12

The patients were hospitalized for 1 month. During this period the only drug they received was i.v. heparin. If an additional drug administration was necessary (for example, antiarrhythmic drugs or β-blocking agents for recurrent pain), the subject was excluded from the study.

After 1 month, a final hemodynamic investigation was carried out, including right- and left-heart catheterization, LV angiography (60-ml injection over 3 seconds in the 30° RAO position), and coronary cineangiography. During LV angiography, right atrial stimulation was performed to drive the heart at the same rate (± 3 beats/min) as during initial angiography. This was done to compare the initial and final angiograms at the same atrial heart rate.

Technical Data Evaluation

End-diastolic and end-systolic LV frames were selected from the film cinemarkers and digitized from a 35-mm Vanguard projector using a sonic Graf pen (SAC) interfaced with a specially programmed 64K
octets HP 9845-B calculator. LV volumes were calculated using Simpson's integration method as the sum of adjacent 1-mm high slices parallel to the aortic base. The end-diastolic longitudinal axis is drawn from the apex to divide the end-diastolic frame into two equal areas ± 0.1 cm² (fig. 1). The longitudinal end-systolic axis is determined using Leighton's method adapted for computer processing. Briefly, the end-systolic longitudinal axis is drawn from the end-systolic apex to the intersection of the end-systolic aortic base and the end-diastolic longitudinal axis. Both end-diastolic and end-systolic longitudinal axes are aligned. The regional wall motion is evaluated for each point of the end-systolic frame (each mm) as the BC/AC × 100 (fig. 1) ratio (shortening). Each point is considered to belong to a hypokinetic (HK) sector if shortening is < 20%. Each point is considered to belong to an akinetic (AK) sector if the value is < 5%. The 20% value was determined by analyzing with the same method 20 angiograms of normal subjects, and is, for any point, below the lower limit of the 99% shortening confidence interval. Therefore, the %HK or the %AK values represent the percentage of the end-diastolic perimeter that shows a shortening value lower than 20% or 5% (fig. 1). Ejection fraction is determined by dividing the angiographically determined stroke volume by the end-diastolic volume. The volume elastic constant (α) was computed from the end-diastolic volume (VD) and from the mean CWP by the equation

\[ \alpha = \frac{\text{Log} 2.33 \text{CWP}}{\text{VD}}. \]

This equation corresponds to a linear relationship: dP/dV = α + β, where P and V are the end-diastolic corresponding pressure and volume with a constant intersection coefficient β (constant P at O volume of 0.43).

Wall stiffness is expressed by the slope k (dimensionless) of the linear relationship d σ/dε = k σ + c, where σ and ε represent the corresponding diastolic stress and strain, using Mirsky's equation for an ellipsoidal model and assuming a constant intercept c parameter. The calculations are done using the end-diastolic volume, mean CWP and the angiographically determined end-diastolic free wall thickness (hD). The normal range (95% confidence interval) in our laboratory for α is 15–32 × 10⁻³ ml⁻¹ and for k is 14–17%.

Enzymatic infarct size is determined by the method proposed by Shell et al. and modified by Norris et al. to compute the individual monoeponential disappearance rate constant using the values of CK-MB determined from the blood samples every 3 hours during the first 72 hours. Enzymatic infarct weight (EIW) is expressed in g-Eq. EIW is normalized for the LV mass determined from the basal cycle of the initial angiogram using the end-diastolic free wall thickness value and the end-diastolic internal dimensions by Rackley's method. Enzymatic infarct size (EIS) is expressed as a percentage of the LV mass.

**Patients**

Twelve patients were studied. On the initial angiogram, all had basal and postextrasystolic cycles of good technical quality. For the final LV angiogram, only the first three cycles were analyzed to avoid the direct influence of contrast medium, excluding extrasystolic or postextrasystolic cycles.

All angiograms were separately digitized by two experienced observers, and reexamined in the case of values showing more than 10% discrepancy between any computer result.

**Results**

Tables 1 and 2 show results of the initial and final calculations. For the initial examination, systolic arterial pressure, mean arterial pressure, cardiac index and stroke index were in the normal range. Heart rate was 82 ± 21 beats/min (mean ± s'd), CWP was 9.0 ± 3.8 mm Hg, and end-diastolic volume was (112 ± 17 ml/m²) of normal 55–105 ml/m², all slightly increased; EF was substantially decreased, to 42 ± 8% (normal 60–73%). The %HK was 43 ± 18% and %AK was 24 ± 14%, but k was increased, to 18.7 ± 4.7% (normal 14–17%). EIW was 20 ± 18 g-Eq and normalized infarct size was 16 ± 10% of the LV mass. Individual values are listed in table 1.

The comparison between initial (basal cycle) and final states for each subject was performed using paired t tests (table 3). There was no significant variation of cardiac index, stroke index or end-diastolic volume; heart rate was the same because of final atrial pacing. Before the atrial pacing, final heart rate was significantly lower (73 ± 18; p < 0.05), CWP decreased 26%, %HK decreased 17%, and %AK decreased 32%. Ejection fraction increased 5% (fig. 2). These variations did not reach a significant level.
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<th>Table 1. Initial Examination</th>
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<tr>
<td>HR (beats/min)</td>
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<td>SAP (mm Hg)</td>
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<td>k</td>
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<td>EIS (EIV/myocardial mass [%])</td>
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Abbreviations: HR = heart rate; CWP = capillary wedge pressure; SAP = systolic arterial pressure; MAP = mean arterial pressure; CI = cardiac index; SI = stroke index; EDV = end-diastolic volume; EF₁ = ejection fraction, basal cycle, initial angiogram; %HK₁ = percent hypokinesis, basal cycle, initial angiogram; %AK₁ = percent akinesis, basal cycle, initial angiogram; EF₂ = ejection fraction, postextrasystolic cycle, initial angiogram; %HK₂ = percent hypokinesis, postextrasystolic cycle, initial angiogram; %AK₂ = percent akinesis, basal cycle, initial angiogram; α = volume stiffness coefficient; k = wall normalized stiffness coefficient; EIW = enzymatic infarct weight; EIS = enzymatic infarct size.

<table>
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<td>k</td>
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Abbreviations: See table 1.

Compliance parameters α and k were reduced 15% and 19%, respectively, but only k was significantly reduced (p < 0.05). Systolic and mean arterial pressures were both significantly reduced (15% and 10%, respectively, p < 0.05).

Figure 3 indicates the correlation between the basal cycle of the initial angiogram (no. 1) and the final cycle (no. 3). A poor correlation was found between EF₁ and EF₃ (r = 0.34), %HK₁ and %HK₃ (r = 0.38), and %AK₁ and %AK₃ (r = 0.48). The correlation was greatly improved when the comparison was made between the postextrasystolic cycle of the initial angiogram (no. 2) and the final angiogram (no. 3): for EF₂ and EF₃, r = 0.84; for %AK₃ and %HK₃, r = 0.96;
TABLE 3. Comparison of Initial (Basal Cycle) and Final Hemodynamic Measurements

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<th>MAP</th>
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<th>%AK</th>
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<th>k</th>
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<td>% variation</td>
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<td>-15%</td>
<td>-10%</td>
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<td>5%</td>
<td>5%</td>
<td>5%</td>
<td>-17%</td>
<td>-32%</td>
<td>-15%</td>
<td>-19%</td>
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Statistical analysis was performed using the *t* test for paired values. Abbreviations: See table 1.

**Figure 2.** Change in resting ejection fraction (EF) from the basal cycle of the initial angiogram (EF₁) to the basal cycle of the final angiogram 1 month later (EF₃).

for %AK₁ and %AK₂; *r* = 0.95 (fig. 4). There was no significant difference between these correlation lines and the identity line (dotted line), particularly for %HK and %AK measurements.

**Discussion**

Although biplane techniques are better for detecting regional wall motion abnormalities in patients with coronary heart disease, single-plane methods are widely used and their limitations understood. In normal subjects there is a good correlation between single-plane and biplane overall and regional wall motion evaluation; in patients with coronary artery disease there is more scatter in the values, especially when inferior or posterolateral zones of asynergy are present. Because of the low reliability of the RAO position to detect inferior hypokinesia, only anterior or anterolateral localizations of TMI were included in our study.

Despite the invasive nature of angiographic procedures, we chose this method because two-dimensional echocardiography or nuclear imaging are not as reliable for computerized quantitative regional wall motion processing, and these methods require validation against accepted standards. The ability of computerized angiography to detect regional wall motion abnormalities as sensitive and specific markers of acute ischemia was recently established by Tzivoni et al. using closed-chest dog preparations. To limit the risk of LV angiography in the first hours of a TMI, pulmonary angiography was performed because it is known to have a very low risk when pulmonary artery pressure is low. (This was an inclusion criterion for our subjects.) Only the levophase of high-quality pulmonary angiograms was used, including a basal and postextrasystolic cycle valid for quantitative processing. In our experience, left ventriculograms from right- and left-sided injections are strongly correlated: the correlation coefficients in our laboratory for the end-diastolic volume and for the EF between these two angiographic techniques are 0.92 and 0.90, respectively, from angiograms of 20 subjects with and without coronary artery disease. The regression lines are not significantly different from the identity line. Some precautions should be observed: only high-quality ventricular angiograms should be used; and the first three cycles of the LV angiogram and the

**Figure 3.** Correlation between the basal cycle of the initial (< 24 hours) angiogram (EF₁) and the final cycle 1 month later (EF₃). EF = ejection fraction; HK = percent of hypokinetic areas; AK = percent of akinetic areas.
first completely visualized LV cycle should be analyzed.

Our results indicate that EF, %HK and %AK are more sensitive in detecting LV failure than hemodynamic measurements (cardiac index and CWP). No correlation was found between EF and cardiac index \((r = 0.39)\), EF and CWP \((r = 0.07)\), or between EF and both combined parameters \((r = 0.41)\). On the other hand, %HK and EF were significantly correlated \((r = -0.64; p < 0.02)\), indicating that the depression of LV function is directly related to the extent of RWM abnormalities.\(^{26}\) LV chamber compliance and wall stiffness were evaluated from the single end-diastolic LV pressure (approximated by the CWP) and volume. This assumes an exponential relationship between diastolic LV pressure and volume with a constant intercept coefficient \((p = 0.43\) for 0 volume). Although the validity of this approach has been established by Gaash et al.\(^{18}\) and others,\(^{31,32}\) the volume elastic constant has been shown to be dependent on initial end-diastolic volume and geometry.\(^{33}\) LV wall stiffness is expressed by the \(k\) coefficient, which has required normalization properties and allows comparison between ventricles of different sizes.\(^{19}\) In our study, \(\alpha\) was in the normal range while \(k\) was increased (above the upper limit of the 95% confidence interval in nine of 12 subjects), indicating an increased wall stiffness in the acute stage of TMI.

Initial (24-hour) and final (1-month) studies were compared using paired \(t\) tests for each subject. The validity of these tests can be assumed because great care was taken to select a homogeneous "normal" population and because of the absence of any drug therapy before or between the initial and final studies. Hemodynamic parameters (cardiac output and LV filling pressure) were not significantly changed 1 month later. LV pump function and regional wall motion abnormalities (%HK and %AK) were improved, but results were variable from subject to subject and did not reach a significant level. LV wall stiffness was significantly reduced in the final study, indicating reversible increased wall stiffness in the acute state of TMI, even in subjects without initial LV cardiac failure. Mean femoral arterial pressure was moderately (10%) but significantly reduced in the final study compared with the initial study. End-diastolic volume was not significantly different. A reduction in afterload without preload variation in the final study may affect LV performance and contribute to moderately increase the final EF.\(^{24}\) However, initial and final EFs were not significantly modified (fig. 2). Obviously, this moderate afterload final reduction does not obscure the correlation made for the overall and regional wall motion, between the postextrasystolic cycle of the initial study (no. 2) and the basal cycle of the final study (no. 3).

The correlation was very poor between EF, %HK and %AK evaluated respectively from the basal cycle of the initial angiogram (no. 1) and from the final angiogram (no. 3). LV pump function and regional wall motion abnormalities had improved in some patients (fig. 2, table 2), but did not in others. The basal initial angiographic study, performed during the first 24 hours after the beginning of TMI, was then unable to discriminate between these two patterns. The correlation between the initial (using PEP beat, no. 2) and final angiogram (no. 3) was much better (EF, \(r = 0.34\) vs 0.84; %HK, \(r = 0.38\) vs 0.96; %AK, \(r = 0.48\) vs 0.95). PEP is therefore effective in detecting the potentially reversible hypokinetic LV wall areas in the very acute (< 24 hours) state of TMI. Our findings are in agreement with those of Dyke et al.,\(^{38}\) who demonstrated during acute experimental regional ischemia that PEP might serve to identify viable but poorly perfused myocardium by enhancing segmental function. During short\(^{38}\) or longer\(^{39}\) periods of experimental ischemia, Boden et al. also showed that PEP augmented border zone segment performance to control levels. These results are consistent with the hypothesis of a potentially reversible ischemic zone surrounding the definitely early jeopardized myocardium, proved in experimental studies.\(^{38}\)

The results of our study prove that basal angiographic shortening measurements are more sensitive indexes of LV function than hemodynamic measurements alone, but are not predictive of potentially reversible wall motion abnormalities.

PEP is effective in discriminating between potentially reversible ischemic and definitely jeopardized
areas, and therefore has reliable prognostic value. The results of our work are encouraging for evaluating noninvasive techniques using dynamic interventions to study potentially viable LV regions in patients with the acute state of TMI. Noninvasively induced PEP has recently been possible without discomfort in patients with coronary heart disease. Afterload reduction is an alternative approach to this problem. This study emphasizes the value of dynamic studies for obtaining early prognostic information on LV function in the acute state of TMI.

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References

Hemodynamic and Metabolic Effects of Morphine in the Critically Ill

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SUMMARY To assess the effects of i.v. injection of morphine, 0.5 mg/kg, hemodynamic studies were performed on 24 critically ill patients under controlled ventilation. An esophageal balloon was used to estimate intrapleural pressure and transmural cardiac filling pressures were calculated. After injection of morphine, there were significant decreases in heart rate (13%), cardiac index (18%), stroke index (17%) and arterial pressure (15%) and there was a nonsignificant increase in esophageal pressure (15%). Transmural cardiac filling pressures decreased significantly (21% for the pulmonary wedge pressure); intravascular filling pressures were unchanged. Oxygen consumption decreased significantly, by 21%, in 10 patients with initially elevated oxygen consumption and by 9% in 14 patients with initially normal oxygen consumption. The oxygen extraction ratio was unchanged, suggesting that the decrease in oxygen consumption was caused by decreased oxygen demand rather than by inadequate oxygen delivery. These results indicate that the hemodynamic effects of morphine (0.5 mg/kg) administered to critically ill patients were associated with a significant decrease in oxygen consumption, which probably reflected sedation and analgesia.

MORPHINE can soothe severe pain, depress respiration and induce sedation without loss of consciousness. In critically ill patients treated in surgical intensive care units, large doses of morphine (0.5–2 mg/kg) can be used to provide sedation and analgesia. Of the 294 patients treated in the Surgical Intensive Care Unit of Pitie-Salpetriere Hospital in 1978, 197 received 45.522 mg of i.v. morphine. Each patient received an average daily dose of 82 mg (range 20–240 mg). The use of such quantities of morphine requires knowledge of whether morphine-induced sedation is associated with deleterious hemodynamic effects. Several studies performed on cardiac patients and on healthy volunteers demonstrated an apparent lack of detrimental hemodynamic changes after i.v. morphine in doses of 0.5–2 mg/kg. Nevertheless, increased cardiac filling pressures occurred during those studies even though morphine was not known to have any deleterious effect on myocardial contractility. Lappas et al. hypothesized that this increase in measured cardiac filling pressures could reflect some changes in pleural rather than transmural vascular pressures.

The aim of this study was to evaluate the changes in hemodynamics and oxygen consumption after high doses of i.v. morphine in critically ill patients. As all patients were mechanically ventilated, the changes in intravascular and transmural cardiac filling pressures were compared.

Methods

Patients

Twenty-four acutely ill patients being treated in the intensive care unit were selected according to the following criteria: absence of known cardiac disease, presence of sinus rhythm and stable hemodynamic condition without evident hypovolemia; absence of cardiotoxic and antiarrhythmic drugs; absence of morphine or sedative drugs in the 24 hours before the study; insertion of arterial and Swan-Ganz catheters within the preceding 3 days as an integral part of patient’s care; adaptation to the respirator; and absence of acute sepsis.

All patients were studied when the most acute phase of the disease had passed (2–5 days) and they were hemodynamically and metabolically stable. After the study, all received continuous i.v. infusion of morphine for 2–23 days. Most of the patients were ventilated with usual tidal volumes (8–9 ml/kg). Nine patients (nos. 3, 4, 9–11, 16, 18, 19, and 22) received larger tidal volumes (10–15 ml/kg) to enhance adaptation to the respirator. Eighteen patients were ventilated with intermittent positive pressure and a 5-cm
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