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Esophageal Doppler monitoring predicts fluid responsiveness in critically ill ventilated patients

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Abstract *Objective:* To test whether fluid responsiveness can be predicted by the respiratory variation in aortic blood flow and/or the flow time corrected for heart rate monitored with esophageal Doppler. *Design and setting:* Prospective study in a 24-bed medical intensive care unit of a university hospital. *Patients:* 38 mechanically ventilated patients with sinus rhythm and without spontaneous breathing activity in whom volume expansion was planned. *Interventions:* The aortic blood flow was measured using an esophageal Doppler monitoring device before and after fluid infusion (500 ml NaCl 0.9% over 10 min). The variation in aortic blood flow over a respiratory cycle between its minimal and maximal values was calculated. The flow time was also measured. *Measurements and results:* Aortic blood flow increased by at least 15% after volume expansion in 20 patients (defined as responders). Before fluid infusion the respiratory variation in aortic flow was higher in responders than in nonresponders (28±12% vs. 12±5%). It significantly decreased after volume expansion (18±11%) in responders only. A respiratory variation in aortic flow before volume expansion of at least 18% predicted fluid responsiveness with a sensitivity of 90% and a specificity of 94%. Flow time increased with fluid infusion in responders and nonresponders. A flow time corrected for heart rate below 277 ms predicted fluid responsiveness with a sensitivity of 55% and a specificity of 94%. The area under the ROC curve generated for variation in aortic blood flow ABF was greater than that generated for flow time. *Conclusions:* The respiratory variation in aortic blood flow reliably predicts fluid responsiveness in patients with sinus rhythm and without breathing activity.

Keywords Monitoring · Esophageal Doppler monitoring · Fluid responsiveness · Respiratory variation

Introduction

By measuring the aortic blood flow (ABF) in the descending thoracic aorta, esophageal Doppler monitoring allows a reliable noninvasive estimation of cardiac output [1, 2, 3]. This monitoring device tracks the changes in cardiac output induced by inotropic drugs [4] or volume replacement [5].

The duration of the aortic velocity signal corrected for heart rate, so-called "flow time corrected" (FTc), is considered a static indicator of cardiac preload [6]. It is currently used in the operating room to guide fluid management since algorithms incorporating this parameter have been demonstrated to reduce the in-hospital stay [7]. Several recent studies have emphasized the value of the respiratory variation in surrogates of stroke volume for predicting preload responsiveness in mechanically

ventilated patients and their superiority over static parameters of preload as filling pressures or echographic left ventricular dimensions [8]. In this regard, the respiratory variation in the peak aortic velocity measured by esophageal Doppler has been demonstrated to be valuable for detecting hypovolemia and for predicting volume responsiveness in rabbits undergoing blood spoliation and restitution [9].

The goal of this clinical study was to test whether a threshold value of the respiratory variation in peak aortic velocity and in ABF (ΔABF) provides a good prediction of fluid responsiveness and to compare this to the predictive value of FTc.

Patients and methods

Patients

We studied mechanically ventilated patients hospitalized in our medical intensive care unit for whom the decision to give fluid was taken by the attending physician. This decision was based on the presence of at least one clinical sign of acute circulatory failure and/or associated signs of hypoperfusion, including signs of renal dysfunction, hepatic dysfunction, and/or increased blood lactate in the absence of a contraindication for a fluid challenge. Clinical signs of acute circulatory failure were defined as (a) systolic blood pressure less than 90 mmHg (or a decrease of more than 50 mmHg in previously hypertensive patients) or the need of vasopressive drugs (dopamine >5 μg/kg per minute or norepinephrine), (b) urine output below 0.5 ml/kg per minute for at least 2 h, (c) tachycardia, and (d) the presence of skin mottling. Contraindication for a fluid challenge was defined by a life threatening hypoxemia (PaO_2/FIO_2 <100 mmHg) and by the evidence of blood volume overload and/or of hydrostatic pulmonary edema on chest radiography. We did not take in account the value of the respiratory variation in pulse pressure for including the patients since its accuracy for predicting fluid responsiveness is already well demonstrated [8]. In addition, patients needed to be on a volume-assist controlled ventilation mode with complete adaptation to the ventilator, as assessed by visual observation of the airway pressure/time curve. Thus patients with spontaneous breathing activity were excluded from the study, as were patients with cardiac arrhythmias and patients having contraindication for the use of esophageal Doppler monitoring (i.e., known or suspected esophageal ulcer, mycosis, malformation, varicose or tumor).

The study originally included 39 patients, but a correct ABF signal could not be obtained in one patient suffering from obesity. The group of remaining patients included 25 men and 13 women aged 56±15 years. They were ventilated with a tidal volume of 8±2 ml/kg, had a respiratory rate of 23±5 cycles/min and an inspiratory/expiratory ratio of 0.25±0.05. Positive expiratory pressure was 6±4 cmH_2O. Twenty-eight patients were receiving sedative drugs. This cohort comprised a broad sample critically ill patients, including medical patients with a variety of primary diagnoses and chronic diseases, including coronary artery disease (n=7), dilated cardiomyopathy (n=3), chronic renal insufficiency (n=5), chronic obstructive lung disease (n=6), diabetes mellitus (n=5) and hepatic cirrhosis (n=2). Acute circulatory failure was related to sepsis in 24 patients and to unknown origin in the remaining 14. Vasoactive drugs were being administered to 19 patients (16 norepinephrine, 2 dopamine, 2 epinephrine, 1 dobutamine).

This study received the approval of three different ethics committees: the institutional review board for human subjects of Bicêtre Hospital in Paris, France (Comité Consultatif pour la Protection des Personnes se prêtant à la Recherche Biomédicale), the institutional board of the University of Pittsburgh, USA, and the ethics committee of the Société de Réanimation de Langue Française in Paris, France. All approved the protocol and considered it to be a part of the routine practice; thus they agreed that informed consent could be waived for inclusion in this study. However, subjects were informed that they participated in this clinical study at discharge.

Measurements

All hemodynamic data were continuously recorded, digitized, and computerized using the HEM 3.5 software (Notocord, Croissy-sur-Seine, France). Arterial pressure was measured either noninvasively using an automatic cuff or through an arterial (femoral or radial) catheter. Heart rate and arterial pressure were recorded through an M1092A bedside monitor (Hewlett-Packard, Les Ullis, France) and averaged over a 10-s period.

Esophageal Doppler monitoring measurements were obtained using the Hemosonic 100 device (Arrow International, Everett, Mass., USA) [10]. The same investigator (X.M.), who is trained for this technique, performed all measurements. This esophageal Doppler monitoring device enables continuous measurement of descending thoracic aorta blood velocity (Doppler transducer) and of aortic diameter (M-mode echo transducer). ABF was calculated continuously by the acquisition software from the aortic blood velocity and diameter signals, and its mean value was calculated over 10 s.

The variation in ABF (△ABF) was calculated automatically as follows [11]:

$$\Delta ABF(\%) = (ABF_{max} - ABF_{min})/[(ABF_{max} + ABF_{min})/2] \times 100$$

where ABF_{max} and ABF_{min} are the maximal and minimal peak ABF values over one respiratory cycle, respectively. The △ABF value was averaged over five respiratory

cycles. The respiratory variation in peak aortic blood velocity between its maximal and minimal values over one respiratory cycle was calculated using an analogous formula. The flow time was measured between the beginning and the end of the aortic velocity waveform. FTc was calculated for minimizing the heart rate dependency:

$$\mathrm{FTc} = \mathrm{Flowtime}/\sqrt{\mathrm{cycletime}}$$

where cycle time is the interval between two successive velocity waveforms. Mean aortic diameter and FTc were averaged over a 10-s period.

Study protocol

The Doppler probe was inserted through the mouth, advanced into the esophagus, and adjusted to obtain the highest Doppler velocity signal from the descending aorta. The simultaneous display of both aortic flow velocity and aortic wall (proximal and distal) images was used to indicate that the probe was optimally positioned with its axis parallel to the aorta and the transducer centered in the middle of the aortic lumen. The time required to obtain an optimal signal was 4±1 min. No sedative or paralyzing agents were used during probe insertion. All hemodynamic parameters were measured at baseline prior to fluid infusion and just after the administration of 500 ml of NaCl 0.9% within 10 min. The transesophageal probe was repositioned during the course of the study if aortic blood velocity and/or aortic diameter signals deteriorated, as defined by rotation of the image away from the aortic centerline. The ventilator settings were kept constant throughout the study period. No change in vasoactive drug therapy were made during the study period.

Statistical analysis

The normality of the parameters was tested with a Kolmogorov-Smirnov test for normality. All variables except respiratory variation in peak velocity were normally distributed. For the normally distributed variables, comparisons before and after fluid infusion and between responders and nonresponders were assessed using two-way analysis of variance. For the respiratory variation in peak velocity the effects of volume expansion on parameters were assessed using the Wilcoxon rank sum test, and responders and nonresponders were compared by the Mann-Whitney *U* test. Patients with increase in ABF induced by fluid loading of 15% or more from baseline were defined as responders (n=20) and those with less than 15% as nonresponders (n=18) [11]. Results are expressed as mean ±SD. Receiver operating characteristic (ROC) curves were generated for △ABF, respiratory variation in peak velocity, and FTc, varying the discriminating threshold of each parameter. The area under the ROC curve for each parameter was calculated and compared using the Hanley-McNeil test. The intraobserver variability in ABF averaged over 10 s was described as bias ±limits of agreement of the ABF mean measured at 5 min intervals. Furthermore, the beat-by-beat variability in ABF signal was tested by calculating ΔABF over a 10-s period during an inspiratory pause in ten patients. Statistical analysis was performed using Statview 5.0 software (Abacus Concepts, Berkeley, Calif., USA). Differences with a p value of 0.05 or less were considered statistically significant.

Results

Variability in aortic blood flow signal

The intraobserver variability in ABF measurement was 0.1 (–0.7 to +0.9 l/min, bias and limits of agreement). The ΔABF measured over a 10-s inspiratory pause was 4±1%.

Effects of volume expansion on ABF and aortic diameter

Hemodynamic data are summarized in Table 1. In the 20 responders ABF increased by 43±24% after fluid infusion (p<0.05) while it did not change significantly in nonresponders (Table 1). Volume expansion was associated with an increase in mean aortic diameter. Considering all patients the changes in mean arterial pressure induced by volume expansion was well correlated with the changes in mean aortic diameter (r=0.52, p<0.001).

Effects of volume expansion on FTc

FTc increased significantly after fluid infusion in both responders and nonresponders, and the changes in FTc induced by fluid loading did not differ significantly between the two groups. FTc before volume expansion was significantly lower in responders than in nonresponders, and considering all patients a level of FTc less than 277 ms before volume expansion predicted fluid responsiveness with a sensitivity of 55% (95% interval confidence 32–77%) and a specificity of 94% (95% interval confidence 73–99%).

Respiratory variation in ABF and of peak velocity

In responders △ABF decreased from 28±12% to 18±11% after fluid infusion (p<0.05; Table 1, Fig. 1). In nonresponders, △ABF before volume expansion was significantly lower than in responders and did not change significantly after fluid infusion. The changes in ΔABF in-

Table 1 Course of hemodynamic and aortic blood flow parameters in responders and nonresponders before and after volume expansion (*VE*)

	Before VE	After VE
HR heart rate (beats/min)		
Nonresponders	98±18	97±16
Responders	115±33	111±30**
Systolic arterial pressure (mmHg)		
Nonresponders	122±28	126±30
Responders	95±20*	119±24**
Diastolic arterial pressure (mmHg)		
Nonresponders	59±18	59±18
Responders	49±13	59±17**
Mean arterial pressure (mmHg)		
Nonresponders	80±19	82±20
Responders	64±14	79±17**
Aortic blood flow (l/min)		
Nonresponders	4.9±2.0	5.1±2.1
Responders	2.6±0.8*	3.7±1.0**
respiratory variation in aortic blood flow (%)		
Nonresponders	12±5	13±7
Responders	28±12*	18±11**
Flow time corrected for heart rate (ms)		
Nonresponders	329±43	350±59
Responders	288±47*	307±62*·**
Aortic diameter (mm)		
Nonresponders	22±3	22±3
Responders	22±3	23±3**

*p<0.05, responders vs. nonresponders, **p<0.05 after VE vs. before VE

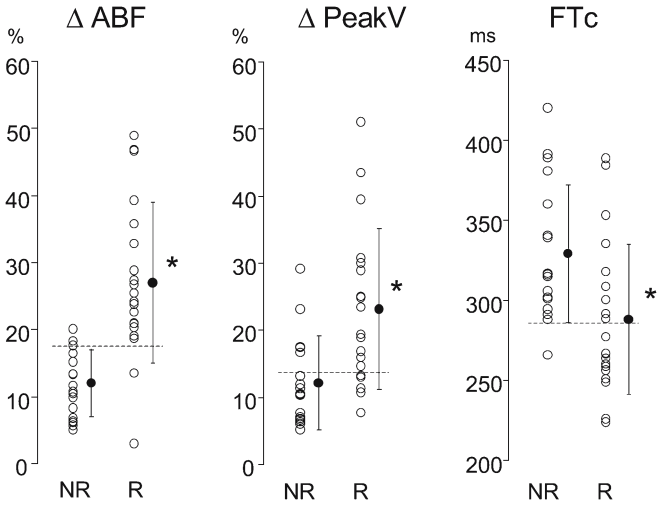

Fig. 2 Individual values (*open circles*) and mean ±SD (*closed circles*) of the respiratory variation in aortic blood flow (Δ*ABF*), of the respiratory variation in aortic peak velocity (Δ*peakV*) and of the aortic flow time corrected for heart rate (*FTc*), all measured before volume expansion in responders (*R*) and nonresponders (*NR*). *p<0.05 vs. responders

duced by fluid loading were significantly greater in responders than in nonresponders. A △ABF of 18% or greater predicted the response to fluid infusion with a sensitivity of 90% (95% interval confidence 68–98%) and a specificity of 94% (95% interval confidence 73–99%; Fig. 2).

In responders the respiratory variation in peak velocity was greater than in nonresponders and decreased significantly after fluid infusion while it remained unchanged in nonresponders. The changes in respiratory variation in peak velocity induced by fluid loading were significantly greater in responders than in nonresponders. A respiratory variation in peak velocity of 13% or more before volume expansion predicted the hemodynamic response to fluid with a sensitivity of 80% (95% interval confidence 56–94%) and a specificity of 72% (95% interval confidence 46–90%; Fig. 2). There was a close correlation between ΔABF and respiratory variation in peak velocity in responders (r=0.57).

The mean area under the ROC curves were 0.93±0.04 for △ABF, 0.82±0.07 for respiratory variation in peak velocity, 0.76±0.08 and for FTc (Fig. 3). The area under the ROC curve generated for ΔABF was significantly greater than that for FTc. The area under the ROC curve

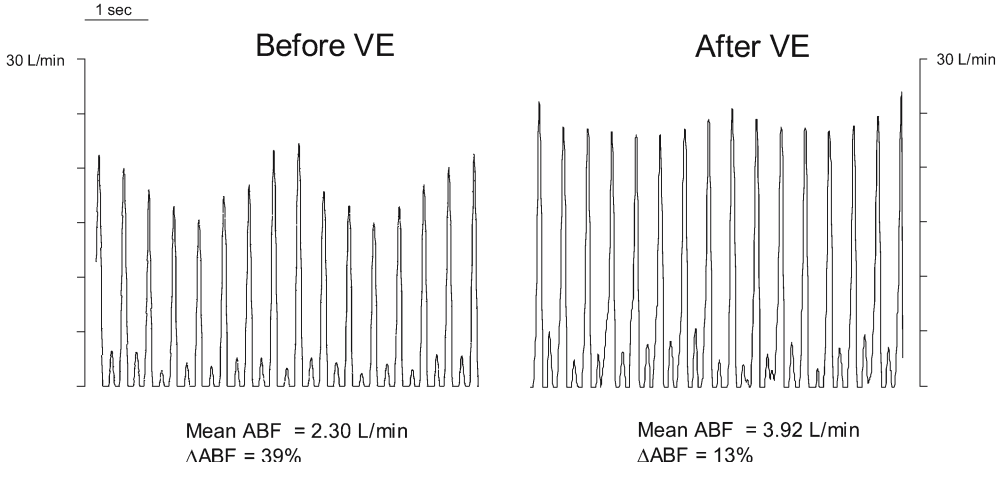

Fig. 1 Typical waveform of aortic blood flow, with mean value of aortic blood flow (*Mean ABF*) and respiratory variation in aortic blood flow (Δ*ABF*), before and after volume expansion (*VE*) in a patient responding to fluid loading

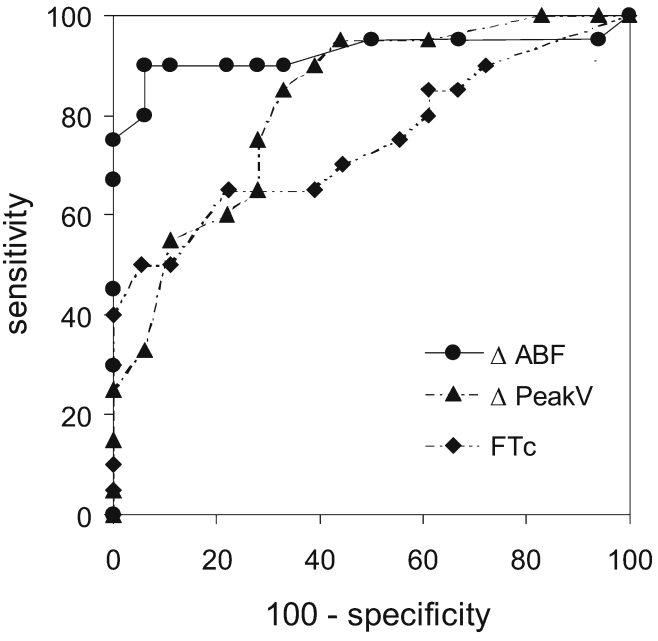

Fig. 3 Receiver operating curves comparing the ability of the respiratory variation in aortic blood flow (Δ*ABF*), respiratory variation in aortic peak velocity (Δ*PeakV*) and flow time corrected for heart rate (*FTc*) to discriminate responders and nonresponders to volume expansion

generated for baseline ABF was 0.77±0.10. It was significantly smaller than that of △ABF. The other comparisons of area under ROC curves demonstrated no significant differences.

Discussion

Our study demonstrates that in patients with acute circulatory failure fluid responsiveness can be predicted noninvasively by measuring the changes in Doppler ABF resulting from the respiratory cycle. A threshold △ABF value of 18% predicted fluid responsiveness with high sensitivity and specificity values.

Only 50% of patients with acute circulatory failure hospitalized in intensive care unit can increase their left ventricular stroke volume in response to volume expansion [8]. Predicting preload-responsiveness at the bedside is therefore an important issue. Recent studies have demonstrated that in subjects fully adapted to mechanical ventilation the respiratory variation in surrogates of left ventricular stroke volume is a reliable predictor of cardiac preload-responsiveness [8, 12, 13]. The cyclic variation in preload induced by mechanical ventilation results in significant cyclic changes in surrogates of stroke volume only if the heart is preload dependent. In this regard Slama et al. [9] recently demonstrated that respiratory variation in the peak velocity in the descending aorta was a reliable indicator of blood spoliation and restitution in rabbits. Our ΔABF results are consistent with these experimental findings and represent an extension of the findings of Feissel et al. [13] who used conventional Doppler echocardiography to measure Doppler blood velocity at the level of the aortic annulus.

In contrast to conventional echocardiography, esophageal Doppler monitoring is easy to perform and does not require a specialist in Doppler acquisition. Furthermore, it should enable continuous monitoring of the respiratory variation in ABF which is not possible with conventional echocardiography. It has been demonstrated to provide a reliable estimate of cardiac output in several studies that have recently been summarized [2]. Furthermore, esophageal Doppler monitoring correctly tracks the changes in cardiac output induced either by the inotropic therapy [4] or by fluid replacement [5]. Since esophageal Doppler monitors measure descending ABF, estimating cardiac output by esophageal Doppler requires a correction for the fraction of stroke volume that is ejected toward the upper part of the arterial tree. The bias possibly resulting from this correction could not have altered our results since we evaluated the effects of fluid loading on blood flow of the descending aorta, without taking into account the value of cardiac output that is estimated from it. Furthermore, our analysis considered only the relative variations in ABF from baseline values, excluding any effect of a potential error of measurement in absolute ABF.

One advantage of the Hemosonic probe is to measure on a beat-by-beat basis not only the descending aorta blood velocity but also aortic diameter. The ABF depends not only on aortic velocity but also on aortic diameter, which physiologically depends on aortic pressure. In this regard we found a close correlation between the changes in aortic diameter induced by volume infusion and those of mean arterial pressure. It is noteworthy that the correlation coefficient between ΔABF and respiratory variation in peak velocity was only 0.57, suggesting that the respiratory variation in aortic diameter accounted for around one-third of the observed ΔABF. This finding is not surprising since the respiratory changes in aortic pressure, and hence in aortic diameter, are expected to be large in preload responsive patients [14]. This may explain why the predictive value of ΔABF was greater than that of the respiratory variation in peak velocity in our study. In this regard six patients who were misclassified in terms of response to fluid infusion by the respiratory variation in peak velocity were correctly classified by ΔABF. Otherwise, two patients exhibited high ΔABF values although they did not respond to fluid infusion (false positive cases). In one case ΔABF was very near from the cutoff value, while it was much higher in the other case. Other investigators addressing the issue of respiratory variation in hemodynamic signals have also reported false-positive values. Some of them attributed these false-positives to an afterload effect of the right

ventricle in the setting of acute respiratory distress syndrome [15]. However, in our study the two "false-positive" patients did not suffer from this syndrome.

In the present study we found that FTc rose with fluid infusion to a similar extent in responders and nonresponders, suggesting that FTc tracks the changes in cardiac preload. In this regard it can be used to ensure that preload is effectively affected during fluid loading. However, we found that FTc at baseline is of less value than ΔABF for predicting fluid responsiveness. This finding is consistent with that of numerous clinical studies showing that "static" markers of preload (filling pressures, end-diastolic left ventricular dimensions) fail to predict hemodynamic response to fluid infusion [8].

It is likely that nonresponders benefited from greater volume resuscitation before the beginning of the study and particularly before ICU admission. Thus nonresponders had probably already used a large part of their preload reserve. This explains why both ABF and FTc were higher in these patients than in responders. It is noteworthy that the preload of responders at baseline was lower, as assessed by a lower FTc, and that the 500 ml volume infusion only partially corrected this preload defect. This explains why the FTc was still low in responders after 500 ml volume administration.

Our study has some limitations. First, we did not study patients with spontaneous breathing activity or cardiac arrhythmias, conditions in which respiratory variation in hemodynamic signals cannot be easily interpreted [8, 16]. This is presently a limitation of all techniques that measure variations in left ventricular output during ventilation as a measure of preload responsiveness. Second, the measurement of aortic blood velocity and of aortic diameter with esophageal Doppler are subject to sources of errors. Small errors in the measurement of aortic diameter may particularly reduce the precision of the ABF value provided by this device. Despite the variability in ΔABF during a short inspiratory pause was low in our study, this could partly explain the respiratory variation observed in nonresponders. The cutoff value of ABF found for the prediction of fluid responsiveness was, however, largely above the mean value observed in nonresponders. Third, the correction of hypovolemia could have decreased the distribution of blood flow toward the heart and the brain and resulted in a greater increase in the descending ABF than in cardiac output and in a lower ΔABF cutoff value.

Fourth, we used sophisticated software to analyze computerized signals, and therefore the extrapolation of our ΔABF findings to routine conditions should be cautious until technological upgrading of the current Doppler monitor occurs. Fifth, we did not compare the predictive value of ΔABF to that of the respiratory variation in pulse pressure. In fact, fewer than one-half of the patients had an arterial catheter in place at the time of Doppler measurement. Moreover, at the time of the present study the significance of pulse pressure variation was still under investigation such that this hemodynamic variable was not included in specific guidelines in our ICU. The values of sensitivity and specificity reported for pulse pressure variation [11, 16] are, however, close to that we found for ΔABF, although further studies should perform this comparison specifically. Finally, as with all measures of respiration-induced changes in left ventricular output, the degree of flow variability is a function of tidal volume. In our study the tidal volume was 8±2 ml/kg. Thus our threshold value of 18% ΔABF may be not applicable to patients with acute lung injury receiving lower tidal volumes.

In conclusion, our study demonstrates that esophageal Doppler monitoring can be helpful in mechanically ventilated patients for noninvasively assessing preload-responsiveness, particularly by quantifying the respiratory variation in ABF.

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