ORIGINAL ARTICLE

Do radial arterial pressure curves have diagnostic validity for identify severe aortic stenosis?

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Abstract

Purpose   A pulsus parvus et tardus of the carotid artery, i.e., a small weak pulse with a delayed systolic peak, is a well-recognized clinical finding of aortic stenosis (AS). However, the diagnostic value of radial arterial pressure curves has not been specifically investigated. In this study, we investigated whether the radial arterial curves of patients with AS had distinguishable characteristics.

Methods   We studied 17 AS patients (valve area less than $0.8\ cm^2$) and 17 control patients. The durations for the following intervals were measured, from the ECG-R to the beginning of the arterial pressure wave upstroke (PTT_{Up}), from the ECG-R to the peak of the arterial pressure (PTT_{peak}), and the difference between PTT_{peak} and PTT_{Up} ($T_{Upstroke}$).

Results   The radial arterial pulse pressures did not differ significantly, indicating absence of a pulsus paruvus in the radial pressure of the AS patients. The PTT_{Up} and PTT_{peak} in the AS patients were 134 ± 18 and 337 ± 44 ms, respectively, while these values in the control patients were 143 ± 21 and 286 ± 64 ms. As a result of the differences in PTT_{Up} and PTT_{peak}, the $T_{Upstroke}$ in the AS patients was greater than that in the control patients. The $T_{Upstroke}$ cutoff point that gave the maximal sensitivity (0.93) and specificity (0.65) was 156 ms.

Conclusion   Our study using the radial arterial curve validated a pulsus tardus as a diagnostic sign for severe AS, while the validity of a pulsus parvus as a diagnostic sign was not confirmed.

Keywords   Aortic stenosis · Pulse contour · Arterial pressure transit time · Monitoring

Introduction

Severe aortic stenosis (AS) poses an increased anesthetic risk for noncardiac surgery [1]. Although an echocardiogram provides a definitive diagnosis of AS, clinical findings are important as clues for the diagnosis. In addition to a systolic murmur in the right upper sternal border, a pulsus parvus et tardus (a small pulse and delayed systolic peak) of the carotid artery is well recognized as a clinical finding for severe AS [2]. However, the diagnostic value of radial arterial pressure curves for identify severe AS has not been specifically investigated, although, in anesthetic practice, arterial pressure is measured invasively from the arterial pressure. The radial arterial curve is not the same as that of the carotid artery, because of distortion produced by damping and reflection through the arterial walls.

The purpose of this clinical study was to determine whether the radial arterial curves of patients with severe AS have distinguishable characteristics in terms of systolic pulse length and pulse intensity. We compared the radial arterial curves of patients with severe AS with those of patients without AS at the induction of anesthesia.

Knowledge about the diagnostic validity of radial arterial pressure curves for identify severe AS is important, because it may lead to prompt echocardiographic examination for AS or, at least, recognition of this possibility may change anesthetic management and reduce perioperative morbidity [3].

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Methods

With institutional approval for this observational study, we collected ECG data and radial artery pressure signals from patients undergoing general anesthesia. Because this study modified neither diagnosis nor interventions, written informed consent was waived. Seventeen asymptomatic patients (age, 74.9 ± 5.9 years) in whom severe AS (defined as an aortic valve area less than 0.8 cm^2) [4] had been demonstrated by preoperative echocardiographic examination and 17 age- and gender-matched control patients (age, 73.9 ± 6.5 years) without AS were studied. Patients with atrial fibrillation or left bundle branch block were excluded from the study. We also excluded patients with a bicuspid aortic valve because this is often associated with aortic coarctation or abnormality of the aortic medial layer [5].

Radial arterial pressure was measured via a disposable fluid-filled catheter system (PX212; Edwards Lifesciences, Tokyo, Japan) after induction of anesthesia. Zero reference was obtained at the mid-axial level. Anesthesia was induced with fentanyl 100 µg i.v. and propofol 1.5 mg/kg i.v. with vecuronium 0.1 mg/kg. The airway was either secured with a laryngeal mask airway or by tracheal intubation. Anesthesia was maintained with inhalation of sevoflurane at 1.5% and with intermittent fentanyl i.v. injection. Measurements were performed after induction of anesthesia but before starting surgery. The ECG and radial pressure signals were analyzed using a PC with an ECG-R synchronization program [6]. Briefly, the two signals were sampled for 10 s at 1000 Hz from a hemodynamic monitor

and transferred to a PC equipped with an analog to digital interface (PCI-3156; Interface Co., Hiroshima, Japan) and custom-made software (R-Synch, Version 1). The software was written by one of the authors (Y.F.). The system provides ECG-R synchronized average traces of the ECG and arterial pressure signals for 10 s, as well as digital readouts of their values (Fig. 1). Arterial pulse pressure, i.e., the difference between systolic and diastolic arterial pressures, was calculated. The durations for the following intervals were automatically measured on the display: from the ECG-R to the beginning of the mean radial arterial pressure wave upstroke (PTT$_{Up}$); from the ECG-R to the peak of the mean arterial pressure (PTT$_{peak}$), and the difference between PTT$_{peak}$ and PTT$_{up}$ (T$_{Upstroke}$). The beginning of the radial arterial pressure upstroke was defined as the point where the differentiated pressure signals reached 20% of the peak differentiated value. The T$_{Upstroke}$ and pulse amplitude were used as indices of the systolic pulse length and pulse intensity of the radial arterial pressure, respectively.

Statistics

All data are presented as means ± SD. We used a one-way analysis of variance to identify any differences between the two groups. A conventional receiver-operating-characteristic (ROC) curve was used to determine the cutoff points that yielded the highest combined sensitivity and specificity with respect to distinguishing between patients with and without severe AS.
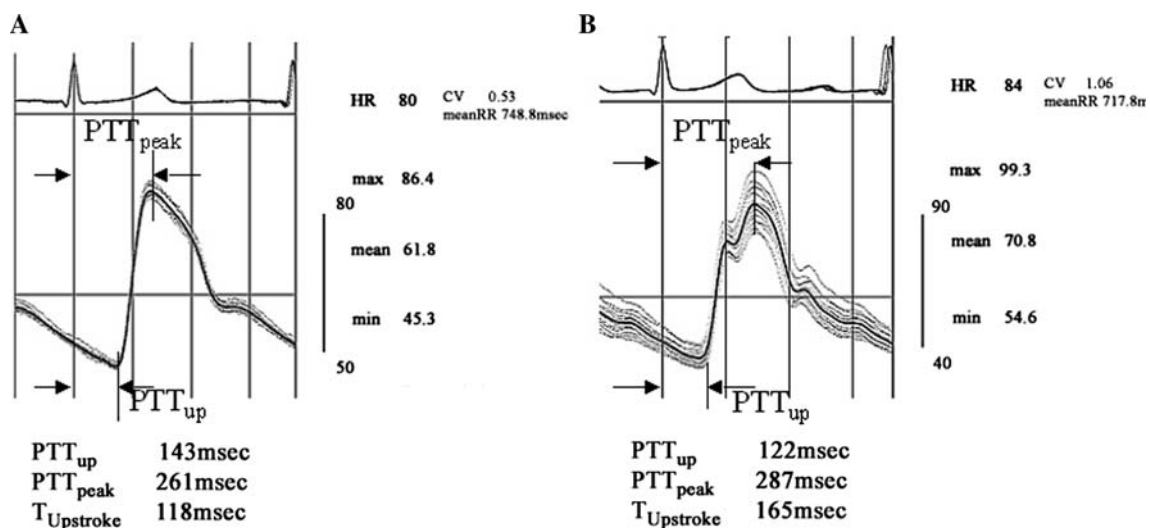
Fig. 1a,b Representative radial arterial pressure forms in **a** a patient without aortic stenosis and **b** a patient with severe aortic stenosis, along with their values. The value from the ECG-R to the beginning of the mean radial arterial pressure wave upstroke (*PTT*$_{Up}$) and that from the ECG-R to the peak of the mean arterial pressure (*PTT*$_{peak}$) were 143 and 261 ms, respectively, in the patient without aortic

stenosis (**a**), while these values were 122 and 287 ms, respectively, in the patient with aortic stenosis (**b**). As a result of the differences in these parameters, the difference between PTT$_{peak}$ and PTT$_{up}$ (*T*$_{Upstroke}$) in the patient with severe aortic stenosis was greater than that in the patient without aortic stenosis (165 vs 118 ms, respectively). *HR*, Heart rate; *CV*, coefficient of variance; *RR*, RR interval

Table 1 Comparisons of control patients and patients with aortic stenosis (AS)

	Control	AS
Patients (*n*)	17	17
Age (years)	74.6 ± 6.6	75.8 ± 7.7
Heart rate (bpm)	63 ± 10	60 ± 12
Systolic arterial pressure (mmHg)	116 ± 24	122 ± 31
Diastolic arterial pressure (mmHg)	51 ± 18	50 ± 13
Arterial pulse pressure (mmHg)	64 ± 27	72 ± 23
PTT_{peak} (ms)	286 ± 64	337 ± 44*
PTT_{up} (ms)	143 ± 21	134 ± 18*
$T_{Upstroke}$ (ms)	143 ± 52	203 ± 38*

Values are presented as means ± SD

PTT_{up} time from the ECG-R to the beginning of the radial arterial pressure wave upstroke, PTT_{peak} time from the ECG-R to the beginning of the radial arterial pressure peak, $T_{Upstroke}$ time from the beginning of the upstroke to the peak of the radial arterial pressure ($PTT_{peak} - PTT_{up}$)

*Significant compared to the control patients ($P < 0.05$)

Results

There were no differences between the two groups in age or hemodynamic data, including heart rate, systolic pressure, and diastolic pressure. The mean radial arterial pulse pressures did not differ significantly (Table 1).

There were statistically significant differences in PTT_{up}, PTT_{peak}, and the $T_{Upstroke}$ between the AS patients and control patients ($P < 0.01$). As a result of the differences in PTT_{up} and PTT_{peak}, the $T_{Upstroke}$ in AS patients was greater than that in the control patients (Table 1).

The ROC curves show the sensitivity and (1-specificity) for various cutoff levels of the $T_{Upstroke}$ with the area under the curve being 0.83 (Fig. 2). The $T_{Upstroke}$ that gave the maximal sensitivity and specificity was 156 ms. At this cutoff point, the diagnostic sensitivity and specificity were 93.1% and 65.0%, respectively.

Discussion

In this study, the $T_{Upstroke}$ was greater in the AS patients than in the control patients, but there were no differences in pulse amplitude between the two groups. These results thus indicate that a prolonged $T_{Upstroke}$ in the radial arterial pressure curve has diagnostic validity for identify severe AS, but pulse amplitude does not.

In the present study, the prolonged $T_{Upstroke}$ in the AS patients resulted from the shorter PTT_{up} and greater PTT_{peak} than those of the control patients. Atherosclerotic calcific degeneration of the aortic valve was thought to be the cause of AS in this study, because the patients were
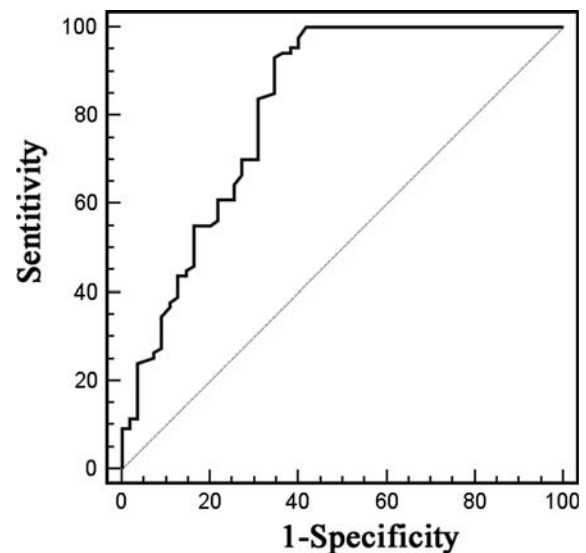
Fig. 2 Receiver-operating-characteristic curve of the $T_{Upstroke}$. The sensitivity (fraction of true positive results) and 1-specificity (fraction of false positive results) of the $T_{Upstroke}$ as a diagnostic value for identify severe aortic stenosis are illustrated. The calculated area under the curve was 0.83

elderly and because patients with a bicuspid aortic valve were not included in this study. Because atherosclerosis affects the entire aorta and arteries, the shorter PTT_{up} may be explained by a fast pulse transit time due to increased stiffness of the aortic and arterial walls [7]. We also speculate that the greater PTT_{peak} is caused by prolonged ejection time and late peaking of left ventricular ejection resulting from aortic valvular obstruction.

A pulsus parvus was not demonstrated in the radial artery pressure in our patients with severe AS, although it is a well-known finding of the carotid pulse in severe AS. The radial arterial pressure curve differs from that of the central aorta or proximal arteries such as the carotid artery [8]. Systolic pressure becomes higher and the dicrotic notch becomes lower as arterial pressure traverses the aorta to a peripheral artery, resulting in a radial arterial pressure curve with a steeper upstroke and flatter diastolic pressure. As a result, the radial arterial pulse pressure becomes greater. We also speculate that the increased stiffness of the arterial wall in the patients with severe AS might have increased the pulse pressure in the radial arterial pressure, because calcific, degenerative AS generally occurs in hypertensive patients [1, 4]. Furthermore, the absence of a pulsus parvus may be interpreted to indicate that stroke volume was relatively preserved in our patients despite the severe AS, because a pulsus parvus is generally common in conditions with diminished left ventricular stroke volume, such as hypovolemic shock and chronic pericarditis. In fact, all the patients in our study were asymptomatic, and were thought to be in a compensated phase hemodynamically.

In contrast to our findings, Christen et al. [4] found significantly lower systolic arterial pressure and pulse pressure in patients with severe AS than in those without AS. The differences, however, were clinically insignificant (9 and 7 mmHg, for AS patients and those with a normal valve, respectively).

AS is the most common cardiac valve disease in the aged population [7]. It exhibits few clinical symptoms such as angina, syncope, dyspnea, or other symptoms of heart failure until the valve area is severely reduced. Because patients with AS are at significant risk with regard to perioperative morbidity and mortality, the diagnosis of AS and proper anesthetic management, such as the maintenance of normovolemia and sinus rhythm and the avoidance of hypotension and tachycardia, are essential [3]. The importance of preanesthetic evaluation cannot be overemphasized. The present study indicated that a late systolic peak in the radial arterial pressure curve provided a clue for the diagnosis of severe AS. On the other hand, the pulse pressure of the radial arterial pressure did not have any diagnostic validity in asymptomatic patients with severe AS.

In conclusion, our study using the radial arterial curve validated a pulsus tardus (more than 156 ms; sensitivity 93.1%, specificity 65%) as a diagnostic sign of severe AS, while the validity of a pulsus parvus in identify severe AS was not confirmed. If a late peaking is recognized in the radial arterial pressure curve perioperatively, auscultation for a systolic ejection murmur at the right sternal border and an echocardiographic examination may be advisable to obtain a definitive diagnosis of severe AS.

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