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# Assessment of pulse transit time to indicate cardiovascular changes during obstetric spinal anaesthesia†

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**Background.** Pulse transit time (PTT) measurement may provide rapidly available beat-to-beat cardiovascular information when conditions change quickly and routine invasive arterial pressure measurement is not justified, for example during obstetric spinal anaesthesia.

**Method.** We obtained ethics approval for an observational study of PTT during the onset of spinal anaesthesia in patients having elective or urgent Caesarean section. PTT was measured as the difference in time between the peak of the ECG R wave and the upstroke of the toe plethysmograph. Arterial pressure was measured by non-invasive sphygmomanometry.

**Results.** We analysed data from 58 normotensive patients and 15 patients with pregnancy-induced hypertension (PIH). PTT increased with the onset of spinal anaesthesia as arterial pressure decreased. An increase of 20% in PTT was 74% sensitive and 70% specific in indicating a decrease in mean arterial pressure of more than 10%. Changes in PTT were related to changes in mean arterial pressure ($r^2$=0.55, $P$<0.0001). Arterial pressure changes were greater and PTT increased significantly more quickly in the normotensive patients than in the patients with hypertension [median, quartiles: 32 (14, 56) ms $min^{-1}$ compared with 7 (6, 18) ms $min^{-1}$; $P$<0.01, Mann–Whitney $U$-test]. However, the relationship between PTT and arterial pressure was similar for the normotensive patients and the patients with PIH.

**Conclusion.** PTT measurement gave a beat-to-beat indication of arterial pressure during spinal anaesthesia, and could be developed to allow prediction of the onset of hypotension.

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In clinical conditions, arterial pressure may change so quickly that intermittent non-invasive measurements may be too slow and inaccurate to allow early detection and prompt treatment, especially in obese subjects. However, routine invasive measurement may be inappropriate, for example in obstetric spinal anaesthesia, where hypotension is the most frequent complication and poses risks to both mother and foetus. In conscious subjects, arm movement can delay the display of an arterial pressure reading through two or three measurement cycles, often at a time when changes may be considerable. An additional non-invasive measurement that could give early warning of arterial pressure change would be useful clinically.

Pulse transit time (PTT) measurement offers beat-to-beat cardiovascular information.[1] Such measurements have been used previously to infer changes in autonomic activity[2] and arterial pressure.[3] PTT, measured as the interval from the ECG R wave to the pulse plethysmograph upstroke, was used recently to assess cardiovascular responses to anaesthesia and intubation.[4] Both the ECG and the plethysmograph wave can be obtained from standard monitoring equipment. We used a custom-built analogue device to acquire automatically the interval between the ECG R wave and the pulse plethysmograph upstroke.

PTT is of clinical interest as an index of arterial stiffness and hence of arterial pressure,[3 5] since arterial stiffness increases as arterial pressure increases.[6–8] However, other factors may affect arterial stiffness. For example, recent studies suggest that hypotension following spinal

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anaesthesia is less likely in patients with pregnancy-induced hypertension (PIH) than in normotensive patients.[9] Changes in PTT following spinal anaesthesia may indicate differences in arterial stiffness in these patients.

The prime aim of this study was to observe PTT in a clinical scenario where sudden onset of hypotension is relatively frequent, and assess its value for predicting such changes. A secondary aim was to compare the responses of patients with and without PIH.

## Methods

The local ethics committee approved collection and recording of data from routine cardiovascular monitoring devices, but not the modification of routine management in any other way. We obtained informed verbal consent for the data collection. We recorded PTT during the onset of spinal anaesthesia in non-labouring women having Caesarean section for routine elective or urgent indications. Patients were recruited as they presented over a 6-month period; of these, 74 were normotensive. Eighteen patients had severe PIH, defined using standard criteria[10] as hypertension which developed after 20 weeks of gestation and required antihypertensive medication with nifedipine, labetalol or methyldopa, singly or in combination. We included patients with or without proteinuria. We undertook this study before our unit introduced i.v. magnesium sulphate treatment for severe PIH.

The values were obtained from before the spinal anaesthetic to the time the patient was ready for surgery. Vasopressor or vagolytic drugs were given by the clinician managing the anaesthesia, according to normal practice, in response to changes in arterial pressure, heart rate, or the onset of symptoms suggestive of hypotension, such as dizziness, nausea or vomiting. Some of these clinicians did not routinely give vasopressor agents prophylactically, others did, and some gave them occasionally.

Patients were placed in a supine wedged position and an infusion of Hartmann's solution was started. ECG monitoring and an automated arterial pressure (NIBP) recording cuff were applied (Cardiocap 2; Datex). The baseline arterial pressure was recorded as the mean of three measurements taken at 2-min intervals. An oximeter probe was placed on the second toe of the left foot. Spinal anaesthesia was then administered with the patient in either the sitting or left lateral position. A 24 gauge Sprotte needle was used to give between 2.5 and 2.7 ml of hyperbaric bupivacaine 0.5% with diamorphine 0.3 or 0.4 mg according to the anaesthetists' preference. The patient was then returned to the wedged supine position. The time was recorded, and the events that were marked electronically included the following: the connection of monitoring equipment; the initial change of position for the spinal; the return to a wedged supine position; the administration of vasopressor or other i.v. drugs; and the transfer of the patient to the operating theatre. IV fluids given before spinal anaesthesia and the total given over the study period were recorded. Heart rate and NIBP were recorded at 2-min intervals. The ECG and photoplethysmograph signals from the analogue output of the Cardiocap monitor were transferred to a purpose-built analogue computer constructed by Leiden University. This computed the time between the peak of the ECG R wave and the maximum rate of the plethysmograph wave upswing. The time intervals and digital signals from the Datex monitor were recorded in digital form on a Satellite Pro 4300 (Toshiba) laptop computer.

Before data analysis, spurious ECG and photoplethysmograph signals generated by patient movement were removed. These artefacts were defined using an Excel function (Excel version 9.0, 1999; Microsoft, Redmond, WA, USA) as values that were 20% less or greater than the rolling mean PTT, and were filtered from the data before analysis.

Statistical analysis was with GraphPad Prism version 3.02 and Analyse-It software for Excel, version 1.71 (Analyse-it Software, Leeds, UK). Data are presented as medians (quartile values) unless stated otherwise.

## Results

Ninety-two patients were studied; we obtained data suitable for analysis from 58 normotensive patients and 15 with PIH. There were no obvious systematic differences in the reasons for exclusion between the two groups of patients (Table 1). The two groups were similar in respect of height and weight, but, as might be expected, heart rate was less, arterial pressure was greater and gestational age was less in the patients with PIH (Table 2). Before the spinal anaesthetic, normotensive patients were given 400 (300, 500) ml of Hartmann's solution and the patients with PIH received 150 (100, 225) ml. The dose of bupivacaine was 13 (12.5, 13.5) mg in both the normotensive and the hypertensive patients. Most patients received diamorphine 300 μg, but nine were given 400 μg. Ephedrine had to be given to 46 of the normotensive patients and three of the patients with PIH ($P<0.001$); other vasopressor and vagolytic agents (phenylephrine, atropine and glycopyrollate) were also used more frequently in the normotensive patients.

Data from five patients were not analysed because vasopressor drugs were given prophylactically by personal preference of the anaesthetist immediately after spinal

**Table 1** Reasons for exclusion from analysis

| | Normotensive | PIH |
|---|---|---|
| Total number before exclusions | 74 | 18 |
| Reasons for exclusion | | |
| Inadequate data | 9 | 1 |
| Vasopressor given prophylactically | 5 | 0 |
| Computer failure | 1 | 0 |
| Conversion to general anaesthetic | 1 | 0 |
| Insulin-dependent diabetes | 0 | 1 |
| Essential hypertension | 0 | 1 |
| Number of patients analysed | 58 | 15 |

**Table 2** Patient details and cardiovascular measurements before and after spinal anaesthesia. Values are median (interquartile values). ns, not significant. Student's *t*-test, **P*<0.05, ***P*<0.0001; Mann–Whitney *U*-test, #*P*<0.01

| | Normotensive subjects | Pregnancy-induced hypertension | Significance |
|---|---|---|---|
| Patient details | | | |
| Number | 58 | 15 | |
| Age | 32 (30, 35) | 32 (30, 37) | |
| Height (cm) | 162 (158, 168) | 162 (160, 170) | |
| Weight (kg) | 70 (61, 78) | 72 (62, 83) | |
| Gestation (weeks) | 39 (39, 39) | 36 (33, 38) | |
| Cardiovascular values | | | |
| Before spinal anaesthesia | | | |
| Heart rate (beats $min^{-1}$) | 89 (78, 102) | 80 (74, 94) | * |
| Arterial pressure (mm Hg) | | | |
| Mean | 99 (91, 104) | 115 (104, 119) | ** |
| Systolic | 126 (118, 138) | 144 (138, 160) | ** |
| Pulse transit time (ms) | 390 (345, 422) | 353 (325, 399) | * |
| After spinal anaesthesia | | | |
| Heart rate (beats $min^{-1}$) | 87 (78, 109) | 80 (71, 101) | ns |
| Arterial pressure (mm Hg) | | | |
| Mean | 92 (86, 99) | 106 (98, 111) | ** |
| Systolic | 124 (116, 136) | 140 (136, 154) | ns |
| Pulse transit time (ms) | 413 (373, 454) | 370 (342, 417) | * |
| Measurements at greatest pulse transit time or first intervention | | | |
| Heart rate (beats $min^{-1}$) | 96 (78, 109) | 79 (75, 98) | ns |
| Mean arterial pressure (mm Hg) | 77 (66, 90) | 89 (98, 111) | ** |
| Change in mean arterial pressure | 18 (5, 33) | 22 (11, 29) | ns |
| Systolic arterial pressure (mm Hg) | 106 (92, 120) | 119 (111, 134) | ns |
| Pulse transit time (ms) | 488 (429, 532) | 417 (389, 495) | * |
| Greatest change in PTT (ms) | 94 (62, 123) | 75 (54, 118) | ns |
| Greatest change in PTT (%) | 24% | 21% | ns |
| Rate of change in PTT (ms $min^{-1}$) | 32 (14, 56) | 7 (6, 18) | # |
| Time to greatest PTT change (min) | 2.4 (1.4, 3.4) | 5.0 (3.2, 7.7) | # |
| Minimum arterial pressure after spinal anaesthesia | | | |
| Mean | 69 (60, 78) | 83 (78, 95) | ** |
| Systolic | 97 (87, 108) | 116 (108, 126) | ** |

anaesthesia and before PTT and arterial pressure recordings could be obtained. A standardized format of clinical practice was not imposed in this observational study. In 10 patients, technical problems with recording either PTT or arterial pressure yielded insufficient data for analysis.

Mean arterial pressure (MAP) before spinal anaesthesia was 99 (91, 104) mm Hg in the normotensive patients and 115 (104, 119) mm Hg in patients with PIH. With the onset of spinal anaesthesia, MAP decreased by 18 (5, 33) and 22 (11, 29) mm Hg in normotensive and PIH patients respectively.

Patient movement and repositioning after carrying out the block disturbed the ECG and plethysmograph signals, and could cause spurious PTT values. These were less than 3% of the total values obtained. Before anaesthesia, PTT was significantly less in the patients with PIH: 353 (325, 383) ms ($P$<0.05) compared with 390 (346, 417) ms in the normotensive group ($P$<0.05). PTT changed during the onset of spinal anaesthesia (Fig. 1) by 24% in the normotensive group and 21% in the PIH group. The greatest change in PTT occurred 2.4 (1.4, 3.4) min after spinal anaesthetic in the normotensive group and 5.0 (3.2, 7.7) min after spinal anaesthetic in the hypertensive group. Thus, PTT increased more rapidly in the normotensive patients [32 (14, 56) ms $min^{-1}$] than in the patients with PIH [7 (6, 18) ms $min^{-1}$] ($P$< 0.01, Mann–Whitney *U*-test).

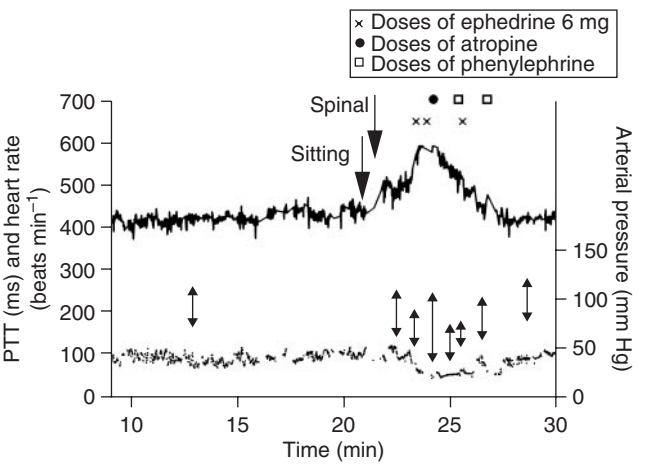


**Fig 1** An example of patient responses. Upper, continuous trace: pulse transit time (ms). Lower, discontinuous trace: heart rate calculated from successive R-R intervals. Systolic and diastolic arterial pressures are shown as vertical bars and arrowheads.

The relationship between PTT and MAP was examined before and after spinal anaesthesia (Fig. 2). There was a

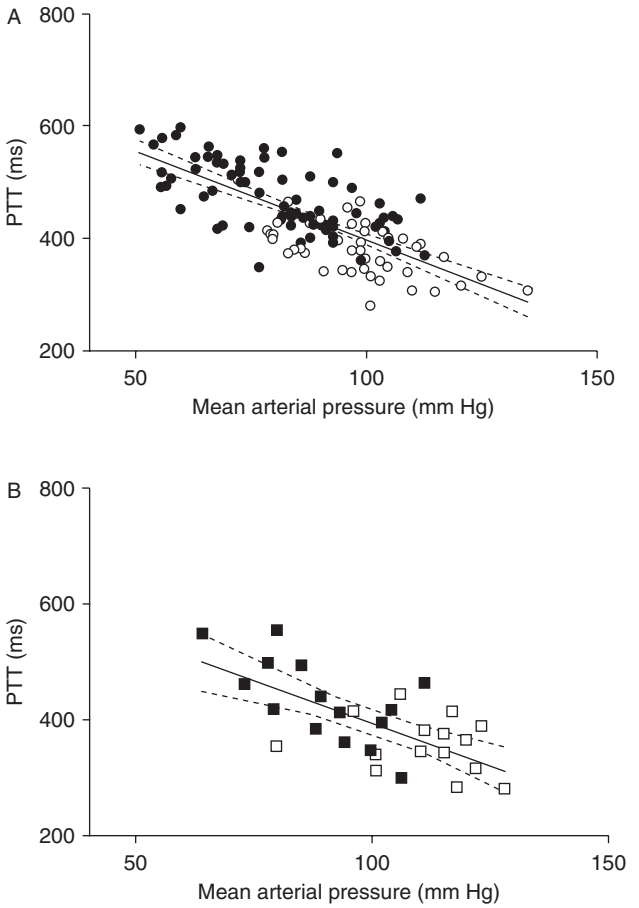


**Fig 2** Relationship of pulse transit time to mean arterial pressure in (A) normotensive subjects and (B) patients with pregnancy-induced hypertension. The open symbols are before and the closed symbols after spinal anaesthesia. The linear regression relationships are shown with the 95% confidence interval for the line. Both relationships are significant ($P$<0.0001).

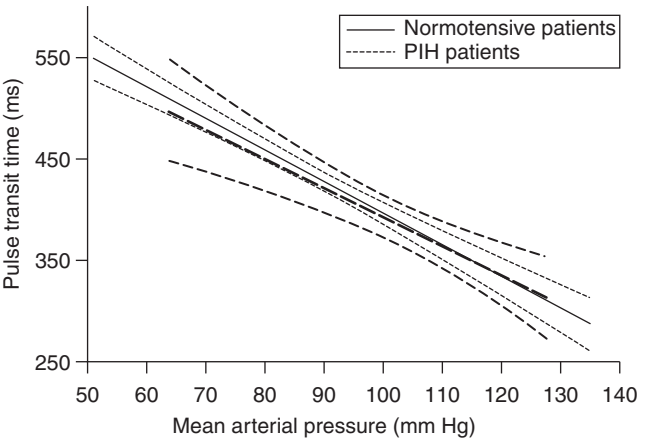


**Fig 3** Comparison of the linear regression relationships of pulse transit time and mean arterial pressure in normotensive subjects and patients with pregnancy-induced hypertension. The outer lines represent the 95% confidence interval for the regression.

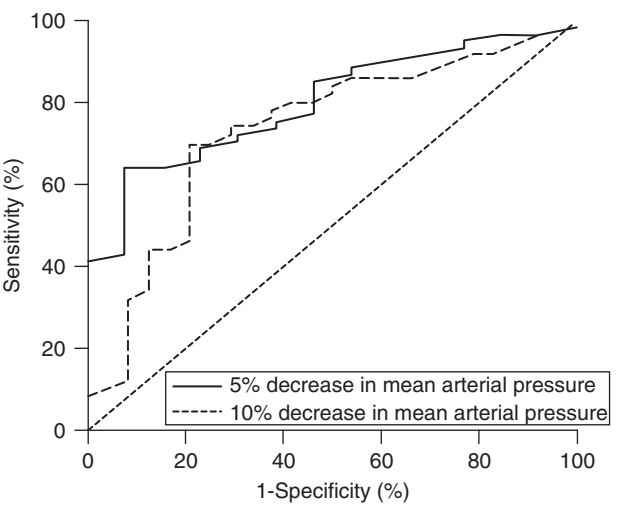


**Fig 4** Receiver operating characteristic curves for changes in pulse transit time in relation to a decrease in mean arterial pressure by 5 and 10%. For the 5% decrease, the area under the curve is 0.79 (0.68–0.90) and for the 10% decrease the area is 0.73 (0.60–0.85) (95% confidence intervals) Both areas are significantly greater than 0.5 ($P$<0.001).

significant correlation in both the normotensive ($r^2$=0.55, $P$<0.0001) and the PIH group ($r^2$=0.45, $P$<0.0001) (Fig. 3). The slopes of the relationships between PTT and MAP were not different when normotensive and hypertensive subjects were compared [slopes (95% confidence interval, CI) were −3.12 (−3.64, −2.59) and −2.88 (−4.12, −1.64) ms (mm Hg)$^{-1}$ respectively].

We examined the sensitivity and specificity of changes in PTT to indicate the onset of hypotension. The resulting receiver operating characteristic (ROC) curves for decreases in MAP of 5 and 10% are given in Figure 4. For a decrease in MAP of 5%, an increase of 10% in PTT was 95% sensitive but only 15% specific. If a cut-off value of a 20% increase in PTT was taken, the sensitivity and specificity were 69 and 77% respectively. The area under the ROC curve was 0.79 (95% CI, 0.68–0.9; $P$<0.001 compared with an area of 0.5). Considering decreases in MAP of more than 10%, an increase of 20% in PTT was 74% sensitive and 70% specific. The area under the ROC curve was 0.72 (95% CI, 0.60–0.85; $P$<0.001 compared with an area of 0.5).

## Discussion

To our knowledge, the relationship between PTT and arterial pressure has not previously been studied systematically during obstetric spinal anaesthesia. We chose to study this scenario because rapid and substantial changes in arterial pressure are relatively frequent. We found that changes in PTT were related to arterial pressure changes and that the relationship between PTT and arterial pressure was the same in normotensive patients and those with PIH.

We measured the time interval between the ECG R wave and the upsweep of the plethysmograph.[11 12] This time includes two principal components, the time between electrical activation of the ventricle and cardiac ejection, and the

time taken for the resultant pressure wave to be transmitted along the artery to generate the plethysmograph upstroke.[11] The time from ventricular activation to cardiac ejection depends upon a number of factors related to preload, heart rate and contractility.[13,14] This time is small compared with the time taken for pulse wave transmission along the vessel, particularly in young normotensive patients. Consequently, the greater part of the PTT we measured indicates vascular elastance according to the Bramwell–Hill relationship[15] described for the velocity of pressure waves:

$$\text{Velocity} = \sqrt{(\Delta P/[V/\Delta V]\times\rho)}$$

where ρ is the density of blood and ΔP/[V/ΔV] is the specific elastance of the vessel. The pressure–volume relationship of arteries is non-linear: as pressure decreases, elastance decreases, pulse wave velocity decreases and PTT is increased, as we have confirmed. Other factors that affect vascular elastance, such as hypertension and change in sympathetic activation, could also affect PTT, but these influences are disputed. For example, although ultrasound estimates of radial artery elastance suggest that arterial infusion of phenylephrine can increase elastance,[16] others have concluded that greater arterial elastance in hypertensive patients can be explained entirely by differences in arterial pressure.[7,17] These findings are supported by the present study, in which we found no discernible difference between normotensive patients and those with PIH in the relationship for arterial pressure and PTT. We conclude that the mechanical properties of the large conducting vessels in patients with this condition are not affected, whereas the resistance vessels are clearly affected. This finding is not altogether unexpected: the site of modulation of arterial resistance depends upon the type of stimulus.[18]

The shape of the pressure waveform in peripheral arteries varies considerably with age and disease.[19] The rate of increase of the pressure varies considerably with age, but in the limited range of ages that we studied this could not cause much variation.

The analogue device that we used detected the maximum rate of change of the plethysmograph waveform. In preliminary unpublished studies of healthy volunteers, we used a method of intersecting tangents to determine the nadir of the plethysmograph waveform. Measurements of PTT using this nadir were less affected by changes in heart rate than measurements made using the time to the maximum rate of change of the plethysmograph signal. These findings confirm those of others.[20] However, this method was not compatible with analogue preprocessing, in that we not could detect the maximum rate of change of the plethysmograph wave. It was therefore not practical for this study. Thus, one source of variation in the relationship between PTT and arterial pressure could result from heart rate changes and the method used by our analogue detector.

There is a difference in time between the occurrence of plethysmograph waveforms in the finger and toe because of differences in distance along the arteries. Epidural anaesthesia increases this time difference and was attributed by the investigators to sympathetic blockade in the leg arteries.[21] However, the time difference only increased between 10 and 20% and this increase was accompanied by a decrease in arterial pressure. Consequently, an equally plausible alternative explanation is that hypotension, causing a proportional increase in pulse wave transmission time, would have a greater absolute effect in the longer vessel. This effect can account for the changes reported by these workers.

Recently, marked changes in PTT were described during general anaesthesia in association with tracheal intubation, and these changes were attributed to autonomic activation.[4] However, no measurements of arterial pressure were reported. Once again, the changes could have been caused by changes in arterial pressure, because the hypertensive response to insertion of the tracheal tube will increase arterial elastance and reduce PTT, as the authors reported.

Monitoring by means of PTT has been compared with invasive arterial pressure measurements. If the directly measured pressure changed by more than 10 mm Hg, then PTT accurately tracked the change on 67% of occasions. However, the authors of this study concluded that PTT did not have sufficient accuracy to replace direct arterial measurements.[22]

Clinically, PTT changes are of interest as a non-invasive beat-to-beat index of arterial pressure changes. Increased arterial pressure itself causes increased arterial stiffness but the relationship is non-linear at high and low pressures.[23] In the present study, by using a large control group, in which there were considerable changes in PTT, we found a correlation between MAP and PTT changes. More than 50% of the variance in PTT is explained by the value of the MAP. The remainder of the variance must result from other factors, such as patient size, variation in accuracy of estimates of both arterial pressure and PTT, and individual variations in vessel wall characteristics. Arterial behaviour can be altered by obesity,[24] longitudinal tension[25] and vasoactive mediators.[16] Although these additional factors increase the variation between subjects, it is likely that they will not influence the variation within an individual, so PTT can be a useful measure of moment-to-moment changes within a particular patient.

Our results were obtained in pregnant subjects, and the mechanical properties of large vessels such as the aorta can be affected by hormonal changes such as may occur in pregnancy.[26] However, the vascular changes of PIH are probably confined to small resistance vessels,[27,28] explaining the similar relationship between PTT and arterial pressure in normotensive and PIH patients.

Non-invasive methods for arterial pressure measurement, such as automated sphygmomanometers, frequently fail to display values when patient movement causes interference.

This is a particular problem in obstetric anaesthesia, where the subjects are awake and may be agitated and the procedures are often urgent, with little opportunity for careful cuff application and even less for arterial cannulation. We found that PTT could potentially be used, in these circumstances, to predict the onset of hypotension. The sensitivity and specificity are sufficient to indicate instantaneously changes in arterial pressure and provide a rapid, non-invasive, within-subject indication of hypotension. This may be of considerable value if invasive monitoring is not justified. We found no evidence that the properties of large arteries are affected by PIH.

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## References


1 Smith RP, Argod J, Pepin J-L, Levy PA. Pulse transit time: an appraisal of potential clinical applications. *Thorax* 1999; **54**: 452–8

2 Weiss T, Del Bo A, Reichek N, Engelman K. Pulse transit time in the analysis of autonomic nervous system effects on the cardiovascular-system. *Psychophysiology* 1980; **17**: 202–7

3 Sawada Y, Yamakoshi K. A correlation analysis between pulse transit time and instantaneous blood pressure measured indirectly by the vascular unloading method. *Biol Psychol* 1985; **21**: 1–9

4 Singham S, Voss L, Barnard J, Sleigh J. Nociceptive and anaesthetic-induced changes in pulse transit time during general anaesthesia. *Br J Anaesth* 2003; **91**: 662–6

5 Landowne M. A method using induced waves to study pressure propagation in human arteries. *Circ Res* 1957; **5**: 594–601

6 Bramwell JD, Downing AC, Hill AV. The effect of blood pressure on the extensibility of the human artery. *Heart* 1923; **10**: 289–95

7 Gribbin B, Pickering TG, Sleight P. Arterial distensibility in normal and hypertensive man. *Clin Sci* 1979; **56**: 413–7

8 Schulze-Bauer CAJ, Holzapfel GA. Determination of constitutive equations for human arteries from clinical data. *J Biomech* 2003; **36**: 165–9

9 Clark VA, Sharwood-Smith GH, Stewart AVG. Ephedrine requirements are reduced during spinal anaesthesia for caesarean section in preeclampsia. *Int J Obstet Anaesth* 2005; **14**: 9–13

10 Davey DA, MacGillivray I. The classification and definition of the hypertensive disorders of pregnancy. *Am J Obstet Gynecol* 1988; **158**: 892–8

11 Steptoe A, Godaert G, Ross A, Schreurs P. The cardiac and vascular components of pulse transmission time: a computer analysis of systolic time intervals. *Psychophysiology* 1983; **20**: 251–9

12 Bugram R, Akhlaghi-Farsi H, Maresch H, Pfurtscheller G. A method for determining pulse transmission time. *Biomed Tech* 1994; **39**: 51–6

13 Weissler AM, White GD, Harris LC. Left ventricular ejection time index in man. *J Appl Physiol* 1963; **18**: 919–31

14 Weissler AM, Peeler RG, Roehll WH Jr. Relationships between left ventricular ejection time, stroke volume, and heart rate in normal individuals and patients with cardiovascular disease. *Am Heart J* 1961; **62**: 367–74

15 Bramwell JC, Hill AV. The velocity of the pulse wave in man. *Proc R Soc B* 1922; **93**: 298–306

16 Grassi G, Giannattasio C, Failla M, *et al.* Sympathetic modulation of radial artery compliance in congestive heart failure. *Hypertension* 1995; **26**: 348–54

17 Laurent S. Arterial wall hypertrophy and stiffness in essential hypertensive patients. *Hypertension* 1995; **26**: 355–62

18 Folkow B, Sonnenschein RR, Wright DL. Loci of neurogenic and metabolic effects on precapillary vessels of skeletal muscle. *Acta Physiol Scand* 1971; **81**: 459–71

19 McLean CE, Clason WPC, Stoughton PV. The peripheral pulse as a diagnostic tool. *Angiology* 1964; **14**: 221–31

20 Chiu YC, Arand PW, Shroff SG, Feldman T, Carroll JD. Determination of pulse-wave velocities with computerized algorithms. *Am Heart J* 1991; **121**: 1460–70

21 Babchenko A, Davidson E, Adler D, Ginosar Y, Kurz V, Nitzan M. Increase in pulse transit time to the foot after epidural anaesthesia treatment. *Med Biol Eng Comput* 2000; **38**: 674–9

22 Young CC, Mark JB, White W, DeBree A, Vender JS, Fleming A. Clinical evaluation of continuous noninvasive blood pressure monitoring: accuracy and tracking capabilities. *J Clin Monit* 1995; **11**: 245–52

23 Schulze-Bauer CAJ, Holzapfel GA. Determination of constitutive equations for human arteries from clinical data. *J Biomech* 2003; **36**: 165–9

24 Toto-Moukouo JJ, Achimastos A, Asmar RG, Hugues CJ, Safar ME. Pulse wave velocity in patients with obesity and hypertension. *Am Heart J* 1986; **112**: 136–40

25 Wang YY, Jan MY, Wang GC, Bau JG, Wang WK. Pressure pulse velocity is related to the longitudinal elastic properties of the artery. *Physiol Meas* 2004; **25**: 1397–403

26 Chelsky R, Wilson RA, Morton MJ, *et al.* Alteration of ascending thoracic aorta compliance after treatment with menotropin. *Am J Obstet Gynecol* 1997; **176**: 1255–9

27 McCarthy AL, Woolfson RG, Raju SK, Poston L. Abnormal endothelial cell function of resistance arteries from women with preeclampsia. *Am J Obstet Gynecol* 1993; **168**: 1323–30

28 Suzuki Y, Kajikuri J, Suzumori K, Itoh T. Mechanisms underlying the reduced endothelium dependent relaxation in human omental resistance artery in pre-eclampsia. *J Physiol (Lond)* 2000; **527**: 163–74