Squatting to standing: an unusual but powerful postural manoeuvre to investigate human arterial blood pressure regulation

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Short title : Squatting to standing postural manoeuvre

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SUMMARY

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1. The aim of this work is to assess the utility of the scarcely used squatting to standing manoeuvre to investigate the orthostatic arterial blood pressure regulation in clinical practice.
2. Two groups of healthy subjects (n=10, n=42) performed squatting to standing manoeuvre. Arterial blood pressure was continuously measured at a finger by means of the Finapres device (Ohmeda) and averaged to obtain beat to beat mean arterial blood pressure (MAP). Reproducibility and effect of ageing are submitted to the Zerbe-Kempthorn randomization analysis.
3. Change from squatting to standing position induces a very reproducible and prompt decrease of MAP with a nadir of -27 (SEM = 7) mmHg (nearly twice the value observed during sitting to standing) after 7 s. The MAP level observed before the manoeuvre is only partially restored after 50 s. Heart rate varies inversely to MAP and is sligthly delayed. It suggests that this postural stress activates the arterial baroreflex.
4. Triggering an abrupt, deep and reproducible hypotension, being easy to perform, the squatting to standing manoeuvre appears, as suggested by Rossberg et al [1] in 1988, to be an appropriate way to investigate the orthostatic blood pressure regulation in clinical practice. To some aspects (for instance in the study of the effect of ageing), this postural change appears to be better than more conventional ones such as sitting to standing manoeuvre.

INTRODUCTION

Since the eighteenth century, it is well known that many patients with cyanotic congenital heart disease spontaneously adopt squatting posture at rest, especially after exercise [2]. More recent studies using haemodynamic investigation explained this surprising sign as an effective way to increase filling of the heart, thereby improving arterial oxygen saturation [3-6]. On the contrary, squatting to standing for subjects without heart failure induces an abrupt hypotension which is rapidly corrected [3].

These complex haemodynamic effects of squatting explain why this postural manoeuvre (standing to squatting or squatting to standing) is often used today in bedside diagnosis of systolic murmurs [7].

Recently, Rossberg and Penaz, using a non-invasive technique of beat to beat blood pressure measurement, confirmed immediate arterial hypotension during squatting to standing and suggested that this manoeuvre could be used to study orthostatic cardiovascular regulation in man [1].

The aim of this work is to submit Rossberg and Penaz's suggestion to experimental verification.

METHODS

Subjects

Experiments were performed in two sets of healthy subjects, normotensive, without medication and especially chosen for absence of orthostatic hypotension. Consent of the subjects was obtained after explanation of the purpose and risk of the procedures. The investigations were made according to the recommendations of the Ethical Committee of the Hospital of the University of liège and all experiments were carried out under medical supervision.

In the first group, composed of 10 male and female subjects (25-56 years old), the sequence: standing-squatting-standing was repeated three times to allow the study of the manoeuvre reproducibility.

The second group gathered 42 male and female subjects (21-76 years old). The oldest ones were sampled in an third age training group. They performed once the squatting to standing and sitting to standing manoeuvres in order to study the ageing effect on short term blood pressure control.

Orthostatic manoeuvres

All the experiments were conducted as follows in the early evening.

Standing-squatting-standing. Blood pressure was first monitorized in erect position at rest (during at least 1 min), then during 30 s after squatting and lastly, after quick standing, during 90 s.

Sitting to standing. After at least 1 min of sitting, subjects abruptly standed up and maintained upright position during 50 s.

Squatting to standing. After 30 s of squatting, subjects abruptly standed up and maintained erect position during 50 s.

Measurements

Arterial blood pressure was continuously measured at a finger by means of the Finapres device (Ohmeda) based on the Penaz's photoplethysmographic technique [8]. During the whole duration of measurement, the finger cuff was maintained at heart level.

Data analysis

The analog output of the Finapres device was sampled on-line at the rate of 150 Hz, by a 12 bits A/D converter (Das8, Metrabyte), fed to an AT compatible PC and stored in a disk file. In a second step, required for statistical analysis, beat-to-beat mean arterial pressure (MAP) and heart rate (HR) were recognized from that record and linearly interpolated at each second in relation to the temporal positions of the postural changes.

Statistical analysis

It must be pointed out that in this work, variations of MAP and HR (i.e. deviations from the mean value calculated on the observations preceding the first postural change) are used rather than the primary values of MAP and HR. These variations are respectively called ΔMAP and ΔHR.

In the study of reproducibility of the time course of ΔMAP and ΔHR over the three consecutive experiments performed by the subjects of the first group, the randomization analysis of randomized blocks design extended to response curves described by Zerbe is applied [9]. This statistical technique allows to decide whether or not the mean response curve remains unchanged in several experiments. The analysis may be performed over any interval of time. As in the corresponding classical randomized blocks design, a total sum of squares is decomposed in three components: one due to the experiments, a second one due to the individuals (blocks) and finally a residual sum of squares. Those sums of squares are obtained by integrating over the considered time interval, the corresponding sums in the classical design for fixed time. A F-statistic is then computed as usual, but the sampling distribution must be approximated by a Snedecor F-distribution with appropriate degrees of freedom. We test whether the mean time course remains unchanged during the three experiments. The peaks of ΔMAP and ΔHR during the postural manoeuvres (abbreviated by ΔMAP_{max} and ΔHR_{max}) are also submitted to analysis of reproducibility (classical two-way ANOVA).

In the study of ageing effect on the mean time course of ΔMAP and ΔHR, the randomization analysis of the completely randomized design extended to response curves proposed by Zerbe is used [10]. This technique is specially designed to compare the mean response curves of several groups of patients during an arbitrary time interval. As in the classical one-way ANOVA, the technique is based on a decomposition of a total sum of squares into a between groups sum of squares and a residual one. Those sums are calculated by integrating over the time interval, the corresponding sums in the classical design for fixed time. A F-statistic is again computed as usual, but its sampling distribution must be approximated by a Snedecor F-distribution with appoprlate degrees of freedom. We test here whether there is no effect of ageing on the mean time course of ΔMAP and ΔHR. The mean values of ΔMAP and ΔHR for the last 25 seconds of the recordings are also used to study the effect of ageing in both manoeuvres (squatting to standing and sitting to standing) and submitted to classical one-way ANOVA.

All the analysis are performed by an inhouse program written in Turbo Pascal.

RESULTS

Standing-squatting-standing: time course of MAP and HR

Fig.1 Fig. 1 illustrates a typical continuous recording of arterial blood pressure during a standing-squatting-standing manoeuvre, first during the entire observation and secondly, at another scale, around the postural changes. Mean variations of beat-to-beat MAP and

HR for the subjects of the first group are depicted in Fig. 2 (time origin is fixed at the first postural change).

Fig. 2 Squatting induces a brief hypertension which is resumed in 10 s. Then, pressure stabilizes at a level somewhat higher than the previous standing value. The evolution of HR is more complex: tachycardia first appears during 3 s, followed by an abrupt bradycardia. Finally, bradycardia progressively regresses and initial standing HR is always totally recovered after 30 s.

Change from squatting to standing position induces a prompt decrease of MAP, with a nadir of -27 mmHg after 7 seconds. The level is restored at the previous standing one after about 35 s. Heart rate varies inversely to MAP, but its changes are slightly delayed.

Reproducibility of the standing-squatting-standing manoeuvre

The test of reproductibility of the mean time course of ΔMAP gives no reason for non reproductibility during the whole time interval [-5 s, 105 s] of the observations (F=1.142 d.f.num=11.6 d.f.den.=104.6 P=0.46). The same result is true for the mean time course of ΔHR during the time interval [25 s, 105 s] after the beginning of the first manoeuvre (F=0.923 d.f.num.=18.6 d.f.den.=167.4 P=0.59). Unfortunately, during the time interval [-5 s, 25 s], the test leads to the rejection of the reproductibility hypothesis (F=5.054 d.f.num.=8.5 d.f.den.= 76.1 P<0.01). Returning to the data during this time interval (see Fig. 3), one can see that during this time interval, the mean time course of ΔHR recorded in the third repetition, differs indeed from those recorded in the two first repetitions. This discrepancy is probably due to experimental procedure subject habituation. Table 1 gives the F and corresponding P-values for the test of reproductibility of the peaks of ΔMAP and ΔHR. These results confirm the above-mentioned results.

Fig. 3
Table 1

Sitting to standing versus squatting to standing: effect of ageing

Fig. 4, 5 The subjects of the second group are classified in three age-groups (20 to 39 years old n=18, 40 to 59 years old n=13, 60 to 79 years old n=11). Fig. 4 and Fig. 5 describe variations of MAP and heart rate during the two postural manoeuvres. After sitting-to-standing, time course and magnitude of the variations of ΔMAP appear identical in the three age-groups. On the contrary, squatting-to-standing reveals a lesser restoration in the aged subjects. As often described, variations of ΔHR are blunted in the aged subjects.

Table 2 From a statistical point of view, Table 2 gives the F and corresponding P-values when testing the effect of ageing on the mean time course of ΔMAP and ΔHR during both manoeuvres (squatting to standing and sitting to standing). Analysis is performed on the whole time interval [-10 , 50] of the observations. Concerning the squatting to standing manoeuvre, the effect of ageing appears highly significant on the time course of both ΔMAP and ΔHR. On the contrary, the effect of ageing is not significant on the time course of ΔMAP in the sitting to standing manoeuvre. Nevertheless, a significative difference in the time course of ΔHR is observed during this manoeuvre.

DISCUSSION

Reliability of MAP and HR measurements

Measurement of arterial blood pressure by the photoplethysmographic technique of the Finapres device is an indirect technique. So, it is necessary to discuss the accuracy of the measurements. In the last years, numerous studies have compared Finapres results with direct intravascular blood pressure measurements in multiple physiological situations (Valsalva's manoeuvre, diving reflex, cold pressure test, isometric exercise). All concluded to the adequacy of Finapres measurements [11-13]. More important for this study, Imholz *et al* (1990) have observed a high correlation between intra-arterial blood pressure and Finapres measurements during postural stresses [14]. All these studies are answerable for our results reliability. From another point of view, it is also well known that eviction of arterial puncture eliminates most of psychological perturbations of circulatory parameters [15].

Nevertheless, at the light of the systolo-diastolic amplification affecting blood pressure waves in peripheral arteries [16], analysis were restricted to the mean arterial pressure, which is nearly constant along the biggest arteries.

In this study, HR is calculated from the arterial blood pressure signal. The so-called R-R electrocardiographic interval usually used to calculate HR is here replaced by the interval between two successive blood pressure maxima. This induces a loss of precision. Indeed, R wave peaks are temporally better defined than maximal blood pressure. This should be kept in mind if a baroreflex gain had to be calculated as did Goedhard et al during lying to standing manoeuvre [17].

Comparison with data from the literature

Most of data published in the literature are focused on the standing to squatting manoeuvre and have been published between 1950 and 1966. During this period, the physiopathological challenge was to explain the curious sign observed in many cyanotic congenital heart diseases.

The data published by Sharpey-Schafer in 1956 [3] and O'Donnel et al in 1962 [6] and obtained in control subjects show that standing to squatting induces an immediate rise of systolic, diastolic and mean arterial blood pressures that culminate after about 5 seconds and then regress to stabilize in most subjects at a level slightly higher than during standing. During squatting, these authors observed that pulse pressure was increased and also pointed out a persistent bradycardia. O'Donnel and McIlroy observed a very brief tachycardia immediately after standing, not discussed in their paper [6].

The squatting to standing manoeuvre induces opposite changes. MAP immediately falls below the initial standing values. According to Sharpey-Schafer [3], O'Donnel et al. [6] and Rossberg et al. [1], minimal values are respectively obtained after 5 to 6 seconds, 8 to 10 beats or 7.1 s. Then blood pressure starts to rise and stabilizes near the initial standing values in about 15 s [1,3]. Sometimes, an overshoot of MAP was observed [3]. On the other side, the heart rate immediately increases, monotonously or, with some subjects, biphasically, reaching its maximum value after 12 to 25 s [1,3,6,18].

All these observations are identical to our results.

However, our results differ from Rossberg and Penaz's observations on one point. According to these authors [1], mean standing hypotension is 57 mmHg versus 30 mmHg in this study. This discrepancy can result from an experimental protocol difference. In Rossberg's study, squatting posture was maintained during 5 minutes instead of 30 seconds in our study.

Determinism of circulatory changes

Initial hypertension in squatting was explained by a rise in cardiac filling pressure due to "squeezing blood out of the veins of the legs", leading to an increase in stroke output by Frank-Starling mechanism. For a minor part, it was also explained by "kinking" of the femoral arteries [3]. O'Donnel and Mc Ilroy [6] observed an increase in central blood volume and accepted the idea of a rise of cardiac filling. However, they did not observe consistent circulatory variations when postural changes were realized in a water tank. Therefore, they concluded that kinking of the arteries and veins of the legs could not be important. In the steady state phase in squatting posture, stabilization of MAP at a level not far from the standing one, was explained by peripheral vasodilatation measured either by plethysmography at the forearm [3] or by dye-dilution determination of cardiac output [6].

Mechanisms of changes observed during squatting to standing were not specifically investigated, but implicitely explained by the same adaptations developed in the opposite direction.

In this study, no complementary analysis was conducted. Nevertheless, we were attentive to the observation of Rossberg and Penaz [1] according to which in squatting to standing manoeuvre, blood pressure decreases during the first beat succeeding the postural manoeuvre. In all of our subjects this phenomenon was observed. Such a rapid variation cannot be easily explained by the previously invoked modifications of cardiac filling pressures. When Hoffman et al. [19] lifted dogs until they stood erect, the right ventricular stroke volume usually fell in the first beat after the postural change, but the left ventricular stroke volume did not fall for another 1-3 beats. When the dogs where rapidly lowered to standing on four legs again, a delay of 2-3 beats was also observed. So, according to us, another interpretation of immediate hypotension appears to be necessary. It should especially take into account the natural gravitational fluid mechanics phenomena imposed to the arterial blood. This is currently investigated in our laboratory [20].

Clinical utility of squatting to standing manoeuvre

In clinical practice, non-invasive investigations of cardiovascular responses to postural changes or Valsalva's manoeuvre are commonly used to study short term autonomic control of the circulation or, in other words, to assess integrity of arterial baroreflex and the so-called cardiopulmonary reflexes [21,22]. However, all these classical manoeuvres have intrinsic technical difficulties or methodological limitations (see discussion of the Valsalva's manoeuvre by Eckberg [23]). So appears the necessity of searching new technics: coughing manoeuvre for instance [24]. In this study we re-examine, using non invasive blood pressure measurement, the potential utility of an old postural test, today abandoned in clinical practice - except to help the cardiac murmurs diagnosis - : squatting to standing.

This manoeuvre, easy to perform for most subjects, is reproducible. It is obviously possible to improve intra-individual dispersion by repeating the procedure several times and retaining mean or median response like Borst et al. [25]. It needs no special device like tilt table or LBNP. Besides, our results confirm that the initial blood pressure peak induced by active lying to standing manoeuvre [22,25] is absent in squatting to standing, which suggests that muscular reflex participation is negligible.

Finally, amplitude of postural hypotension induced by the manoeuvre is rather large, approximatively twice the variations measured in sitting to standing. So, the ratio signal/noise is high. This fact probably explains why the still debated effects of ageing on the efficiency of short term blood pressure control (see Imholz et al. [26]) are clearly revealed by squatting to standing but not by the almost identical manoeuvre of sitting to standing.

According to our opinion, all these facts and arguments suggest that squatting to standing is the choice orthostatic stress for clinical practice as long as no muscular or skeletal anomaly prevents the manoeuvre.

Limitations of this study

For technical reasons, respiration was not recorded. So, we cannot be sure that all subjects avoided, as recommended, deep inspiration or short Valsalva's manoeuvre during changes of posture. Careful control of these respiratory perturbations could probably improve the signal-to-noise ratio of MAP changes [1].

Obviously, squatting in steady state phase is a very peculiar basal state to analyse circulatory modifications induced by quick standing. If we admit the classical opinion that postural arterial pressure variations are linked to repartition changes of the blood volume [27], to consider squatting as departure position lead to amplify the effect by increasing intrathoracic volume. On another hand, if squatting duration appears to be important to define the amplitude of hypotension after adoption of erect posture, time optimization of the manoeuvre will have to be achieved.

Conclusion

As suggested by Rossberg et al. [1], squatting to standing manoeuvre allows the investigation of the orthostatic regulation of arterial blood pressure. This manoeuvre appears to be reproducible and easy to perform using a non-invasive device. So, it is appropriate for a clinical use [28-30]. It is noteworthy that this postural manoeuvre, contrary to others, shows the effect of ageing.

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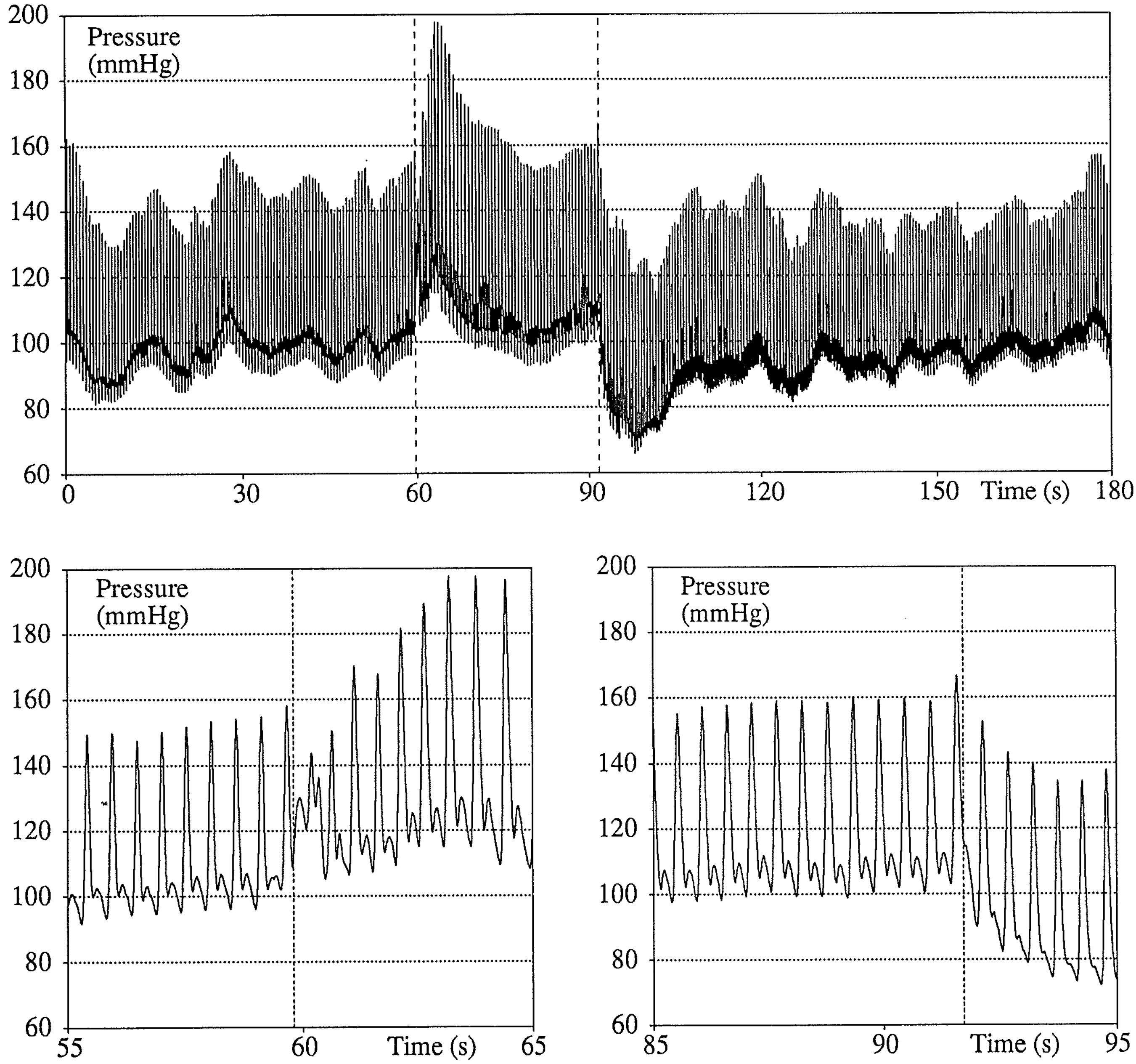
REFERENCES

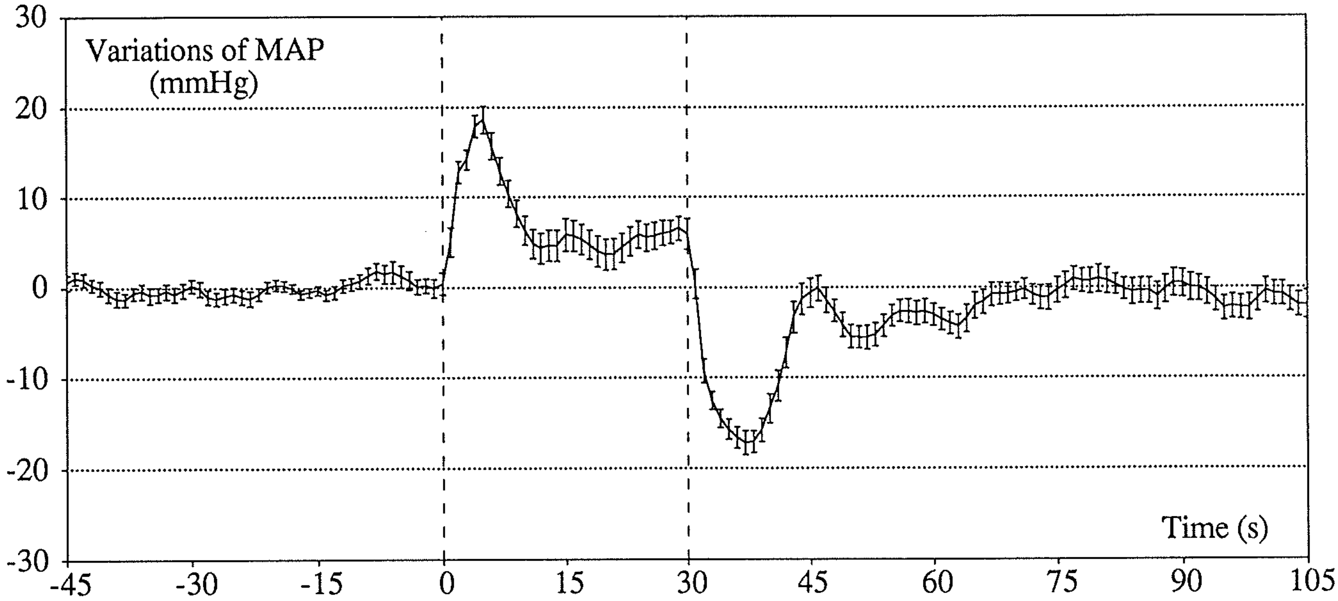
1. Rossberg F, Penaz J. Initial cardiovascular response on change of posture from squatting to standing. Eur J Appl Physiol 1988; **57:** 93-7.
2. Hunter W. Three cases of malformation of the heart. Medical Observations and Inquiries by a Society of Physicians in London 1784; **6:** 291. Cited by [6].
3. Sharpey-Schafer EP. Effects of squatting on the normal and failing circulation. Br Med J 1956; **i:** 1072-4.
4. Brotmacher L. Haemodynamic effects of squatting during repose. Brit Heart J 1957; **19:** 559-566.
5. Brotmacher L. Haemodynamic effects of squatting during recovery from exertion. Brit Heart J 1957; **19:** 567-73.
6. O'Donnel TV, McIlroy MB. The circulatory effects of squatting. Am Heart J 1962; **64:** 347-56.
7. Lembo NJ, Dell'Italia LJ, Crawford MH, O'Rourke RA. Bedside diagnosis of systolic murmurs. N Engl J Med 1988; **318:** 1572-8.
8. Penaz J. Photoelectric measurement of blood pressure, volume and flow in the finger. In: Digest of 10th International Conference on Biological Engineering, Dresden 1973: 104.
9. Zerbe GO. Randomization analysis of randomized blocks design extended to growth and response curves. Commun Statist: Theor Meth 1979; **A8(2):** 191-205.
10. Zerbe GO. Randomization analysis of the completely randomized design extended to growth and response curves. J Amer Statist Assoc 1979; **94:** 215-21.
11. Gradwell DP. Validation of a method of continuous monitoring of blood pressure during positive pressure breathing in man (Abstract). J Physiol (Lond.) 1991; **438:** 170P.
12. Imholz BPM, van Montfrans GA, Settels JJ, van der Hoeven GM, Karemaker JM, Wieling W. Continuous non-invasive blood pressure monitoring: reliability of Finapres device during the Valsalva manoeuvre. Cardiovasc Res 1988; **22:** 390-97.
13. Parati G, Casadei R, Gropelli A, Di Rienzo M, Mancia G. Comparison of finger and intra-arterial blood pressure monitoring at rest and during laboratory testing. Hypertension 1989; **13:** 647-55.
14. Imholz BPM, Settels JJ, van der Meiracker AH, Wesseling KH, Wieling W. Non-invasive continuous finger blood pressure measurement during orthostatic stress compared to intra-arterial pressure. Cardiovasc Res 1990; **24:** 214-21.
15. Stevens PM. Cardiovascular dynamics during orthostasis and the influence of intravascular instrumentation. Am J Cardiol 1966; **17:** 211-18.
16. Kroeker EJ, Wood EH. Comparison of simultaneously recorded central and peripheral arterial pressure pulses during rest,exercise and tilted position in man. Circ Res 1955; **3:** 623-32.
17. Goedhard WJA, Wesseling KH, Settels JJ. Baroreflex pressure control responding to orthostatic changes with age. In: Orlebeke JF, Mulder G, van Doornen LJP, eds. Psychophysiology of cardiovascular control. Model, methods and data. New York: Plenum Press, 1983: 191-202.

18. Guell A, Braak L, Bousquet J, Barrere M, Bes A. Orthostatic tolerance and exercise response before and after 7 days of simulated weightlessness. The Physiologist 1980; **23:** S151-52.
19. Hoffman JIE, Guz A, Charlier AA, Wilcken DEL. Stroke volume in conscious dogs; effect of respiration, posture, and vascular occlusion. J Appl Physiol 1965; **20:** 865-77.
20. Pochet TF, Pirotton MJ, Gerard PL. Isolation of the physiological regulation component of the arterial pressure time variation after postural stresses by a model of the gravitational and arterial kinking effects. The Physiologist 1993; **36(1)** suppl: 162-3
21. Hainsworth R. Non-invasive investigations of cardiovascular reflexes in humans. Clin Sci 1990; **78:** 437-43.
22. Wieling W. Standing, orthostatic stress, and autonomic function. In: Bannister R, ed. Autonomic failure. A textbook of clinical disorders of the autonomic nervous sytem. 2nd ed. New York: Oxford University Press, 1989: 308-20.
23. Eckberg DL. Parasympathetic cardiovascular control in human disease: a critical review of methods and results. Am J Physiol (Heart Circ Physiol) 1980; **239:** H581-93.
24. Van Lieshout EJ, Van Lieshout JJ, Ten Harkel ADJ, Wieling W. Cardiovascular response to coughing: its value in the assessment of autonomic nervous control. Clin Sci 1989; **77:** 305-10.
25. Borst C, Wieling W, van Brederode JFM, Hond A, de Rijk LG, Dunning AJ. Mechanisms of initial heart rate response to postural changes. Am J Physiol (Heart Circ Physiol) 1982; **243:** H676-81.
26. Imholz BPM, Dambrink JHA, Karemaker JM, Wieling W. Orthostatic circulatory control in the elderly evaluated by non-invasive continuous blood pressure measurement. Clin Sci 1990; **79:** 73-9.
27 Blomqvist CG, Stone HL. Cardiovascular adjustments to gravitational stress. In: Sheperd JT, Aboud FM, Geiger SR, eds. Handbook of Physiology. The cardiovascular system III. Bethesda, Maryland: American Physiological Society, 1983: 1025-63.
28. Di Maro G, Juchmès J, Fossion A, Scheen AJ, Lefèbvre PJ. Study of the baroreflex by beat-to-beat changes in finger arterial pressure during squatting in diabetic patients (Abstract). Diabetologia 1991; **34:** A158.
29. Scheen AJ, Juchmès J, Pochet Th, Marfella R, Dimaro G, Lefebvre PJ. Non invasive, beat-to-beat investigation of the effects of posture on arterial blood pressure in diabetic neuropathy. Diabete Metab 1990; **16:** 382-4.
30. Scheen A, Juchmès J, Di Maro G, Marfella R, Fossion A, Lefèbvre P. Hypertension artérielle importante en position accroupie chez le patient diabétique avec hypotension orthostatique : étude à l'aide d'un enregistrement continu cycle par cycle (Abstract). Arch Mal Coeur 1991; **84**: 36.



Pressure
(mmHg)
200
180
160
140
120
100
80
60
0
30
60
90
120
150
Time (s)
180
Pressure
(mmHg)
55
60
Time (s)
65
Pressure
(mmHg)
85
90
Time (s)
95

Fig. 1. **Typical recording of arterial blood pressure during a standing-squatting-standing manoeuvre.** A typical example of continuous recording of arterial blood pressure by the Finapres device during a standing-squatting-standing manoeuvre is presented first during the entire observation and secondly, at another temporal scale, around the postural changes. The vertical dotted lines are the postural changes marks manually introduced at the keyboard by the operator.


Variations of MAP
(mmHg)
30
20
10
0
-10
-20
-30
-45
-30
-15
0
15
30
45
60
75
90
105
Time (s)

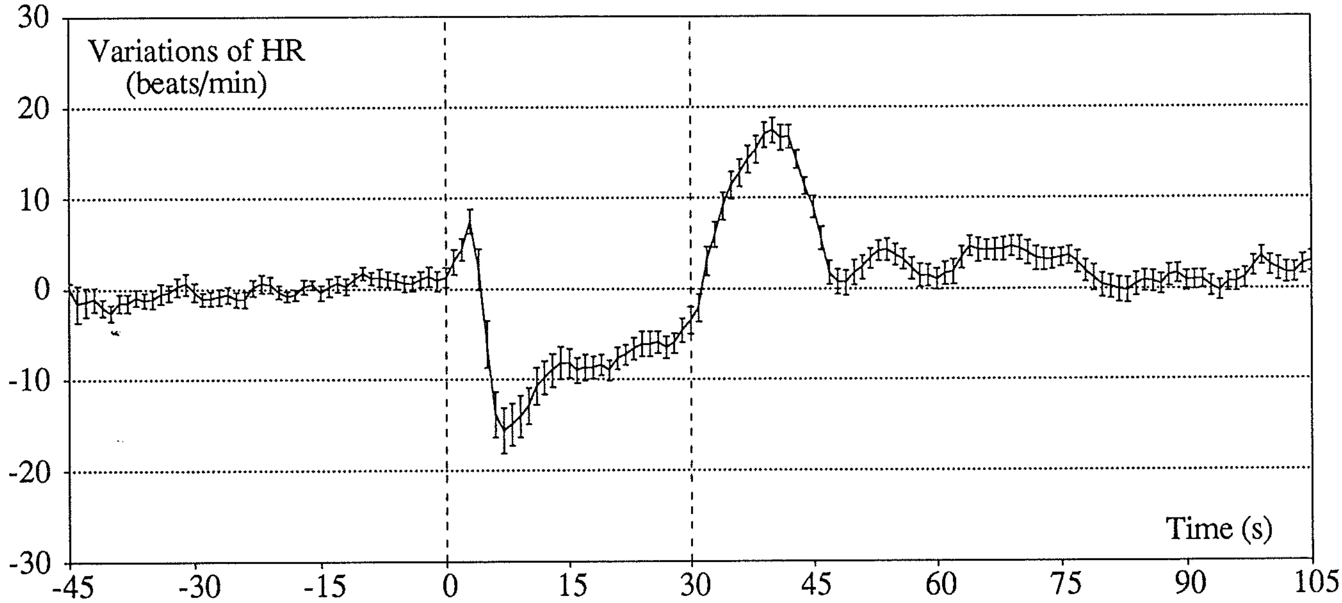
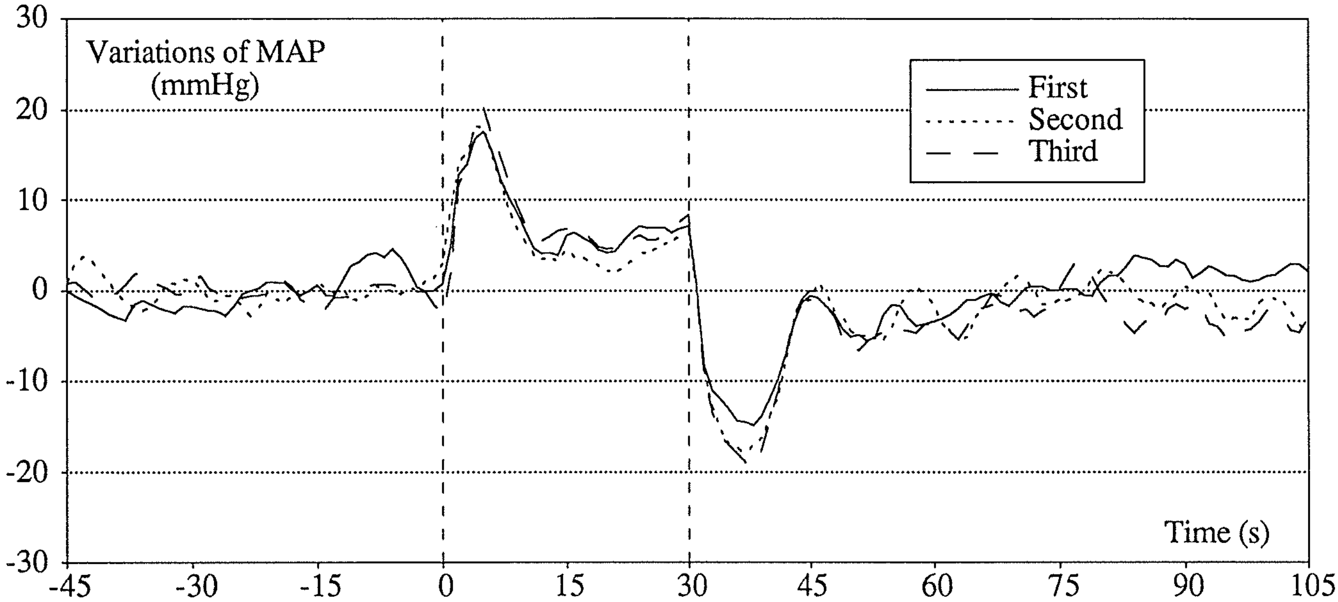

Variations of HR
(beats/min)
30
20
10
0
-10
-20
-30
-45
-30
-15
0
15
30
45
60
75
90
105
Time (s)

Fig. 2. Mean variations and SEM of beat-to-beat (interpolated at each second) MAP and HR during the standing-squatting-standing manoeuvre performed by the subjects of the first group. The departure levels used to compute the variations are the mean values on the period during which the subjects are in erect position at rest. The vertical lines show the temporal position of the postural changes.


Variations of MAP
(mmHg)
First
Second
Third
Time (s)
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-10
-20
-30
-45
-30
-15
0
15
30
45
60
75
90
105

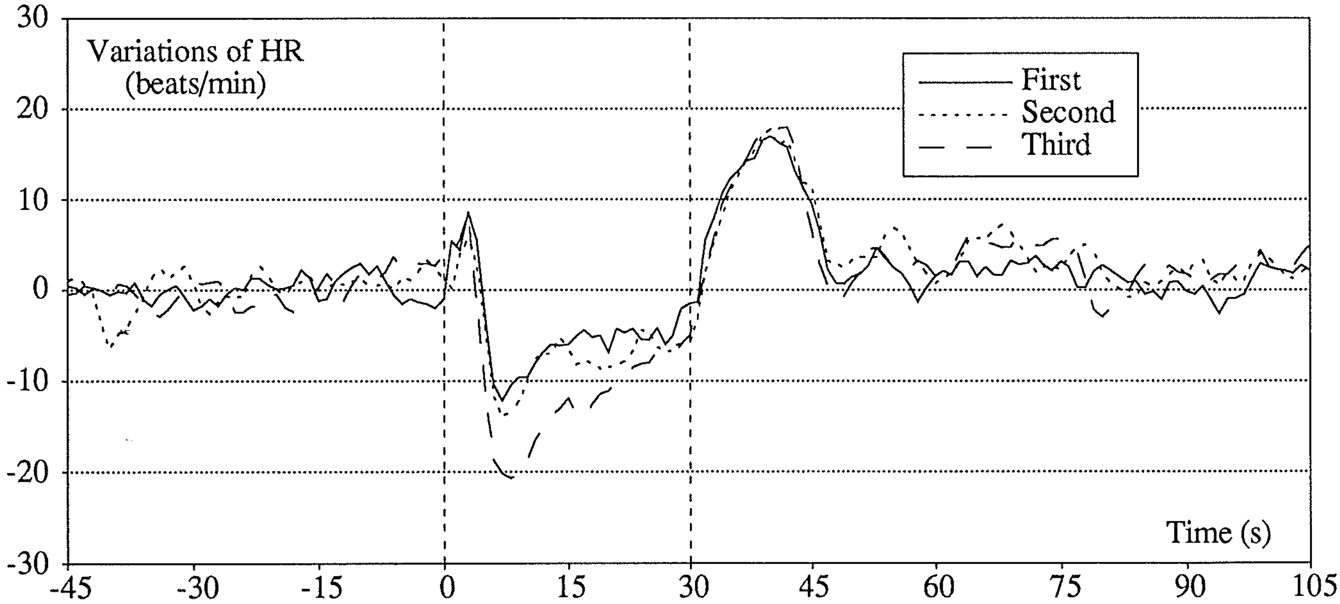
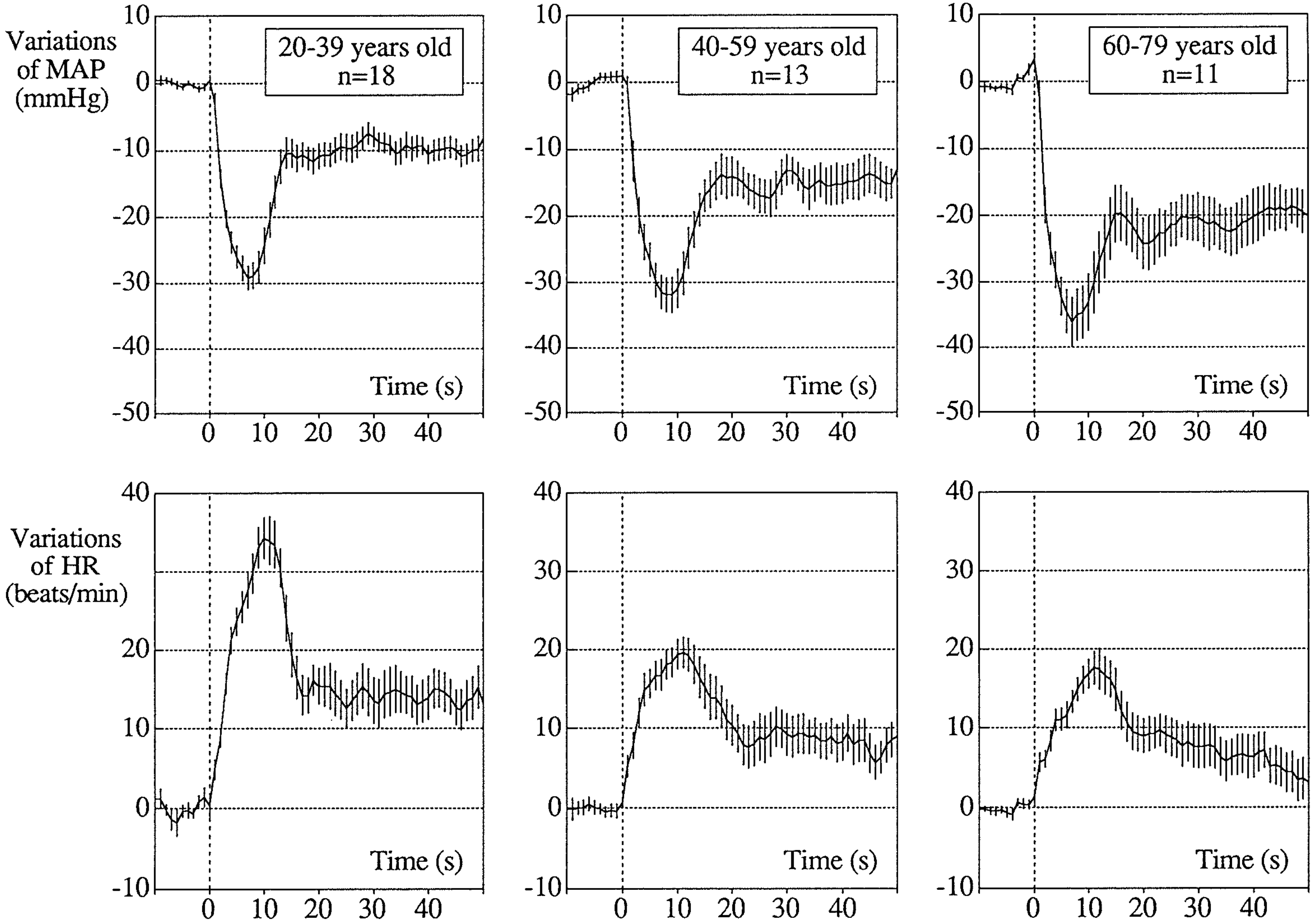

Variations of HR
(beats/min)
First
Second
Third
Time (s)
30
20
10
0
-10
-20
-30
-45
-30
-15
0
15
30
45
60
75
90
105

Fig. 3. Reproducibility results of the standing-squatting-standing manoeuvre. Mean variations of beat-to-beat (interpolated at each second) MAP (in the upper windows) and HR (in the lower windows) are compared for the first, second and third repetition of the sequence: standing-squatting-standing. The departure levels are the mean values before the postural change.



Variations of MAP (mmHg)
Variations of HR (beats/min)
20-39 years old n=18
40-59 years old n=13
60-79 years old n=11
Time (s)

Fig. 4. **Effect of ageing in the squatting to standing manoeuvre.** Mean variations and SEM of beat-to-beat (interpolated at each second) MAP (in the upper windows) and HR (in the lower windows) during the squatting to standing manoeuvre are compared for the three age-groups of the second group of subjects. The departure levels are the mean values before the postural change.

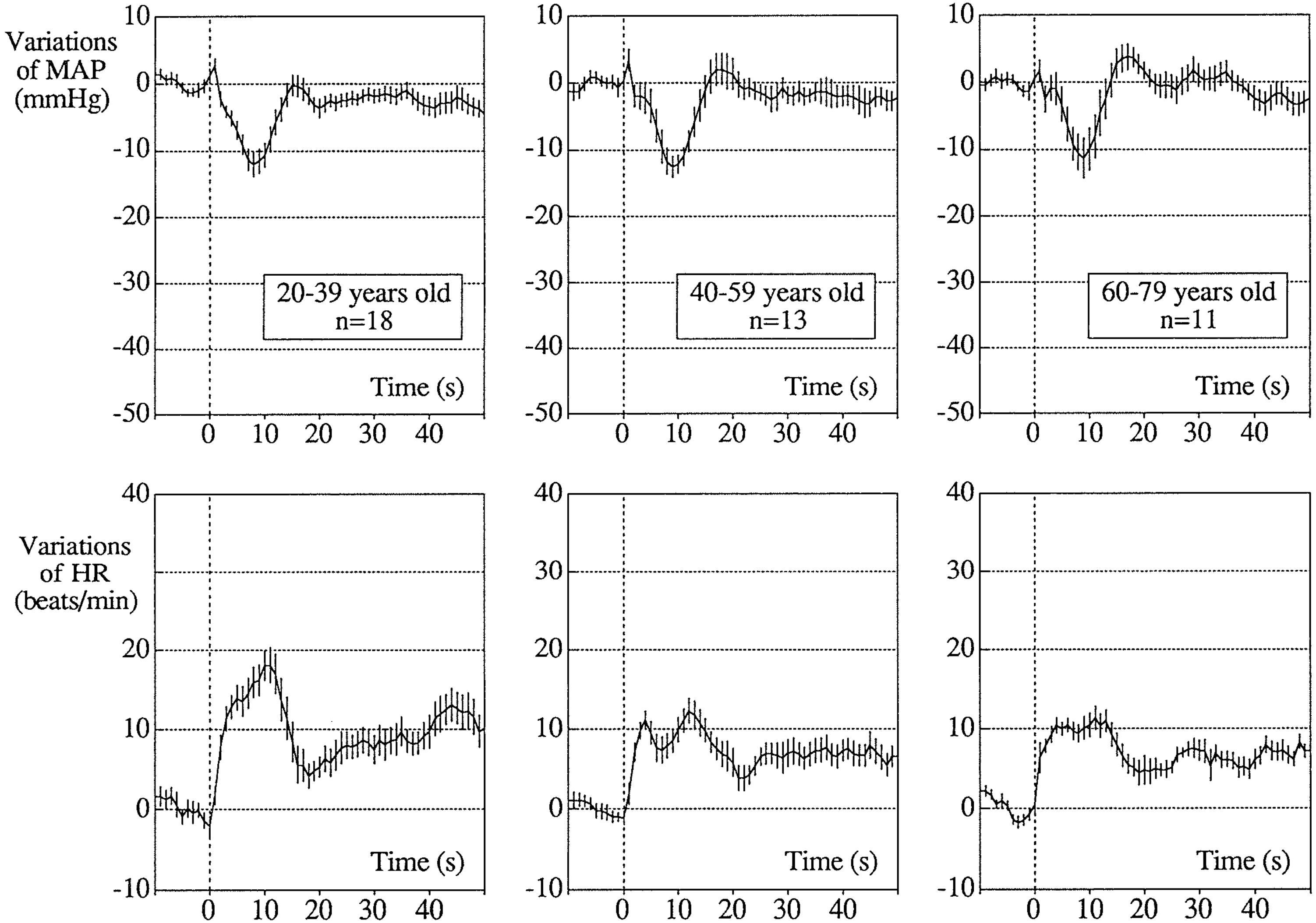

Variations of MAP (mmHg)
Variations of HR (beats/min)
20-39 years old n=18
40-59 years old n=13
60-79 years old n=11
Time (s)

Fig. 5. Effect of ageing in the sitting to standing manoeuvre. Mean variations and SEM of beat-to-beat (interpolated at each second) MAP (in the upper windows) and HR (in the lower windows) during the sitting to standing manoeuvre for the three age-groups of the second group of subjects. The departure levels are the mean values before the postural change.

	standing to squatting	squatting to standing
Peak of ΔMAP	F = 0.01 n = 2 m = 18 *P = 0.99*	F = 2.02 n = 2 m = 18 *P = 0.16*
Peak of ΔHR	F = 11.58 n = 2 m = 18 *P < 0.01*	F = 0.05 n = 2 m = 18 *P = 0.95*

F = F - statistic; n = d.f. of numerator; m = d.f. of denominator; *P* = *P*-value.

Table 1. Reproducibility of the peaks of ΔMAP and ΔHR during the standing-squatting-standing manoeuvre (two-way ANOVA). Low *P*-values (*P*<0.05) indicate poor reproducibility of the measurements during the three repetitions of the manoeuvre.

time interval [-10, 50] seconds	squatting to standing		sitting to standing	
	ΔMAP	ΔHR	ΔMAP	ΔHR
F	4.141	4.032	0.820	2.206
n	3.5	4.5	8.9	16.1
m	69.0	87.2	173.4	193.1
P	<0.01	<0.01	0.60	0.02

F = F - statistic; n = d.f. of numerator; m = d.f. of denominator; *P* = *P*-value.

Table 2. **Effect of ageing on the mean time course of ΔMAP and ΔH R during squatting to standing (sq-st) and sitting-standing (si-st) manoeuvres (Zerbe analysis).** Analysis is performed on the whole time interval [-10 s, 50 s] of the observations. Low *P*-values (*P*<0.05) indicate a significant effect of ageing.