STRESS AND THE CARDIOVASCULAR SYSTEM

by

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A thesis presented at
Victoria University of Wellington
in fulfilment of the thesis
requirements for the degree
of
Doctor of Philosophy
in
Psychology

1986

ACKNOWLEDGEMENTS

I would like record my sincere thanks to John Dutch for his help, encouragement and constructive comments throughout the course of the work on this thesis.

Deep personal thanks go to Ruth and Douglas Lake for their encouragement, proof reading and other practical support, and to Polly, Ruth, Prue and Mabel who together provided an incentive.

Finally, I would like to thank my subjects, many of whom spent long hours in the laboratory without reward.

LIST OF ABBREVIATIONS

ANS   Autonomic Nervous System

APP   Arterial Pressure Pulse

BP   Blood Pressure

BV   Blood Volume

c   pulse wave velocity (equations only)

Ca   Arterial Compliance

CAD   Coronary Artery Disease

CHD   Coronary Heart Disease

CF   Contractile Force

CO   Cardiac Output

CNS   Central Nervous System

DBP   Diastolic Blood Pressure

EKG   Electrocardiograph

EPI   Eysenck Personality Inventory

HP   Heart Period

HR   Heart Rate

Ia   Characteristic Impedance

IBI   Interbeat Interval

JAS   Jenkins Activity Survey

LVET   Left Ventricular Ejection Time

MATH   Mental Arithmetic

P   Pressure

p   blood density

PEP   Cardiac Pre-ejection Period

PERS   Personal Tempo

PROB   Problem Solving

PTT   Pulse Transit Time

PWV   Pulse Wave Velocity

RPI   R-wave to Pulse Interval

REAC   Reaction Time

SBP   Systolic Blood Pressure

SACL   Stress Arousal Checklist

STAI   State Trait Anxiety Inventory

TPR   Total Peripheral Resistance

V   Volume

ABSTRACT

The research reported in this thesis is primarily concerned with systemic arterial pulse transit time (PTT) which is of physiological significance because it is the most appropriate indicator of arterial compliance, the primary determinant of cardiac load, PTT acceleration or deceleration being associated with an increase or decrease in cardiac load respectively. Initially, PTT was investigated in the context of active/passive coping. Obrist, et al., (1978) proposed that active coping is generated by tasks of moderate difficulty and is characterized by large, sustained cardiac accelerations. Interbeat interval (IBI) was measured as the reference response for active/passive coping.

The assumption is that active coping is the behavioural state which provides the link between psychological stress and hypertension. However, contemporary cardiovascular physiologists put more emphasis on arterial compliance than IBI because it is not only significant in essential hypertension, but is also a critical determinant of circulation efficiency in health and disease.

In Experiment 1 men and women completed mental arithmetic, problem solving, reaction time and personal tempo (voluntary button pressing). Acceleratory and deceleratory PTT changes were found during all tasks; IBI changes were predominantly acceleratory and their magnitude was determined by task difficulty.

In Experiment 2 subjects completed problem solving tasks at two levels of difficulty. The results confirmed that unexplained directional variability characterized PTT changes and acceleration characterized IBI changes.

It was hypothesized that subject state could be a determinant of PTT response direction. In Experiment 3 the State-Trait Anxiety Inventory, the Stress Arousal Checklist, the Eysenck Personality Inventory and the Jenkins Activity Survey were administered before problem solving at three levels of difficulty and personal tempo were completed. PTT directional variability was not accounted for by any of the measures used. IBI changes were again acceleratory and their magnitude was determined by task difficulty.

Two extensions of the active/passive coping hypothesis were proposed: 1) that task difficulty and magnitude of IBI change are related along a continuum of behavioural coping; 2) that task type, not difficulty, determines the frequency of IBI acceleration. The effect of task contingency on IBI change was identified as requiring more detailed investigation.

It was further hypothesized that relative subject state as measured by a change in resting IBI (Malmo, 1959) could predict the direction of PTT change during task. In Experiment 4 subjects completed all tasks from Experiment 3 in both parts of an extended experimental session. Half the subjects ingested caffeine. IBI decelerated during baseline from part 1 to part 2 and deceleratory PTT changes dominated task responding in part 2. However, a caffeine-induced deceleration in IBI across baselines was not associated with deceleratory PTT change, and directional variability continued to characterize PTT changes.

In order to eliminate the directional variability of PTT changes during tasks a further experiment was undertaken which manipulated relative behavioural state prior to task by informing subjects on their first attendance at the laboratory that they would be required to perform a demanding problem solving task on their fifth attendance. From sessions 1 to 4 subjects attended the laboratory for short rest periods only during which cardiovascular activity was recorded. On the fifth session subjects also completed a task. In that experiment (Experiment 5), IBI acceleration over multiple session baselines in anticipation of a task was associated with uniformly acceleratory PTT changes during tasks, supporting the hypothesis that subject initial state is an important determinant of the direction of PTT change, and hence in whether cardiac load increases or decreases under stress.

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PREFACE

Over the past four decades, research on the relationship between cardiovascular and psychological variables has increased as technological advances have made physiological recording more precise and have also made the simultaneous recording of physiological and behavioural processes a reality (Greenfield & Sternbach, 1972; Hassett, 1978).

Early emphases were on obtaining a small sample of phasic (beat-by-beat) cardiovascular activity in the short period immediately before, during and after presentation of a stimulus in order that cardiovascular changes could be linked to stimulus events and interpreted in terms of the accompanying emotions (Ax, 1953), cognitive processes (Sokolov, 1963), psychological processes (Miller, 1978) or behavioural states (Obrist, 1976).

Over the past decade, emphasis has transferred to obtaining continuous recordings of tonic cardiovascular activity over longer periods (e.g. minutes or tens of beats) in order that cardiovascular changes can be linked with behavioural performance or psychological measures as a function of task parameters such as difficulty. These changes may then be interpreted in terms of physiological mechanisms of biological significance (Obrist, et al., 1974; Obrist, 1976, 1981). Tonic cardiovascular responses have been suggested to be of particular importance to the organism as they represent sustained physiological activity with relevance to the maintenance of cardiovascular homeostasis and biological survival.

Cardiac responses in particular have been suggested to index behavioural states of active or passive coping, and effortful active coping is hypothesized to be of particular physiological significance in that it may provide a link between psychological events and the etiology and development of essential hypertension (Obrist, 1976) as it is characterized by large magnitude, sustained acceleratory changes in cardiac responses which are likely to be mediated by sympathetic activity.

The purpose of the current work is to further the understanding of tonic cardiovascular changes during tasks from a biological perspective. Vascular dysfunction is implicated in over 50% of death and disease in the western world (O'Rourke, 1982), and focus was placed on arterial pulse transit time (PTT) as it is an index of arterial compliance. Interbeat interval (IBI) was recorded as the cardiac response, and systolic (SBP) and diastolic (DBP) blood pressures were recorded as additional measures of physiological significance. In addition, R-wave to pulse interval (RPI) was recorded as a non-invasive cardiac measure of interest (Pollack & Obrist, 1983), and those data are presented in Appendix 2.

Survival of the biological organism depends on the homeostatic functioning of the cardiovascular system. Sustained acceleratory or deceleratory changes in cardiovascular responses reduce the quality of life, and extreme acceleratory or deceleratory changes are likely to be life-threatening. To date, psychological research in tonic responding has focussed exclusively on acceleratory changes. Throughout the present work, the relative frequency and magnitude of acceleratory and deceleratory changes during tasks are described.

The work begins with a discussion on PWV and the other responses which are measured, followed by a review of the relevant literature.

The research comprises five experiments in all. The first 3 focus on changes in tonic measures of IBI and PTT during tasks. The last 2 describe an exploration into the direction, acceleratory or deceleratory, of PTT changes.

1 THE RESPONSES

1.1 DETERMINANTS OF PWV

The propagation of the pressure pulse wave from the heart, or pulse wave velocity (PWV), is an inverse function of arterial compliance: a faster velocity is associated with less compliance (Bramwell & Hill, 1922; McDonald, 1974; O'Rourke, 1982).

The pressure pulse is ejected from the left ventricle into the aortic arch. Each subsequent segment of artery stretches to accommodate the pulse as it is propelled forward by the elastic recoil of the preceding segment, generating the pressure pulse wave. The blood vessel responses reflect both the amount of change in blood volume and the rate at which the volume changes. Thus the distension of the vasculature in response to changes in volume flow reflects viscoelastic properties, and these increase as a direct function of the amount of smooth muscle in the vessel wall (Rushmer, 1976). Arterial compliance refers to the viscoelastic properties of the arteries which determine the blood vessel volume (or radius) associated with a particular transmural pressure, and both the change, and the rate of change, in the volume (or radius) which accompanies a change in pressure (Berne & Levey, 1977; Cox, 1979; O'Rourke, 1982).

Momentary arterial compliance (C_a) can be expressed as the ratio of changes in mean arterial volume (V_a) and mean arterial pressure (P_a)

$$C_a = dV_a / dP_a \qquad (1)$$

Characteristic impedance (I_a) is the load against which the heart must work (O'Rourke, 1982; Avolio, et al., 1983). It extends the concept of peripheral resistance by describing impedance from all vessels distal to the site of measurement (Cox, 1979). It is defined as the ratio of pressure and volume, and is therefore the inverse of arterial compliance,

$$I_a = P / V = 1/C_a \qquad (2)$$

By corollary, PWV is a direct function of characteristic impedance with a faster velocity associated with greater impedance (O'Rourke, 1982; McDonald, 1974). Characteristic impedance can also be expressed as the product of PWV and blood density (Avolio, et al., 1983; O'Rourke, 1982) and as blood density is approximately 1.05 g cm^{-3} PWV and characteristic impedance are numerically similar allowing changes in cardiac load to be quantified (Avolio, et al., 1983; O'Rourke, 1982).

1.11 PWV as a Function of Pressure and Volume

Like perturbations in any physical system, PWV can be shown to be a function of the elasticity and density of the medium through which it travels. McDonald (1974), Laird (1980) and O'Rourke (1982) all record Thomas Young as the first to calculate PWV in accordance with Newton's law of sound travel through air: velocity is equal to the ratio of elasticity over density.

The standard form of Young's equation expresses PWV as a function of volume and transmural pressure as follows (O'Rourke, 1982):

$$c = / dP/(dV/V)p \qquad (3)$$

where c is PWV, and dP is the change in arterial pressure required to increase the arterial volume, V, by the amount dV, and p is blood density.

The equation states that PWV is the square root of the ratio of the pressure increase over the product of the associated proportional volume increase and blood density. Thus PWV is directly related to both arterial pressure and arterial volume. The expression dP.V/dV is Young's coefficient of volume elasticity, and therefore PWV increases directly with arterial elasticity or rigidity, and inversely with arterial compliance or distensibility.

1.12 PWV as a Function of Vessel Properties

The Moens-Koetwig equation was derived from studies which related PWV to the properties of a thin elastic tube (Bramwell & Hill, 1922) and thus described PWV as a function of blood density and the physical properties of the arterial vessels, specifically the internal diameter, the elasticity and the thickness of the vessel wall.

$$c = / \ Eh/2rp \qquad\qquad (4)$$

Again, c is PWV and p is blood density. E is Young's elastic modulus of the vessel wall, h is wall thickness and 2r is vessel internal diameter.

The equation states that PWV is the square root of the ratio of two products, vessel wall elasticity and thickness over vessel internal diameter and blood density. Thus PWV is directly related to vessel rigidity and inversely related to vessel size. Both these properties vary from one vascular bed to another, and can also vary from moment to moment within a single vascular bed as a function of local sympathetic neural and humoral activity (Cox, 1979).

Notably, in addition to quantifying PWV, the equivalent equations (3) and (4) also serve to emphasize the interdependence of volume, pressure and vessel characteristics both over time and over arterial segments.

1.13 Noninvasive Measures of PWV

PWV cannot be determined non-invasively using either of the two equations (3) or (4) given above as in each case the physical measurements could only be estimated. *In vivo* studies therefore usually record actual arterial pulse transit time (PTT) as the difference in pressure pulse arrival time at two distinct arterial sites, one more peripheral than the other (Bramwell & Hill, 1922; Dutch & Redman, 1983). It is then possible to transform the recorded PTT into measures of PWV if the length (L_a) of the arterial segment over which recording took place can be calculated. By definition,

$$PWV = L_a / PTT \qquad (6)$$

As values of PWV obtained noninvasively are thus carefully derived estmates of actual PWV (Avolio, et al., 1983; Geddes, et al., 1981) it is not uncommon for the transformation to be omitted (Lane, et al., 1983; Dutch & Redman, 1983; Marie, et al., 1984; Redman & Dutch, 1984).

It should be noted that, because PTT is a measure of time, and PWV is a measure of speed, they are inversely related. Therefore, as PWV is directly related to both arterial pressure and volume, PTT is inversely related to those two measures.

1.2 ARTERIAL COMPLIANCE

Baseline, or resting, levels of arterial compliance are determined by mechanical properties such as that defined by Young's modulus of elasticity (McDonald, 1974), and geometrical properties particular to each blood vessel segment, both of which are partly determined by the physical constitution of the arterial tree, but both of which can also vary as a function of controlling variables such as sympathetic tone and changes in sympathetic activity (Cox, 1979).

1.21 Structural and Geometrical Properties of the Arteries.

Elastin and collagen are the structural proteins which comprise the connective tissues in the arterial walls. Arteries closer to the heart contain greater amounts of elastin than collagen and therefore have more nearly pure elastic qualities than arteries more distal to the heart which have an increased amount of collagen. Smooth muscle cells are probably the only cells in the media of normal adult blood vessels, although those arteries close to the heart are very poorly muscled (Cox, 1979). At rest, blood vessels have a basal tone determined primarily by sympathetic neural activity which reflects the tonic partial contraction or tension of the smooth muscle in the arterial wall. Basal tone can be reduced through vasodilation or increased through vasoconstriction (Cox, 1979; O'Rourke, 1982) and changes in smooth muscle tone alter characteristic impedance and therefore also alter aortic PWV (Stone & Dujardin, 1984).

As blood vessels stretch, they increase their resistance to further distension, resulting in a nonlinear relation between volume and pressure (or

length and tension of the arterial wall) which is particularly marked over the physiological range (75mmHg-150mmHg) (Berne & Levey, 1977).

1.22 Compliance in Different Vascular Beds.

There is a general increase in elastic modulus (stiffness) as a function of the distance from the heart and reduced size of vessel luminal diameter. The increased rigidity in smaller, more peripheral arteries is an important feature in the amplification of the pressure pulse as it approaches the peripheral vasculature and the subsequent dispersion of the blood through the capillary beds (Rushmer, 1976; Berne & Levey, 1977), as well as in generating increased wave reflection back toward the heart from the periphery (O'Rourke, 1982).

PWV increases as the pulse travels toward the periphery (O'Rourke, 1982). Laszt & Muller (1952) reported PWV of 400-500 cm/sec in the thoracic artery which increased to 850-130 cm/sec in the femoral artery. Avolio, et al., (1983) reported faster leg and arm PWV than aortic PWV particularly for people under 50. Learoyd and Taylor (1966) reported PWV of 5-6 m/sec in the thoracic and abdominal arteries, 8-9 m/sec in the iliac artery and 20 m/sec in the femoral artery for subjects aged less than 35 years, compared to PWV of 7-9 m/sec in the thoracic, abdominal and iliac arteries, and 12-14 m/sec in the femoral arteries in subjects older than 35 years.

1.23 Compliance and Age.

In the process of growth and natural development, vessel walls become stiffer as the total amount of connective tissue increases and also becomes more concentrated because the extracellular water content decreases. In addition, the ratio of collagen to elastin increases, but the amount of smooth

muscle decreases. Thus the larger arteries (and veins) become more rigid as a function of age, but the peripheral vessels become more flaccid due to the reduction in smooth muscle (Berne & Levey, 1977; Cox, 1979; Learoyd & Taylor, 1966) and the vasculature is less responsive to stimulation, including exercise (Yin, Weisford & Milnor, 1981).

There is a large body of literature which shows that PWV increases with age (McDonald, 1974; O'Rourke, 1982). Bramwell & Hill (1922) were the first to make a detailed report on changes in PWV associated with age. They measured PWV between the proximal aorta and radial artery and found an increase from 5.2 m/sec at age 5 years, to 8.6 m/sec at age 84 years.

Subsequent studies have demonstrated age-related increases in many populations (Avolio, et al., 1983; Hallock, 1934; Bramwell, Hill & McSwiney, 1923; Gozna, Marble, Shaw & Holland, 1974).

In an epidemiological study conducted on Chinese in the northern region of China, Avolio, et al., (1983) reported that, when pressure levels were held constant, PWV increased from 5.99 m/sec in the first decade of life to 11.29 m/sec in the ninth decade.

Gozna, et al., (1974) found that both the elastic modulus, or stiffness index, and PWV in the ascending thoracic artery and right pulmonary artery increased linearly with age, approximately doubling over 4 decades, particularly in the systemic vasculature. PWV in the thoracic artery was recorded as 2-4 m/sec at age 10 years, and 7-8 m/sec at age 50 years; that of the right pulmonary artery was recorded at 1-2 m/sec at age 10, and 3-4 m/sec at age 40 years.

1.24 Compliance and Disease.

Vascular dysfunction has been implicated in 50% of western morbidity (O'Rourke, 1982), and premature degeneration of the arteries is a characteristic of some disease states (Sands, 1925; Rushmer, 1976; Guyton, 1981).

PWV increases in some disease states including hypertension (Avolio, et al., 1983; O'Rourke, 1976). It has also been shown to increase with increased atherosclerosis (Malinow, 1980) and decrease with the regression of atherosclerosis (Farrar, et al., 1980). Similarly, Simonson & Nakagawa (1960) demonstrated that faster PWV is associated with coronary disease, and they suggest that acceleratory change in PWV may precede cardiac insufficiency. They proposed the use of PWV measures for large scale clinical and epidemiological exploration for early detection of atherosclerosis and vascular degeneration. It has since been shown that PWV is effective in early detection work with diabetic individuals and those thought to be at risk (Scarpello, Martin & Ward, 1960; Woolam, et al., 1962).

1.25 Transient Changes in Compliance and PWV.

Smooth muscles are connected to passive connective tissues which mediate vascular reactivity and are the active elements of the blood vessel. They are instrumental in controlling arterial blood pressure and distributing cardiac output. When the muscle is activated and contracts, there is a reduction in the luminal diameter of the vessel, or vasoconstriction, with an associated increase in PWV (Jones, 1983). A vasodilation response increases the luminal diameter of the arterial vessel and is associated with a decrease in PWV (Jones, 1983; O'Rourke, 1982).

The contractile proteins in smooth muscle are actin and myosin, and like striate muscle, smooth muscle contracts with maximal force at an optimal length, and with maximum shortening at an optimum tension. Thus, the contractile response to a common stimulant may differ for smooth muscles at different sites, and even for the muscles at a common site on different occasions (Cook, 1974; Cox, 1979). In general, contractile response magnitude increases as the calibre of the vasculature decreases, and maximal smooth muscle contraction can produce total closure of the lumen in vessels as large as the secondary branches of the aorta, as well as in other medium and small arteries and arterioles, particularly when internal pressure is low.

Both the amount and the rate of vasoconstriction produced by smooth muscle contraction is influenced by the initial transmural pressure. In the total vasculature the pressure dependent nature of smooth muscle reactivity means that when systemic BP decreases, maximal vasoconstriction occurs in the area of the larger arteries, but when systemic BP increases, maximal response occurs in the smaller arteries (Cox, 1979).

Smooth muscle is both neurally and humorally reactive. In general, sympathetic neural stimulation increases smooth muscle contraction which reduces the luminal diameter of arteries and arterioles. Catecholamines are also discharged during sympthetic activation and function as an extension of the neural impulse. They include norepinephrine, epinephrine, angiotensin and vasopressin, all of which generally induce vasoconstriction responses (Rushmer, 1976; Berne & Levey, 1977). However, there are at least two types of sympathetic receptors in the cardiovascular system, alpha and beta, with possible sub-types within those two kinds (Guyton, 1981). Epinephrine evokes

vasoconstriction from one type, the alpha receptors, but may evoke either vasoconstriction or vasodilation from the other, beta receptors, depending on the vascular bed (Cox, 1979; Berne & Levey, 1977). As far as is currently known, alpha receptors are found exclusively in the vasculature and mediate a vasoconstriction response. Beta receptors occur in both vascular sites where they can evoke vasodilation, and in cardiac sites where they can increase cardiac rate and force. It is possible that the beta receptors in the heart which are known to be reactive to both norepinephrine and epinephrine are distinct from those in the vasculature which have been so far documented to be sensitive to epinephrine only.

The amount of smooth muscle in the arterial wall increases as vessels become more distal to the heart. Small arteries and arterioles are described as muscular vessels. They are also richly innervated with sympathetic neural fibres and are neuro-reactive vessels. In contrast, the large elastic arteries proximal to the heart are poorly supplied with smooth muscle and innervation. Their response to neural stimulation is relatively weak and slow, and they are more responsive to sympathetically induced humoral control exerted by circulating catecholamines (Jones, 1983).

The documentation of controlling influences on the vasculature is increasing. It was only relatively recently reported that circulation processes in several vascular beds are also responsive to parasympathetic vasodilatory mechanisms, and it is likely that more such processes may be identified. However, parasympathetic influences on the vasculature tend to be more localized and transient than those of the sympathetic system (Jones, 1983). In addition, vascular smooth muscle contractility can also be

influenced by circulating electrolytes, particularly cations, as well as other chemical factors such as blood gases. Smooth muscle contractility is also reactive to physical and mechanical stimulation such as stretch or external touch, and to local factors such as temperature (Berne & Levey, 1977). But these various influences are recognized as being less important and more localized than sympathetic influences.

Thus, transient vasoconstriction and vasodilation responses, associated with transient accelerations and decelerations in PWV respectively, are generally considered to be primarily determined by changes in sympathetic activity on the vasculature (Cox, 1979; Jones, 1983; O'Rourke, 1982). In particular, during psychological research, PTT recorded over peripheral muscular arterial segments has been found to accelerate in response to psychological stressors, suggesting a sympathetically mediated vasoconstriction response in the peripheral vasculature associated with an increase in peripheral resistance (Dutch & Redman, 1983; Redman & Dutch, 1984).

1.3 PWV FROM A BIOLOGICAL PERSPECTIVE

In the preceding sections, PWV was shown to index arterial compliance and, by corollary, its inverse, vascular impedance. Changes in sympathetic activity on the vasculature were shown to be the most influential determinant of transient changes in vessel luminal diameter, and hence of transient changes in PWV.

In the current section some aspects of cardiovascular functioning mediated by arterial compliance, and thus indexed by PWV, are outlined, and the concept of characteristic impedance as a measure of cardiac load and its non-invasive quantification by PWV are discussed.

1.31 Mechanorecptors

Control of mechanoreceptor activity is the primary mechanism through which arterial compliance influences cardiovascular functioning. Mechanoreceptors can also be termed stretch-receptors, baroreceptors or pressoreceptors. Previous work has attributed the neural activity mediated by mechanoreceptor activation to an increase in transmural pressure. However, it is now established that it is the change in luminal diameter, or distension of the arterial wall, which is critical (Rushmer, 1976; Cox, 1979; Bergel, 1979).

Mechanoreceptors are sited in the elastic segments of arterial walls at major branches. As the arterial site housing the mechanoreceptor distends to accommodate each pressure pulse, the mechanoreceptor fires bursts of impulses which travel to cardiovascular regulatory centres in the central nervous system. Firing is reduced or ceases during the subsequent elastic recoil which propels the pulse forward to the next segment.

Current evidence shows that impulses from particular sites have specific effects. Overall, changes in luminal diameter are known to mediate central control processes which influence arterial volume, peripheral resistance and cardiac function (Rushmer, 1976; Berne & Levey, 1977; Bergel, 1979). Distension of the mechanoreceptors activates impulses which travel to the cardio-regulatory centre in the medulla and slow the heart by stimulating the motor nucleus of the vagus and inhibiting the cardio-acceleratory centre. Similarly, the contractility of the ventricles and the atria are increased and the vasoconstriction centre inhibited, thereby increasing stroke volume and reducing peripheral resistance.

Mechanoreceptors have also been identified in the ventricular and atrial walls of the myocardium, and in major cerebral vessels, and there is little doubt that there are many, even in the arterial tree, which have not yet been located. Mechanoreceptors have connections to and from other sympathetic and parasympathetic centres in addition to the ones cited above, but the specific regulatory functions of most are not yet known (Rushmer, 1976).

1.32 Mechanoreceptors and Heart Rate

Activity of the mechanoreceptors in both carotid sinus and the aortic arch is a major determinant of heart rate (HR) changes. In general, increased firing of these receptors, reflecting increased distension and corresponding with increased pressure, results in cardiac deceleration through stimulation of the vagus nerve and inhibition of the cardio-acceleratory centre, while decreased firing engenders cardiac acceleration through the reverse process (Berne & Levey, 1977).

1.33 Mechanoreceptors, Heart Rate and Blood Pressure

Mechanoreceptor activity underlies the inverse relation between HR and blood pressure (BP) termed Marey's Law of the Heart (Berne & Levey, 1977; Cox, 1979).

The predominant interpretation of Marey's Law is that reciprocal activity of both branches of the autonomic nervous system participates in homeostatic adjustments of HR associated with moderate changes in BP (up to 20mmHg-30mmHg). Subsequent changes in BP reflect activity of one branch only. Thus an increase of up to 30mmHg in arterial pressure is likely to be associated with

bradycardia caused by a progressive withdrawal of sympathetic activity accompanied by an increase in vagal stimulation. Larger magnitude increases would be associated with further bradycardia which could be attributed entirely to an increase in vagal activity in the absence of sympathetic tone. Analogous effects ensure that decreases in BP are associated with tachycardia, although the functional absence of vagal tone may occur following a decrease of 20mmHg (Berne & Levey, 1977).

That interpretation can be readily accounted for by the underlying mechanism. In young, healthy arterial systems, an approximately linear relationship obtains between increases in blood volume and pressure over a wide range of values (Berne & Levey, 1977). Thus increases in pressure are associated with increases in volume and mechanoreceptor activation through arterial wall distension, facilitating sympathetic withdrawal and vagal stimulation. Further increases in volume and arterial rigidity associated with further increases in pressure enhance that response, while decreases in volume and rigidity associated with decreases in pressure and mechanoreceptor activity reverse it through arterial wall contraction facilitating sympathetic activation and vagal withdrawal.

1.4 COMPLIANCE AND ARTERIAL PRESSURE

As shown in section 1.11, arterial compliance, arterial blood volume and arterial blood pressure are interdependent facets of the circulatory processes. Briefly, the more compliant the arterial walls, the smaller the increment in blood pressure for any increase in blood volume and the smaller the reduction in blood pressure for any decrease in blood volume. Thus pressure is

stabilized as a function of arterial compliance, with a consequent minimizing of left ventricular hydraulic workload (see section 1.5)

1.41 Compliance and Pulse Pressure.

Arterial pulse pressure (APP) is defined as the arithmetic difference between systolic (SBP) and diastolic (DBP) blood pressure,

$$APP = SBP - DBP \qquad\qquad (7)$$

It corresponds to the change in volume during one cardiac cycle, and varies inversely with arterial compliance. Larger pulse pressures are associated with more rigid arteries, partly due to increased wave reflection (O'Rourke, 1982), and larger pulse pressures are also indicative of increased left ventricular hydraulic workload, as the pressure load is spread unevenly across the cardiac cycle (see section 1.5).

1.43 Implications of Reduced Compliance

As indicated in section 1.2, compliance in any vascular bed can be reduced as a result of increasing age, some disease states and also as a result of increased sympathetic influences on the vasculature.

As compliance decreases, less arterial distension and elastic recoil occurs as a function of volume changes and thus the mechanoreceptors receive less stimulation. The likely consequence is a potentiation of sympathetic influences and a minimizing of parasympathetic influences. That consequence may have some implications for increasing the understanding of the relatively high levels of sympathetic influence on cardiac responses which have been postulated to be of significance in the etiology and development of hypertension (Julius & Esler, 1975; Obrist, 1981; O'Rourke, 1982).

In addition, as compliance decreases, it is constant over a narrower range of pressures, and a non-linear relationship is found between increases in volume and pressure. Thus, a smaller increase in volume becomes associated with a larger increment in pressure, particularly at pressures of 80mmHg and above (Berne & Levey, 1977; Cox, 1979).

Further, as compliance decreases, larger pressure pulses occur indicating greater fluctuation between pressure limits in the cardiac cycle and increased cardiac load (see section 1.5). However, if volume increment remains constant, but peripheral resistance is increased, decreased compliance results in a larger magnitude increase in pressure upper limit (SBP) and a smaller magnitude increase in pressure lower limit (DBP). Persistently elevated total peripheral resistance (TPR) is a characteristic of chronic hypertension, and the described BP responses which occur as a direct function of the rigidity of the arterial wall closely resemble those of the hypertensive patient. Thus decreased arterial compliance appears likely to have an important mediating role in hypertensive symptoms (Berne & Levey, 1977).

1.5 HYDRAULIC FILTERING AND COMPLIANCE

The arterial system constitutes a hydraulic filter with the myocardium functioning as a pressure pump (Berne & Levey, 1977). Hydraulic filtering makes two important contributions to arterial function. Firstly, it allows the intermittent ejections of blood from the heart into the aorta to be converted into a steady flow at the capillaries. Secondly, it reduces the workload on the heart by maintaining a relatively stable pressure level over the entire cardiac cycle (Berne & Levey, 1977).

The large arteries and their main branches function as elastic conduits, distributing blood to high resistance terminals (O'Rourke, 1982; Berne & Levey, 1977). The principal sites of peripheral resistance are the small arteries and the larger arterioles, while smaller vessels function to regulate blood flow in accord with local metabolic requirements (Abboud, 1972; Cox, 1979). As blood flow through the vasculature is controlled primarily by changes in vessel luminal diameter, it is important that there is an adequate pressure head in the arterial system. Within optimal physiological limits (O'Rourke, 1982), the more distensible or compliant the arteries are, the more effective hydraulic filtering is and the more consistent the pressure level remains.

1.6 CHARACTERISTIC IMPEDANCE

Arterial compliance represents the positive component of cardiac work. The negative component, or the load against which the heart must work, can be expressed in terms of peripheral resistance or as vascular impedance which represents the physical properties of all vessels distal to the site of measurement, thereby extending the concept of peripheral resistance (Cox, 1979).

Vascular impedance varies considerably in different beds, and is primarily a function of the mechanical and geometrical properties of the vessels. To the extent that peripheral vessels distal to the measurement site are reactive, vasoconstriction increases vascular impedance, and vasodilation decreases vascular impedance at any site. Larger changes occur with respect to vasodilation (O'Rourke, 1982).

The effects of vasoconstriction and vasodilation on vascular impedance are most satisfactorily explained in terms of changes in pulse wave reflection from peripheral sites (O'Rourke, 1982; Cox, 1979), reflection rate being a direct function of pulse propagation rate or PWV. Changes in impedance during vasoconstriction and vasodilation can be attributed to changes in PWV (see section 1.4) and therefore in wave reflection rate. The small changes in impedance observed during vasoconstriction reflect a high basal sympathetic (constrictive) tone on the small arteries and arterioles, while the larger changes during vasodilation suggest that peripheral sites are the major source of wave reflection (O'Rourke, 1982). The influence of wave reflection is hypothesized to cause vascular impedance to oscillate around a true mean impedance which is designated as characteristic impedance.

Thus characteristic impedance is a theoretical concept of true mean impedance or load against which the heart must work. It does not represent actual vascular impedance at any site since those values fluctuate as peripheral wave reflection changes, but provides a value of tonic mean load (Cox, 1979; O'Rourke, 1982). As noted in section 1.1, characteristic impedance is the product of blood density and aortic PWV. Therefore, changes in cardiac load can be quantified when systemic PWV is recorded (Avolio, et al., 1983; O'Rourke, 1982).

1.7 DETERMINANTS OF HEART RATE OR INTERBEAT INTERVAL

Tonic levels of HR are determined by the frequency with which spontaneous changes in membrane potential occur in the sino-atrial (SA) node (Berne & Levey, 1977). Sympathetic and vagal nerves both terminate in the region of the

SA node, and hence both branches of the autonomic nervous system influence HR. However, in normal, healthy individuals, vagal influences dominate resting HR (Rushmer, 1976).

1.71 Transient Changes in HR

Changes in HR usually involve alterations in the relative contribution by both branches of the autonomic nervous system (Berne & Levey, 1977). The branches can act synergistically, one activating and the other withdrawing, or antagonistically, both activating or withdrawing, to control HR (Randall, 1965, 1976). In addition to direct impulse firing, catecholamines are also discharged by nerves from both branches. The sympathetic discharge is norepinephrine which accelerates membrane depolarization by the SA node cells, resulting in tachycardia; the vagal discharge is acetylcholine which retards membrane polarization by SA node cells, resulting in bradycardia. Once activated, sympathetic influences diminish gradually, but vagal control can terminate abruptly. Hence an acceleratory change in interbeat interval (IBI) may be mediated by withdrawal of vagal influence, or an increase in sympathetic influence or both of these. However, it could also be the result of a withdrawal of vagal influence over-riding a withdrawal of sympathetic influence, or an increase in sympathetic influence over-riding an increase in vagal influence (Berne & Levey, 1977).

Changes in HR can occur to afferent responses from tissues and organs in virtually every part of the body. In general, visceral afferent nerves produce an effect of bradycardia, while somatic pain from the skin produces tachycardia (Rushmer, 1976). Phases in the normal respiratory cycle are also associated

with phasic changes in HR which can be enhanced by vagal tone and are mediated by central processes (Berne & Levey, 1977). Further, somatic muscle activity has been shown to be associated with parallel changes in HR (Obrist, et al., 1970), and stimulus rejection or intake have been postulated to be associated with tachycardia and bradycardia respectively (Lacey, 1967).

1.72 Heart Rate From a Biological Perspective

Heart Rate derives its physiological significance mainly from its contribution to cardiac output, which is the product of stroke volume and heart rate. Cardiac output refers to the blood ejected from the left ventricle into the aorta which is then circulated throughout the arterial tree providing nutrients in response to metabolic requirements via the peripheral vessels; stroke volume is the amount of blood ejected during each cardiac cycle and is the source of the change in arterial blood volume on each cardiac cycle referred to in the previous sections; heart rate is the frequency with which cardiac cycles occur. An increase in heart rate (tachycardia) may be accompanied by a decrease in stroke volume, in which case there could be either no change or a decrease in cardiac output; a decrease in heart rate (bradycardia) may be accompanied by an increase in stroke volume and possibly an increase in cardiac output. Only if both heart rate and stroke volume change in a common direction must cardiac output also reflect the change in HR. Thus unless the associated changes in cardiac output or stroke volume are important, changes in heart rate are of relatively minor significance physiologically, and either acceleratory or deceleratory changes in HR can be associated with an increased workload on the myocardium (O'Rourke, 1976).

1.8 DETERMINANTS OF BLOOD PRESSURE

Mean arterial pressure is the mean pressure during a given cardiac cycle that exists in the aorta and its major branches (Berne & Levey, 1977). It is dependent on arterial blood volume and the compliance, or elastic properties, of the arterial walls (see section 1, equations (1),(2),(3)). It is the more important measure of blood pressure, while systolic (SBP) and diastolic (DBP) are the upper and lower limits of periodic oscillations around that mean pressure (O'Rourke, 1982; Berne & Levey, 1977). But, at least partly because SBP and DBP are more readily measured non-invasively than mean pressure, they are the more commonly used to date (O'Rourke, 1982).

1.81 Transient Changes in Blood Pressure.

The change in mean arterial pressure over each cardiac cycle is the ratio between the change in arterial volume and arterial compliance (Berne & Levey, 1977). That is,

$$dP_a = dV_a / C_a \qquad (8)$$

The change in arterial volume is the difference between cardiac output (Q_i) and the peripheral runoff (Q_o) from the arteries through the arterioles and capillaries over time (dt).

$$dV_a / dt = Q_i - Q_o \qquad (9)$$

As described in the previous section, cardiac output is the product of heart rate and stroke volume. Peripheral runoff is determined by total peripheral resistance which can be expressed as the ratio of mean arterial pressure and flow at output (Berne & Levey, 1977).

Thus each change in mean arterial pressure can be described by the function

$$dP_a / dt = (Q_i - Q_o) / C_a \qquad (10)$$

indicating that arterial compliance determines the rate of mean arterial pressure change, but not the level at which it will stabilize nor the magnitude of the change (Berne & Levey, 1977). Compliance as a determinant of blood pressure change is discussed in section 1.4.

In order to be stable, cardiac output must be equal to peripheral runoff, i.e.

$$Q_i = Q_o \qquad\qquad (11)$$

When cardiac output is greater than peripheral runoff, blood pressure increases; when peripheral runoff is greater than cardiac output, blood pressure decreases.

Vasoconstriction or vasodilation of the peripheral vessels will impede or facilitate peripheral runoff. Thus, sympathetically mediated changes in the luminal diameter of the peripheral vessels will combine with the concurrent increase, decrease or no change in cardiac output (see section 1.72) to determine whether BP also has an associated increase, decrease or remains stable.

1.83 Blood Pressure from a Biological Perspective

As noted in section 1.5, the arterial system requires an appropriate pressure head to maintain functioning, and extreme values of blood pressure are taken as symptoms of dysfuction in themselves. Although the measures of BP are primarily symptomatic, and do not elucidate mechanisms, BP responses are interesting in their own right because BP is recognized as such a significant component of cardiovascular activity (Steptoe, 1981).

2 CARDIOVASCULAR CHANGES AND BEHAVIOUR

2.1 THEORETICAL DIRECTION

In 1976, Obrist presented a landmark paper, the thesis of which was that biological considerations must give direction to the study of psychological and cardiac variables.

Promotion of a biological perspective transferred research focus from the traditional emphasis on the emotional or cognitive significance of stimulus-evoked changes in cardiovascular responding to the current focus on the physiological significance of changes, in particular whether any change can be shown to be metabolically appropriate or inappropriate given the nature of the evoking stimulus event and the experimental context, with a view to identifying links between psychological events and cardiovascular dysfunction. Thus the corollary of a biological perspective is that the study of cardiovascular and psychological variables has a dual-faceted health perspective: 1) to identify responses and events which elucidate the relation between psychological events and stress-related cardiovascular dysfunction (metabolically unwarranted responding), and 2) to identify responses and events which elucidate the relation between psychological events and healthy cardiovascular functioning (metabolically warranted responding). To date, the focus has been on the former approach with particular interest in the contribution of acceleratory cardiac change to the etiology and development of hypertension (Light & Obrist, 1980a; Light & Obrist 1983; Obrist 1976).

2.11 Research Strategies

The second major consideration discussed by Obrist (1976) was the importance of considering the autonomic mediation and mechanisms of recorded cardiac change as these can have particular significance within the biological framework.

That issue is of particular importance when cardiac responses are considered as the myocardium is both vagally and sympathetically innervated, and both branches of the autonomic nervous system influence cardiac change (see section 1.71).

The issue is also important for vascular responses although the vasculature is understood to be dominated by sympathetic influences and changes in vascular responding can be interpreted to reflect changes in sympathetic activity on the vasculature (Berne & Levey, 1977; Cox, 1979).

One method of attempting to isolate response mechanisms and mediators is to use pharmacological manipulations (Obrist, 1976; Miller, 1978). Current evidence suggests beta-blockers as the pharmacological manipulation of choice when attempting to isolate sympathetic influence on the myocardium. But there are some problems associated with pharmacological manipulations (Obrist, 1976; Miller, 1978). First, they are invasive, and therefore less desirable. Second, sympathetic influence on the myocardium is predominantly beta-adrenergic, but alpha-adrenergic and humoral influences also contribute. Pharmacological manipulations are likely to control one source of sympathetic activity only; therefore they may be too specific in their action. Third, the innervation of the cardiovascular system is very complex and still not fully

understood. Thus while the primary effect of any manipulation may be known, associated side-effects are not well-documented especially if they are a-symptomatic. Although dominated by alpha-adrenergic influences, the vasculature is also sensitive to beta-adrenergic and humoral influences, and therefore any pharmacological manipulation directed toward controlling sympathetic influence on the myocardium is also likely to have some effect on the vasculature. To the extent the vasculature is affected, either in terms of arterial flow from, or venous flow to, the heart, myocardial performance is also likely to be reflexively influenced (Berne & Levey, 1977; Stehbens, 1979). Therefore any pharmacological manipulation may not be specific enough.

An alternative strategy to pharmacological manipulations is to simultaneously measure one or more cardiovascular responses of physiological significance in addition to, or instead of, HR, thereby allowing some consideration of the biological relevance of cardiovascular changes observed in response to stimulus events (Obrist, et al., 1979; Langer, et al., 1985; Ax 1953). Recording can be tonic or phasic where "tonic" and "phasic" are time-based descriptions which are still ill-defined (Dutch & Redman, 1983b). However, tonic measures are understood to have an additional value within a biological framework in that they represent periods of sustained cardiovascular activity. Phasic measures are typically beat-by-beat changes recorded immediately before, during and after stimulus presentation and thus represent momentary variations in cardiovascular responding (Dutch & Redman, 1983b; Obrist, 1976). Within the present work, measures which represent responding over a 1 minute period or longer are described as tonic (Lacey & Lacey, 1974; Obrist, 1976).

Tonic measures enable responses with different latencies and rise times to be recorded simultaneously, facilitating the study of the effect of the stimulus event on multiple components of the cardiovascular system. Because that system is an integrated homeostatic system, changes in one component may be alternatively complemented or exacerbated by changes in another component (Rushmer, 1976; Guyton, 1981). Measurement of simultaneous change in a constellation of responses should ultimately provide a better understanding of the biological significance of particular stimulus events.

2.12 Data Sampling and Subject Variables

There are two additional considerations in the study of psychological and cardiovascular variables. One is the issue of data sampling, and the other is that of subject variables.

2.121 Data Sampling

Cardiovascular responses can be sampled either discretely or continuously throughout the experimental conditions. The most common practice has been to sample discretely, particularly in the study of phasic responses. That practice has been due in part to an interest by psychologists in stimulus-evoked responding (Greenfield & Sternbach, 1972; Obrist, Black, Brener & DiCara, 1974), and also in part to practical limits imposed on sampling and analysis by traditional measurement methods which have primarily been manual. Apart from technical considerations, the quality of information provided by discrete sampling is dependent on the appropriate timing of the sampling.

The alternative, continuous sampling, is more suited to the study of cardiovascular responding from a biological perspective in that it monitors ongoing activity and detects event-related changes which are superimposed on that activity. Thus the stimulus-evoked changes can be more accurately considered in relation to overall cardiovascular functioning. The advent of computer-based data collection and analysis has meant that continuous sampling is now increasing (Martin & Venables, 1981). The present research used computer-based continuous data collection.

2.122 Subject Variables

Individuals differ in their autonomic reactivity, including their cardiovascular reactivity (Duffy, 1962; Engel, 1972). Individual differences in cardiovascular reactivity are believed to be important as a mediating variable in stress-related cardiovascular disease (Malmo & Shagass, 1949; Redman & Dutch, 1983b) and have also been associated with differences in the direction of task-generated change, in particular in HR changes (Bunnell, 1982; Lawler, 1980). Attention has typically been directed toward consistent responding over occasions, or toward the magnitude of task-generated change, but it is likely that the direction of response change is also important.

The direction of change can be either acceleratory or deceleratory, and the response measures can be either phasic or tonic. Phasic acceleratory and deceleratory changes in both cardiac and vascular responses immediately prior to and during presentation of a stimulus event are relatively well documented and have been variously interpreted as reflecting opposing emotional, cognitive or sensorimotor processes (Lacey & Lacey, 1979; Obrist, 1976; Obrist, et al., 1970; Sokolov, 1963).

Due, at least in part, to the interest in sympathetically mediated cardiac change and its potential contribution to stress-related cardiovascular dysfunction (Obrist, 1976), studies of tonic responding have focussed on acceleratory changes and minimized deceleratory ones. But to the extent that cardiovascular responding is relevant to biological considerations, change in either direction is important and the literature relating to tonic acceleratory and deceleratory changes is reviewed in the following section.

2.2 DIRECTION OF RESPONSE CHANGE⁻

Acceleratory and Deceleratory Cardiac or Vascular Responding.

From a biological perspective, tonic deceleratory responding should be an important change, and could represent a more immediate threat to survival in that acceleratory changes can be sustained for longer periods physiologically (Gunn, et al., 1972), while repeated tonic cardiac decelerations have been associated with subject deaths in laboratory animals (Miller & DiCara, 1965).

Lacey & Lacey (1974) presented 4 tasks to 24 subjects. Each task was presented twice to each subject in a counter-balanced order. The tasks included a tone detection (discrimination) task, a mental arihmetic task, a task named Rules of the Game and a combination of the mental arithmetic and tone detection tasks. Post-task judgments by subjects categorized the mental arithmetic task as the easiest, the combined mental arithmetic with tone-detection as the hardest, and Rules of the Game and tone detection as of equal and moderate difficulty. Response during task was calculated as the difference between a 1 minute alert and a 1 minute task period. The tasks were presented twice to each subject and the changes during the two presentations

were combined for analysis (Lacey & Lacey, 1974). A deceleratory change in HR occurred during the tone detection task which was rated as the most difficult, and the largest acceleratory change occurred during the mental arithmetic task, rated as the easiest. The HR changes during the other two tasks were acceleratory. Lacey & Lacey (1974) concluded that their results suggest "*that the differential requirements of tasks for attention to external events may be, for appropriately chosen tasks, a more important determinant of heart rate than the comparative difficulty of the tasks*" (p542).

A second experiment on the effect of levels of difficulty in a tone detection task over 4 sessions showed that individual differences were also influential in determining the direction of the HR response: for 10 of the 16 subjects deceleratory change during task was significant, for 3 subjects an acceleratory change during task was significant and these differences were consistent over difficulty level (Lacey & Lacey, 1974). Acceleratory and deceleratory changes have been found in continuously sampled tonic IBI and R-wave to pulse interval (RPI) measures during the foreperiod of a reaction time task (Dutch & Redman, 1983b; Redman & Dutch, 1984). Half the subjects had deceleratory changes and the other half had acceleratory changes in IBI and RPI which progressively increased in magnitude over an extended 40-beat foreperiod (Dutch & Redman, 1983b). In addition, some subjects had acceleratory, and others had deceleratory, IBI changes during a 32-second extended foreperiod, and that difference was maintained over a 6 week interval (Redman & Dutch, 1984).

Deceleratory HR changes during task for some subjects have been reported by Lawler (1980) who described both tonic and phasic data. She divided subjects

into HR reactive (n=15) and non-reactive (n=15) according to the magnitude of acceleratory HR change during a stressful mental arithmetic task. Comparison of phasic HR changes during a two-part tone detection task showed acceleratory changes for HR reactive subjects and deceleratory changes for non-reactive subjects. The tonic deceleratory changes, collected over two 5 minute periods, were not significant changes from baseline, probably because of the baseline value used. Five minute rest and task periods were alternated throughout the experimental session, beginning and ending with a rest period. Task order was counter-balanced across subjects, but response change during task was invariably calculated as the difference between task response and minute 4 of the final rest period because "*Rest 3 produced the most relaxed readings available*" (Lawler, 1980, p467). The aim of selecting the minimum activity level as a baseline is to maximize the acceleratory change produced by the stress of a novel and demanding task superimposed upon a novel and potentially threatening laboratory environment (Obrist, 1981). However, it also serves to minimize any deceleratory changes during stimulus events and may therefore mask a biologically relevant cardiovascular response to the task itself.

Bunnell (1982) collected data from the final 30 seconds of the rest period which preceded each of 4 tasks as the baseline value for that task. The tasks were mental arithmetic, word formation, a light detection reaction time task and a tone detection reaction time task. Each task was of 2 minutes duration. Change scores during task were calculated over 20 successive artefact free cardiac cycles sampled discretely during each task. Acceleratory HR change occurred during mental arithmetic, word formation, and light detection reaction time tasks. Deceleratory HR changes occurred during the tone detection reaction time task. The most HR reactive (n=16) and HR non-reactive (n=16)

subjects during the mental arithmetic task were found to have acceleratory and deceleratory changes respectively during the tone detection reaction time task. The data are describing tonic events, but sampling was not continuous and, in addition, Bunnell selected the sampled cardiac cycles on the basis of associated behavioural responses on each trial within the task. The degree of bias introduced by selective sampling has not yet been determined.

There is a clear need for systematic exploration of both the relative frequency and the magnitude of acceleratory and deceleratory changes in tonic responding, particularly during tasks. Both the frequency and magnitude of acceleratory and deceleratory cardiovascular changes were examined in the present research.

2.3 PULSE WAVE VELOCITY AND BEHAVIOUR

As discussed in section 1.1, arterial pulse wave velocity (PWV) is the propagation rate of the pressure pulse from the heart towards the periphery, and can be measured as the speed of pulse travel between two arterial sites one more distal than the other (Bramwell & Hill. 1922; McDonald, 1974; O'Rourke, 1982). PWV has an inverse relationship with the compliance or distensibility of the arterial segment over which it is measured and is therefore an index of an important criterion of the general efficiency of the circulation (Bramwell & Hill, 1922). PWV can also be expressed as arterial pulse transit time (PTT), or the time taken for the pressure pulse to travel between two arterial sites. PTT is readily measured non-invasively in *in vivo* preparations, and is therefore suitable for use in psychological and epidemiological studies which use human subjects, whether or not it is subsequently converted to PWV (Avolio,

et al., 1983; Geddes, et al., 1981). The most important distinction between the two measures is the expression of the relationship with arterial compliance: PWV has an inverse relationship, PTT has a direct relationship.

In 1976, Gribbin, Steptoe and Sleight published a paper titled "*Pulse Wave Velocity as a Measure of Blood Pressure Change*" in which high correlations between SBP, DBP and RPI were reported. That paper and its sequel (Steptoe, et al., 1976) were influential in determining the emphasis on the relationship between PTT, RPI and blood pressure (BP) in psychological studies over the following five years. These papers are considered to have fostered two sources of confusion. Firstly, Gribbin, et al., (1976) described a measure triggered by the R-wave of the electrocardiograph (EKG) and terminated by the arrival of the arterial pressure pulse at a distal arterial site as synonomous with PTT when measured between two arterial sites. In actuality, that measure is more correctly described as a pulse arrival measure and includes intra-cardiac components, in particular part of the cardiac pre-ejection period (PEP) and the left ventricular ejection time (LVET) which have no overlap with the pulse propagation time (Newlin, 1981; Newlin & Levenson, 1979; Pollack & Obrist, 1983). PEP is that time interval in the cardiac cycle which begins at the Q-wave of the EKG, includes the isovolumetric contraction period which coincides with the R-wave, and is terminated at the S-wave of the EKG. Its duration is determined by myocardial sympathetic influences, with a shorter duration reflecting greater sympathetic influence. LVET is the time interval in the caridac cycle which begins at the S-wave of the EKG when the semi-lunar valves open and blood from the left ventricle is ejected into the aorta. It terminates when the aortic valve closes generating the second heart sound and coinciding with the incisura on the descending limb of the aortic pressure

wave. The duration of LVET reflects the contractility of the myocardium: the shorter the duration, the greater the contractility (Berne & Levey, 1977; Rushmer, 1976). Secondly, Gribbin, et al., (1976) interpreted the value of pulse propagation measures as being a continuous, noninvasive measure of BP, rather than recognising the value of a continuous, noninvasive index of circulation efficiency and cardiac load. O'Rourke (1982) has argued that there is an over-emphasis on SBP and DBP as cardiovascular measures of significance which may be partly attributable to their relative accessibility historically, rather than to the particular quality of the information they provide. As outlined in section 1.4, SBP and DBP are the peak and trough pressure values respectively, are local to the site of measurement and are primarily symptomatic indicators rather than diagnostic indices. He suggests that as understanding of the mechanisms of cardiovascular dynamics increases, and technological advances make accessible noninvasive continuous measures of significant aspects of cardiovascular activity, such as arterial compliance, the importance attached to SBP and DBP will decline, and may indeed already be partially superseded by the recognition of the greater significance of mean arterial pressure (Berne & Levey, 1977; O'Rourke, 1982).

The majority of psychological studies have used the pulse arrival measure described by Steptoe and his colleagues, largely because the initiating R-wave is easy to detect and artefact free (Obrist, et al., 1979). It has variously been called ECG-initiated transit time (or ECG-TT, Redman & Dutch, 1983a, 1983b, 1984), pulse arrival time (Geddes, et al., 1981), pulse transit time (Obrist, et al., 1979; Bunnell, 1980; Light & Obrist, 1983) and E-PTT (Newlin & Levenson, 1979). In order to reduce the inevitable ensuing confusion between it and true transit time, Obrist (1981) suggested that the terms *"R-wave to*

pulse interval" *(RPI)* or *"Q⁻wave to pulse interval"* *(QPI)*, depending on the initiating component of the EKG, provided the least ambiguous, most precise nomenclature and that is the convention followed throughout this work. Evidence suggests that RPI reflects myocardial performance, not vascular functioning (Newlin & Levenson, 1979; Obrist, et al., 1979). In particular, changes in RPI parallel changes in PEP which are predominantly sympathetically determined, and thus changes in RPI can be used as an indication of changes in beta-adrenegic influences on the myocardium (Newlin, 1981; Newlin & Levenson, 1979; Obrist, et al., 1979). PTT and RPI are not equivalent measures. Not only are they each comprised of distinct components, but negative correlations have been obtained between them (Lane, et al., 1985), most likely due to an inverse relationship between PTT and PEP (Pollack & Obrist, 1983).

As an additional dependent measure of interest (Pollack & Obrist, 1983), RPI was recorded simultaneously with PTT in the present study. The RPI data are presented in Appendix 2.

2.31 *The Experimental Study of Pulse Wave Velocity*

The first paper published in the psychological literature which examined PTT was that by Geddes, et al., (1981). They investigated the relationship between PTT and DBP as well as between RPI and DBP using pharmacological manipulations on anaesthetized dogs. Geddes, et al., (1981) reported significant correlations between variations in PTT and DBP and followed Gribbin, et al., 1976 in identifying PTT as a continuous, noninvasive measure of blood pressure.

In 1983, Lane, et al., investigated the relationship between SBP and DBP with measures of R-wave to radial pulse interval, R-wave to brachial pulse interval and brachial-radial PTT. They reported only moderate correlations between SBP and RPI to either arterial site, and no correlations at all for any BP measure with PTT. They concluded that neither RPI nor PTT were satisfactory substitutes for BP measures. Marie, et al., (1984) investigated the relationship between SBP and DBP with PTT from brachial to radial, and from radial to dorsalis pedis arterial sites during exercise. They also measured RPI to brachial, radial and dorsalis pedis sites. The results were variable. Brachial-radial PTT was correlated with SBP and DBP during static exercise. Radial RPI was correlated with SBP and DBP under some exercise and rest conditions. Despite the variability of the results, they concluded that radial RPI may function as a convenient index of SBP and brachial-radial PTT may function as a less reliable index of DBP.

The use of PTT or RPI as indices of blood pressure change has been criticized by Pollack & Obrist (1983) as more likely to obscure, or confuse, than clarify, the issues involved. In that study, Pollack & Obrist (1983) recorded Q-wave to pulse interval (QRPI) and then used the chart-recorded wave form to disentangle the component parts: PEP, LVET, aortic-radial PTT (AR-PTT) and Q-wave to second heart sound. They found that QRPI and the component parts PEP and AR-PTT were related to SBP and DBP and to each other. PEP was reported to be the primary contributor to variations in QRPI, and QRPI was reported to vary inversely with SBP and DBP, reflecting an inverse relationship between AR-PTT and PEP. They concluded: *"Both PTT and PEP are variables worthy in their own right of study in psychophysiological contexts. For example, PTT might provide important information about behaviourally-related changes in*

arterial wall stiffness in health and disease; PEP might provide information about behaviourally-related normal and abnormal left ventricular function. In order to fully develop the usefulness of these time intervals to the fullest, each must be studied individually with the appropriate consideration of the physiological mechanisms determining the duration of each. Neither should be demoted in significance to a mere index of something else, nor ignored as only a trivial confounder of some other relationship of more interest." (p.27).

The study of PTT in a psychophysiological context has been developed by Dutch & Redman who have demonstrated the sensitivity of PTT to psychological events or stress (Dutch & Redman, 1983), the classical conditioning of PTT (Redman & Dutch, 1983; 1984), and the hyper-reactivity of PTT in sensitized subjects during blood pressure measurements (Redman & Dutch, 1984b). In the Dutch & Redman (1983) study, brachial to radial PTT and popliteal to dorsalis pedis PTT were recorded while subjects participated in laboratory stressors: cold pressor test, problem solving, unsignalled reaction time and a video game. Significant acceleratory PTT changes occurred during all stressors, indicating increased rigidity in the vessel walls of the arterial segment over which measurement took place. They conclude: *"Pulse transit time as measured here appears to be a sensitive and robust response which can be measured noninvasively on a continous basis and provides information on significant aspects of cardiovascular activity. It therefore seems likely that the use of PTT may enable psychologists to make a more substantial contribution to the understanding of the relationship between psychogical events and cardiovascular activity." (p.609).*

Apart from a single exception by Marie, et al., (1984) who included a measure of PTT from the radial to the dorsalis pedis arterial sites, the psychological study of PTT has employed measures recorded over short peripheral arterial segments. The associated transient changes in PTT reflect local changes in peripheral vasoconstriction and vasodilation, and provide information on changes in peripheral resistance.

Information on systemic arterial distensibility and left ventricular load can be accurately and noninvasively determined if PTT is measured over a long arterial segment which includes major vessels, particularly the aorta, because that measure reflects properties of the whole vascular segment (Avolio, et al., 1983). Therefore, in the present work, PTT was recorded from the auricular artery on the right ear (Lance & Spodick, 1977) to the dorsalis pedis artery on the right foot in order to maximize the significance of the information obtained. The work followed Dutch & Redman (1983) in examining the PTT response to psychological events.

2.4 ACTIVE/PASSIVE COPING

A major contribution by Obrist (1976) was to reaffirm the biological emphasis and to offer a means for the experimental study of cardiac responding from a biological perspective described as the study of active/passive coping. The study of active/passive coping has been developed as being of particular relevance in the study of the etiology and development of essential hypertension (Light, 1981).

The experimental investigation of active/passive coping was fully detailed in 1978 (Obrist, et al., 1978). It was argued that the extent to which a

subject has control over experimental contingencies would determine the extent of the sympathetic influence on the myocardium and be manifest in the magnitude of cardiac acceleratory changes during measurement. Increased sympathetic activity is believed to underlie the symptoms of chronic essential hypertension (Julius & Esler, 1975) and thus the study of possible links between environmental contingencies, behavioural states and cardiac responding is likely to be relevant in classifying the etiology and the development of that clinical condition (Light, 1981; Obrist, et al., 1978).

In the Obrist, et al (1978) study, male subjects participated in avoidance reaction time tasks with electric shocks contingent on set criterion response latency. In addition, a monetary incentive for performance faster than criterion was also offered. Three conditions, or levels of difficulty, were described: "easy", where subjects could readily meet the set criterion (100% success), or had control over events; "hard", where subjects could meet the set criterion some, but not all, of the time (50% success), or partial control; and "impossible", where subjects could rarely meet the criterion (0% success) or no control. Hence, control was operationally defined in terms of percent success, but shock delivery was actually equated across the three conditions. It was predicted that task engagement or involvement would be maximal during the hard condition, generating effortful active coping characterized by sympathetically mediated, large magnitude and sustained acceleratory cardiac changes. It was further hypothesized that engagement would be minimal during the easy and impossible conditions, generating passive coping characterized by vagally mediated, smaller magnitude, more transient acceleratory cardiac changes. Thus subject engagement or involvement was considered to be the defining characteristic of the behavioural coping states. Each subject participated in

one 15-minute reaction time condition, preceded by a 90-second cold pressor condition and an 8-minute pornographic movie condition, both expected to generate passive coping. Rest periods, from which base level readings were taken, were interspersed between stressor conditions (tasks). Heart rate (HR), SBP, DBP and the maximum slope of the ascending limb of the carotid pulse wave (carotid dP/dt) were recorded at discrete intervals during minutes 1-2, 3-4, 8-9 and 13-14 of each condition. Acceleratory changes during each stressor condition were calculated from the baseline recording taken prior to that condition.

Measures recorded during minute 1-2 showed no difference in response changes between the 3 reaction time conditions, but the cold pressor and the pornographic movie generated smaller acceleratory changes in HR, SBP and carotid dP/dt and larger magnitude acceleratory changes in DBP than during the reaction time tasks. Measures recorded during the second measurement period (minute 3-4) of the reaction time tasks showed larger HR and carotid dP/dt acceleratory changes during the hard condition than during the easy or impossible conditions. The HR response was sustained from that point for the duration of the conditions. Larger SBP changes during the hard condition were not apparent until minute 8-9, and DBP changes were not sensitive to the difference in difficulty level (Obrist, et al., 1978).

The avoidance reaction time condition was replicated (Light & Obrist, 1980b) measuring HR, SBP, DBP, carotid dP/dt and R-wave to pulse interval (RPI). Half the subjects either had the opportunity to avoid shock, contingent on response latency, the other half acted as yoked controls. Subjects in the avoidance conditions had larger acceleratory changes in HR, SBP, and RPI than

the control subjects. Responding in the avoidance condition was interpreted as reflecting active coping, while that in the control condition was interpreted as reflecting passive coping.

The active/passive coping concept has been applied to the study of individual differences in cardiovascular reactivity (Hastrup, Light & Obrist, 1982) as high cardiovascular reactivity is hypothesized to be a possible precursor of essential hypertension (Light & Obrist, 1980a). In one such study (Light & Obrist, 1983), twentyfour subjects were each assigned to a reaction time task at one of the three levels of difficulty described above: easy, hard or impossible. Monetary reward was contingent on response latency, but electric shock was not used. Responses measured were HR, BP, and RPI. In addition, cardiac pre-ejection period (PEP), left ventricular ejection time (LVET) and PTT were handscored from the chart record. Each subject then returned to the laboratory one week later to participate in a 35 minute period of uninterrupted relaxation in which they were instructed to doze if they could. Recordings were obtained for 3 minute intervals every 10 minutes, and basal readings were scored as the lowest 1 minute of HR obtained, the lowest two consecutive BP readings and the longest mean RPI. Task-generated change was calculated from that baseline for HR, SBP, DBP and RPI. Task-generated changes in PEP, LVET and PTT were calculated from pre-stress (task) baseline due to loss of relaxation data (Light & Obrist, 1983).

Light & Obrist (1983) report larger, more sustained changes in PEP, SBP, DBP and RPI during the easy and hard conditions than during the impossible conditions. These findings are considered to contradict fundamental tenets of the active/passive hypothesis; firstly, because the HR responses were not

the active/passive hypothesis; firstly, because the HR responses were not determined by environmental contingencies, namely task difficulty, and secondly, because the hard task was not distinguished from the easy task by the generated cardiac change and, by inference, the associated level of engagement. Thus it is not clear whether task demands or difficulty determine cardiac responses only under avoidance conditions, nor whether the cardiac response to hard tasks is also only unique under avoidance conditions.

The Light & Obrist (1983) study differed from the Obrist, et al (1978) study in two procedural matters: firstly, no electric shock was used; secondly, the baseline values were obtained as minimum readings on the second exposure to the laboratory thus maximizing any acceleratory changes which occurred during the task one week previously. If the contradictory results are attributable to the absence of contingent shock then the generality of the active/passive concept would appear to be severely limited. However, it seems likely that the discrepancy in the results may have been due to the selection of the baseline, which was justified as being appropriate for the paper's emphasis on individual differences in HR reactivity. Subjects were divided into 12 high HR reactors and 12 low HR reactors in each condition on the basis of the difference between the HR relaxation baseline and the HR response during the first minute of task. High and low reactors were differentiated during the reaction time task in measures of HR, SBP, RPI, PEP and LVET responses during the reaction time task regardless of difficulty suggesting that task requirements may be less influential than individual reactivity, or subject variables, in determining cardiac responses.

The studies on active/passive coping have several limitations. Firstly, active coping as first proposed has only been demonstrated under avoidance conditions, and using reaction time tasks. Secondly, no women have been included as subjects. Thirdly, all the reported studies have involved single session recording and between subjects comparisons on the critical responses. Obrist (1981) has noted that the cardiac response is typically largest on the first exposure to the laboratory and reduced on subsequent exposures. Thus recording on the first session, like basal value selection, can maximize some manifest responses and minimize others. If active coping is indeed important within a biological perspective in general, and in the etiology and development of essential hypertension, which is a progressive disease, in particular (Obrist, 1976), then it should be robust enough to be demonstrated under various conditions, over multiple exposures, during a variety of tasks and involving a wider range of subjects.

3 EXPERIMENT 1

Arterial Pulse Transit Time (PTT)

Changes in Response to Psychological Events

INTRODUCTION

Obrist (1976, 1981) has suggested that task-generated changes in cardiac responses may index behavioural states, with the behavioural states being defined in terms of the prevailing environmental conditions. The behavioural states with which he is particularly concerned are categorized as either active or passive coping and the environmental conditions with which he is particularly concerned are defined in terms of task requirements: easy, hard or impossible (Obrist, 1976; Obrist, et al., 1978; Light 1981; Light and Obrist, 1983).

Active coping is produced by tasks with hard requirements (operationally defined as having about 50% success rate) and is characterized by large magnitude, sustained acceleratory changes in cardiac responses such as heart rate (HR) or inter-beat interval (IBI). Passive coping is produced by tasks with easy (100% success) or impossible (0% success) requirements and is characterized by smaller, more transient cardiac accelerations. Alternatively, passive coping is generated by tasks such as the cold pressor, a pornographic movie (Obrist, et al., 1978) or a simple reaction time task (Obrist, 1976). The fundamental distinction between the two states is the subject's level of engagement or involvement (Light, 1981; Obrist, 1981).

Active coping is associated with sympathetically mediated cardiac activity while passive coping is indicative of vagal control over cardiac responses (Light, 1981; Obrist 1976, 1981). Active coping is therefore of particular interest in psychophysiological investigation as sympathetic mediation of cardiovascular responding is seen as a critical feature which links stressful events with cardiovascular disease (Julius & Esler, 1975; Obrist, 1976, 1981).

As discussed in Chapter 2, the experimental study of active/passive coping typically describes male subjects in avoidance reaction time tasks with contingent electric shocks and pre-set success criteria (Obrist, et al., 1978; Light & Obrist, 1980). Under conditions of monetary incentive, HR changes were not determined by task requirements and changes in PEP, LVET and RPI, the other cardiac responses measured, were equivalent during the easy and hard tasks (Light & Obrist, 1983), thus contradicting two of the basic tenets of the active/passive hypothesis. Women have not been included in any of the studies and it is not known whether their cardiac responses are also determined by task requirements.

The purpose of the following experiment was to determine the generality of the active/passive hypothesis by using a variety of tasks, by varying experimental procedures; by including measures of task performance; by including women as subjects and by extending the range of cardiovascular responses measured.

In the present experiment, IBI was measured in order to replicate previous work which has used HR in the study of active passive coping (Obrist, et al., 1978) and systemic PTT was recorded as a measure of sympathetic (alpha and beta adrenergic) influence on the vasculature. As PTT has not been previously

measured in the study of active/passive coping its measurement was exploratory and was intended to begin a quantification of vascular response changes under conditions of active and passive coping. Both men and women subjects participated under identical conditions to test the robustness of the active/passive phenomenon.

Two tasks likely to generate active coping were selected. One of these, mental arithmetic performed under time stress, has been reported to reliably generate large magnitude, sustained acceleratory changes in cardiac responses for male subjects in particular (Lane et al., 1983) but less reliably for female subjects (Dembroski, et al., 1978; Lane, et al., 1983). Thus a second task, letter sequence problem solving performed under time stress, was included as evidence suggests that women are cardiovascularly more reactive to verbal tasks (Dembroski, et al.1978; Lane,et al., 1978).

Two tasks likely to generate passive coping were also selected. One of these, simple reaction time, has been cited by Obrist (1976) as generating passive coping; the other was a voluntary button pressing task adapted from a personal tempo task (Corcoran, 1981) with a priori no demands associated with it in that each subject was asked to press the button if and only if he or she wanted to.

Response divergence has previously been reported for IBI and RPI during the foreperiod of a signalled reaction time task (Dutch & Redman, 1983(b); Redman & Dutch, 1984) as well as for IBI during unsignalled tone-detection reaction tasks (Lawler, 1980; Bunnell, 1982) and a further aim of the present study was to investigate whether similar response divergence would be apparent for any or all cardiovascular responses measured.

METHOD

Subjects

Fifty undergraduate psychology students participated in the experiment, 31 women and 19 men. None had any previous experience of being subjects in a psychophysiological experiment.

Apparatus and Physiological Recording

Experimental events and data collection were controlled by a Hewlett-Packard 9835B desktop computer in a control room adjacent to the experimental room. Instructions and tasks were presented to the subject via a Hewlett-Packard 2623A Graphics terminal (VDU) in the experimental room.

Beckman electrodes were used to detect the EKG signal which was coupled to a Type S Beckman Dynograph, then amplified, filtered and fed into a Hewlett-Packard 5823A counter-timer which measured the interval between successive R-waves with millisecond accuracy to give IBI.

Two photometric transducers (photocells) were used to detect the arterial pulses, one at the auricular artery on the right ear and one at the dorsalis pedis artery on the right foot. The ear was chosen as the proximal site because of the stability of the signal received from there (Lance & Spodick, 1977).

The pulse signals were ac coupled to the Beckman Dynograph then filtered at 10Hz LP. The pulse from the auricular artery was fed into channel 1 of a Gould

dual channel storage oscilloscope (OS 4000) and the pulse from the dorsalis pedis artery was fed into channel 2. Each sweep of the oscilloscope was triggered by the R-wave of the EKG, with sweep speed set at 0.05cm/sec. Pulse signals were digitized by the oscilloscope and output to the computer.

Arterial pulse transit time (PTT) was measured as the difference in arrival time of the pulse at the auricular and dorsalis pedis arteries. It was calculated as the interval between the foot of the auricular pulse and that of the dorsalis pedis pulse. The foot of the pulse was determined as the point which is at 12.5% of the pulse height, as measurements between points at that height of the pulse were relatively free of noise related artefact, were minimally altered by changes in the slope of the pulse and were accurate to 0.5ms (Dutch & Redman, 1983).

PROCEDURE

Subjects each attended for two 1-hour sessions, selecting their own preferred times. During each session, two of four tasks were completed with task order counterbalanced across subjects.

After arrival at the laboratory, each subject was interviewed briefly, then entered a sound-attenuated, temperature-controlled experimental room and was seated in a comfortable chair with both legs extended horizontally and supported under the calves and heels. The experimenter positioned the transducers, then dimmed the light and the subject was left alone.

Each session began with a 10-minute Initial Baseline during which a message asking the subject to relax was displayed on the VDU in the experimental room.

The initial baseline was followed by a 1-minute pre-task baseline with no change in the VDU display. Instructions for the imminent task were then presented on the VDU and displayed for 1 minute. The instructions were followed by a 4-minute task period and a 4-minute post-task period. The sequence from pre-task baseline to post-task recovery was repeated for the second task of the session and the session was then terminated.

The following four tasks were presented to each subject.

Mental Arithmetic.

The computer's random number seed was used to generate equations of the form:

$(x+y)*z+10=.$

Each equation was presented to the subject via the VDU and was displayed on the VDU for 4 seconds. A cue "ANS" was then displayed after which the subject was given 1 second to key in the answer (2 digits) to the equation via the terminal console before the screen cleared and the next equation was presented. Each response was categorized as either correct, an error of commission (incorrectly answered) or an error of omission (failure to answer). No feedback was given to the subject.

Problem Solving.

The Nufferno Speed Test (Furneaux, 1956) formed the basis of the problem solving task. A letter sequence was presented to the subject via the VDU. Each sequence was displayed on the VDU for 4 seconds. A cue "ANS" was then displayed after which the subject was given 1 second to key in the next 2 letters of the sequence via the terminal console before the screen cleared and

the next sequence was presented. Each response was evaluated as either correct, an error of commission (answered incorrectly) or an error of omission (failure to answer). No feedback was given to the subject.

Reaction Time.

This task consisted of unsignalled fixed interval reaction times, with an imperative occurring every 5 seconds. Every time a tone sounded and the word "GO" appeared on the screen, the subject was required to press a button as quickly as possible. Response latency was measured in milliseconds using a Hewlett-Packard counter-timer (5308A). No feedback was given to the subject.

Personal Tempo.

The subject was provided with a small box encasing a microswitch activated by pressing a button, and told to press the button if and when he or she wanted to. The message "please press the button at your own pace" was displayed on the VDU for the duration of the task. Both the number of button presses and the latency between presses were recorded.

RESULTS

The median value for each cardiovascular response over every minute of both sessions was obtained. These medians formed the bases of all the reported analyses.

Analyses of Absolute Levels

Absolute levels of cardiovascular activity during the initial baseline were analysed for evidence both of sex differences and of deceleratory changes over time which would indicate the subject settling down. Absolute levels of cardiovascular activity during the pre-task baseline periods were analysed for evidence both of sex differences and of task-related differences which could confound subsequent task-generated responding. Analyses of absolute levels of cardiovascular activity during instruction, task and post-task periods are presented in Appendix 3.

Initial Baseline

The absolute levels of IBI and PTT during the initial baseline were analysed using a Sex (2) X Minute (10) X Session (2) analysis of variance (ANOVA) with repeated measures on the last two factors. A general deceleration occurred over the minutes of the initial baseline for both responses (Main Effect of Minutes: IBI, $F(9/432)=3.28$, $p<.0008$; PTT, $F(9/432)=3.46$, $p<.0004$).

The lack of any Sex or Session Effects or Interactions indicated there was no difference in the absolute levels of any cardiovascular responses between sexes or across days. No other significant effects or interactions were found.

Pre-task Baseline Periods

The absolute levels of IBI and PTT during the pre-task baseline periods were analysed using a Sex (2) X Task (4) X Session (2) ANOVA (2) with repeated measures on the last two factors. In addition Task Order Effects were examined

using a Sex (2) X Task (4) X Order (4) ANOVA with repeated measures. No significant effects or interactions were found.

Analyses of Response Change

Response change from pre-task baseline was calculated for IBI and PTT during each pre-task instruction period and for each minute of all task periods. Analyses of post-task periods are given in Appendix 4. Analyses were performed to ascertain the magnitude and variability of the changes which occurred during each of the experimental periods. The analyses to be described were performed on the actual change scores calculated. Those scores were converted to mean percentage change from pre-task baseline for use in graphs and tables. Mean percent changes for men and women over each condition are displayed in Figure A, Appendix 1.

Instruction Periods

The change scores during the instruction periods were analysed using a Sex (2) X Task (4) X Session (2) ANOVA with repeated measures on the last two factors. A secondary analysis which substituted a factor of Task Order for Task was also performed to assess the presence of any order effect.

The magnitude of instruction-generated accelerations in IBI was sensitive to the nature of the imminent task (Main Effect of Task, $F(3/144)=4.78$, $p<.004$). Follow-up Ryan-Einot-Gabriel-Welsch multiple F tests (REGWF) on the associated means confirmed a significant difference between instruction-generated change in the following order of decreasing magnitude: mental arithmetic (-42ms), problem solving (-34ms), reaction time (-25ms) and personal tempo (-15ms).

No other significant effects or interactions were found.

Task Periods

The change scores during the task periods were analysed using a Sex (2) X Task (4) X Minute (4) X Session (2) repeated measures ANOVA. A secondary analysis which substituted a factor of Task Order for Task was also performed to assess the presence of an order effect.

The magnitude of task-generated accelerations in IBI was sensitive to task content (Main Effect of Task: IBI, $F(3/144)=32.78$, $p<.0001$). Follow up REGWF tests on the means confirmed a significant difference between task-generated acceleratory responses for IBI in the following order of decreasing magnitude across tasks: mental arithmetic (-72ms), problem solving (-60ms), reaction time (-11ms) and personal tempo (+3ms). That effect is shown in Figure 1.1, graphed as a function of task difficulty.

Further, women had an acceleratory IBI response during task which increased in magnitude over successive task minutes while the men had an acceleratory response which decreased in magnitude over successive task minutes (Sex X Minutes Interaction, $F(3/144)=3.11$, $p<.03$). That interaction is shown in Figure 1.2 accompanied by the post-task response which followed the changes.

There were no other significant effects or interactions involving IBI.

The magnitude of task-generated accelerations in PTT was found to differ over successive minutes of task as a function of the day (Session X Minute Interaction, $F(3/144)=5.22$, $p<.002$). The PTT response diminished over successive minutes of tasks during Session 1, but increased over successive minutes of tasks during Session 2. The Interaction is shown in Table 1.1.

The secondary analysis confirmed that the effect was also present within sessions (Order X Minutes Interaction, $F(9/432)=2.59$, $p<.007$). A deceleratory PTT change occurred to the first task, a diminishing acceleratory change occurred to the second task (both during session 1), then an acceleratory response occurred to both tasks during session 2 (tasks 3 and 4). That Interaction is shown in Figure 1.3 with the post-task recovery which followed each task-generated response.
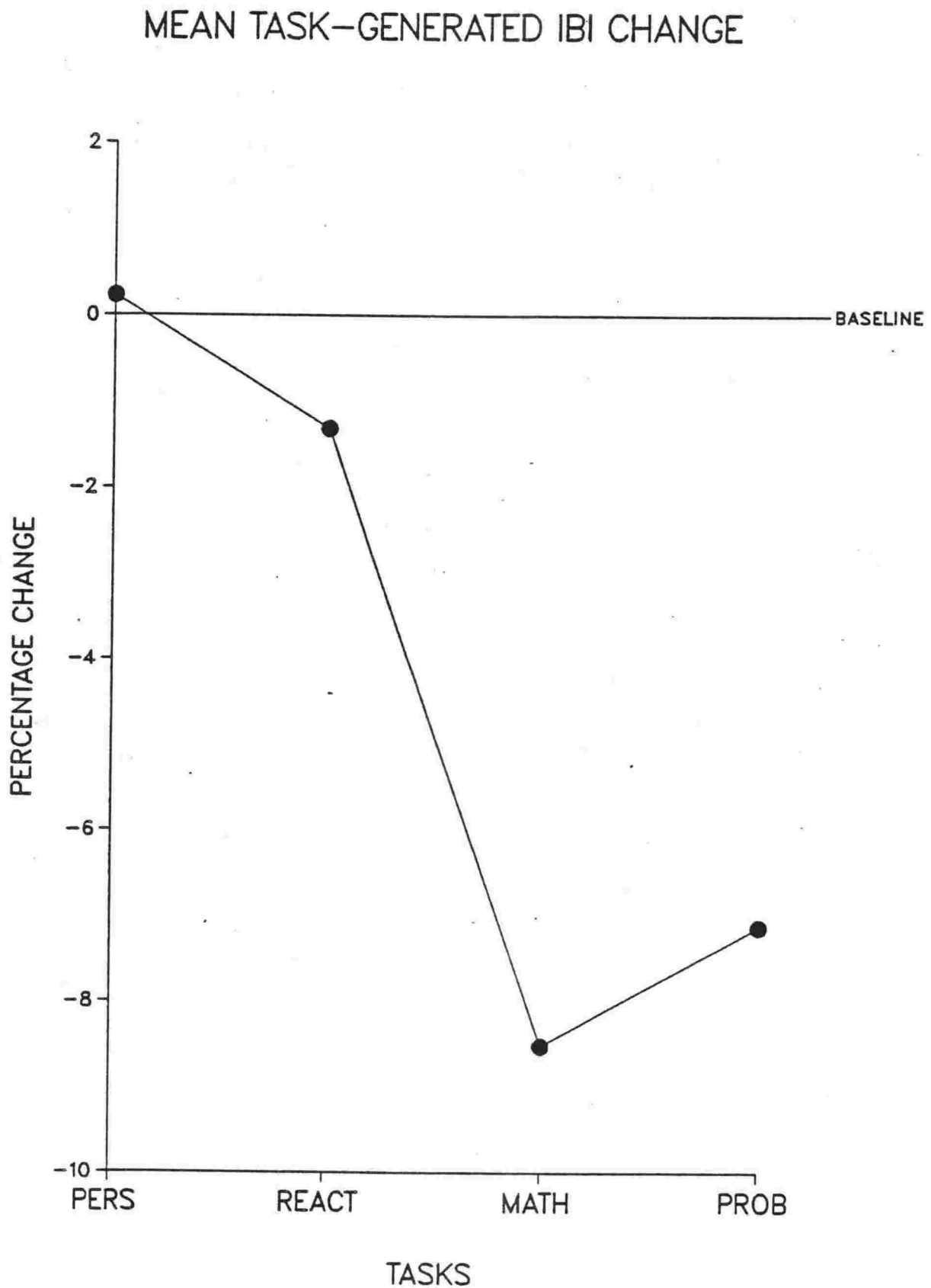
Figure 1.1 Percent change in Interbeat Interval during tasks
(Pers=personal tempo; React=reaction time;
Math=mental arithmetic; Prob=problem solving)
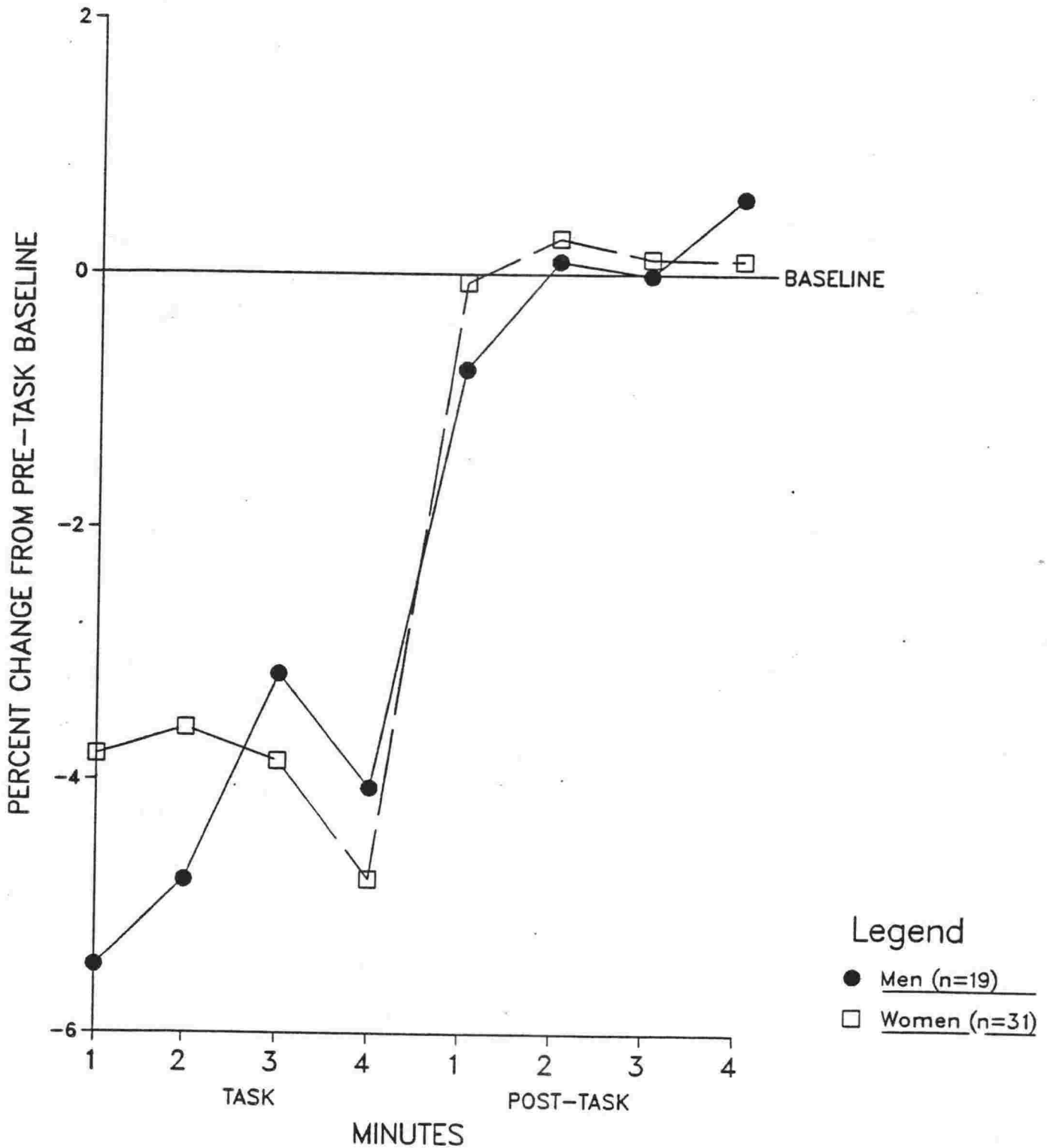
Figure 1.2 The Interbeat Interval Response during task and post-task, showing the Sex X Minute Interaction during task.

TABLE 1.1

The Session X Minute Interaction (p<0.002) found for task-generated changes in Arterial Pulse Transit Time shown as percentage change from pre-task baseline levels.

Task Minutes	Session 1 % Change	Session 2 % Change
1	-2.38	-1.35
2	-1.46	-1.08
3	+0.48	-1.19
4	+0.89	-2.43

ARTERIAL PULSE TRANSIT TIME RESPONSE
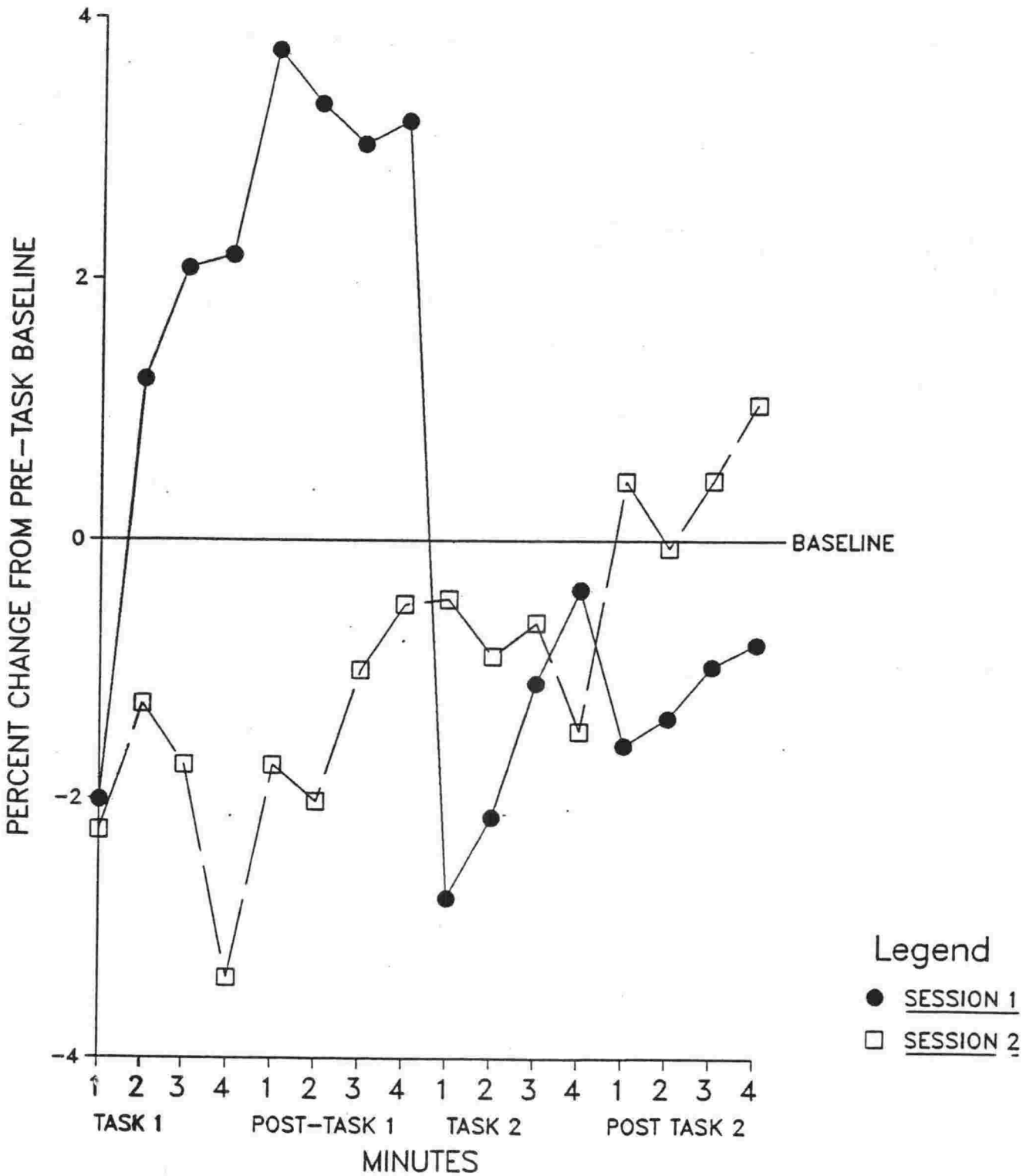
Figure 1.3 The Arterial Pulse Transit Time Response showing task order and session effects during task and post—task.

Behavioural Task Performance

The behavioural response made to each problem solving and mental arithmetic item by each subject was scored as either correct, an error of omission or an error of commission. The overall score for each of the three measures was then converted to a percentage of the total number of items presented in that task for that subject. Reaction time task performance for each subject was calculated as mean response latency over all trials. Personal tempo performance was scored as both the total number of button presses made and the mean interval between button presses.

Relationship Between Performance & Cardiovascular Responses

A Pearson Product Moment correlation analysis was performed between cardiovascular response change during each task period and behavioural performance on that task. That analysis indicated relationships between the magnitude of change in cardiovascular response and some measures of task performance. These relationships were then tested using regression analyses.

The regression analyses confirmed that a larger change in IBI during mental arithmetic and problem solving tasks was associated with fewer errors of commission made during the respective tasks (mental arithmetic ($F(1/48)=4.101$, $p<.05$), problem solving ($F(1/48)=4.531$, $p<.04$)).

A larger change in PTT during the problem solving task was associated with more errors of omission ($F(1/48)=10.484$, $p<.002$) and fewer correct responses ($F(1/48)=9.47$, $p<.003$). A larger change in PTT during the reaction time task was associated with a slower reaction time response latency ($F(1/48)=8.545$,

p<.005). A larger change in PTT during the personal tempo task was associated with more button presses made (F(1/48)=7.284, p<.01).

Relationship Between Task Performance and Sex

Behavioural performance was also analysed using a simple ANOVA with a factor of sex. There was no difference between the women's and the men's task performance on any behavioural measure.

Direction of Response Change

As indicated in Chapter 2, recent studies have found that an examination of the data of individual subjects reveals that on certain tasks some subjects may respond with accelerations in IBI while others respond with decelerations. In a follow-up of those findings, a further analysis of the data was undertaken in order to determine, firstly, whether or not that divergence occurred in the present experiment, and secondly, if divergence did occur on one response measure, whether there was any particular pattern or clustering of other cardiovascular responses associated with the diverging response.

Because the present experiment used multiple tasks and multiple cardiovascular measures, it seemed appropriate firstly to classify subjects as accelerators or decelerators on each task and for each cardiovascular response separately, and then, secondly, to consider the question of pattern of changes in the other concurrently measured cardiovascular responses. The method of classifying subjects as accelerators or decelerators was to first find the mean of a task-generated cardiovascular change score; then if the mean was less than pre-task baseline, the subject was classified as an accelerator, but if the

mean was greater than pre-task baseline, the subject was classified as a decelerator. That method of classifying subjects was first applied to IBI for each of the four tasks, see Table 1.2, with the minute-by-minute IBI response change shown in Figure 1.4; then to PTT, see Table 1.3, with the minute-by-minute PTT response change shown in Figure 1.5.

It should be noted that the tables also present details of the concurrently measured other cardiovascular change scores, and that the mean change scores for all the groupings thus formed were tested for the significance of their deviation from pre-task baseline using t-tests for paired means comparisons. The results of those tests are also indicated in the tables.

As will be seen from the tables, IBI responses showed divergence on reaction time and personal tempo tasks, but PTT responses showed divergence on all of the tasks.

In addition, the data from these groupings were tested for differences between accelerators and decelerators in sex, in pre-task baseline levels and in behavioural measures of task performance. No differences were found.

Individual Response Stereotypy

The possibility that response direction formed part of an individual response stereotypy, that is, response direction across tasks characterized responding in a particular cardiovascular measure for any or all subjects was then considered. The numbers of subjects who had acceleratory or deceleratory changes to multiple tasks for each cardiovascular response are shown in Table 1.4.

Table 1.2

Interbeat Interval (IBI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup.[a] The concurrently measured Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds. The significance of deviation from pre-task baseline for each change is indicated. (Math=mental arithmetic, Prob=problem solving, Reac=reaction time, Pers=personal tempo)..

Task	N	Acceleratory IBI	Concurrently Measured PTT
Math	41	-89*	-1.5
Prob	44	-72*	-1.8
Reac	25	-37*	-1.4
Pers	20	-34*	-0.2

Task	N	Deceleratory IBI	Concurrently Measured PTT
Math	7	+ 1	-7.9*
Prob	6	+29	+1.4
Reac	23	+17*	+2.1
Pers	28	+30*	-0.1

[a] two subjects had zero response change on each task except problem solving
* p<.05 or better

Table 1.3

Arterial Pulse Transit Time (PTT) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup shown with the concurrently measured Interbeat Interval (IBI) changes also in milliseconds. The significance of deviation from pre-task baseline for each change is indicated. (Math=mental arithmetic, Prob=problem solving, Reac=reaction time, Pers=personal tempo).

Task	N	Acceleratory PTT	Concurrently Measured IBI
Math	36	− 8.9*	−63*
Prob	31	− 5.9*	−64*
Reac	21	− 6.2*	−16*
Pers	29	− 3.5*	− 2

Task	N	Deceleratory PTT	Concurrently Measured IBI
Math	14	+13.1*	−95*
Prob	19	+ 5.8*	−54*
Reac	29	+ 5.2*	− 7
Pers	21	+ 4.3*	+11

* p<.05 or better
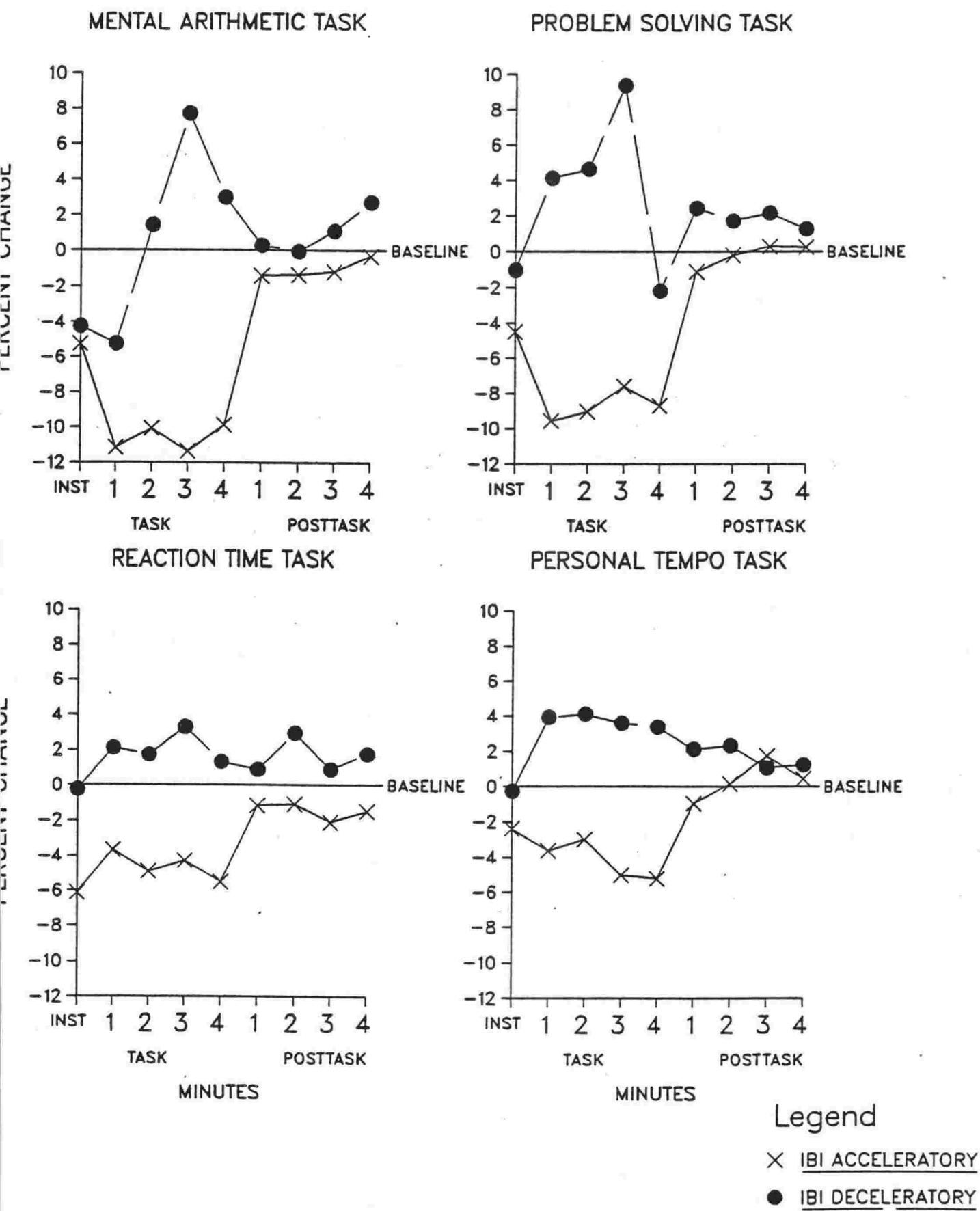
Figure 1.4 Minute−by−minute Interbeat Interval
acceleratory and deceleratory responses, Experiment 1.
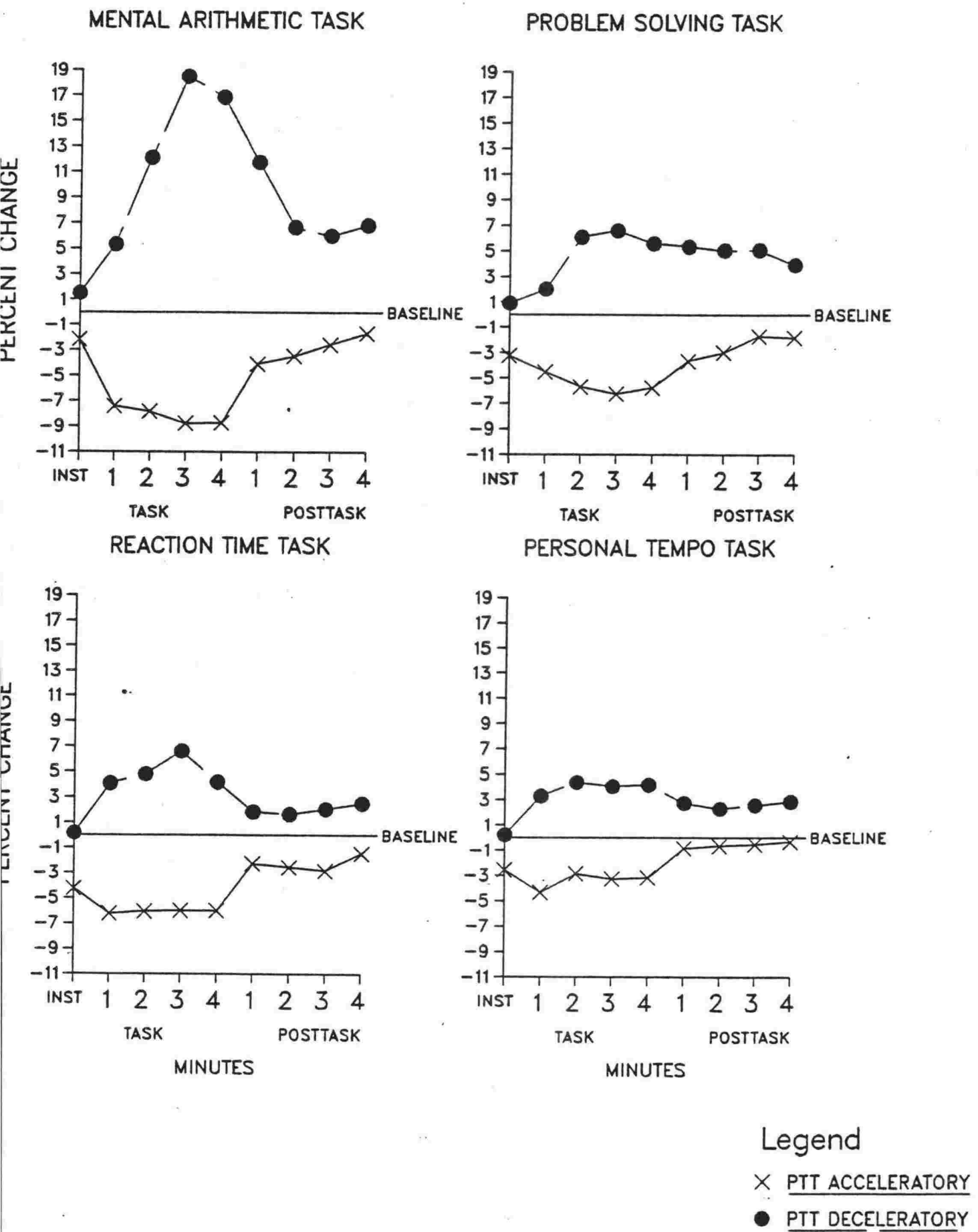
Figure 1.5 Minute—by—minute Arterial Pulse Transit Time acceleratory and deceleratory responses, Experiment 1

Table 1.4

The number of subjects who had task-generated changes in a single direction over multiple tasks for each response.

Response	IBI				PTT		
	4	3	2		4	3	2
Accelerators	10	21	12		7	17	16
Decelerators	1	6			4	6	

DISCUSSION

The central tenet of the active/passive coping hypothesis is that the magnitude of cardiac acceleratory change is determined by the level of task difficulty and the subject's ability to cope (Obrist, 1976).

The primary consideration of the current experiment was to examine the generality of the hypothesis that active coping, characterized by sympathetic mediation of cardiac activity could be demonstrated using a wider variety of tasks and a less restricted subject population than had been reported previously.

If successful coping can be measured in terms of percentage problems correct, then subjects were more successful during the mental arithmetic task than during the problem solving task. The 48% success rate obtained during mental arithmetic approximates that which defines a hard task (50%) and generates active coping (Obrist, et al., 1978) while the 37% success rate during the problem solving task places it between the hard and the impossible (Obrist, et al., 1978). As predicted by the active/passive hypothesis, magnitude of IBI acceleration was largest during the second most difficult task, and smallest during the least difficult. If tasks were placed along a continuum of difficulty with personal tempo at the lower extreme and problem solving at the higher extreme, then magnitude of IBI change would show a curvilinear relationship with task difficulty (see Figure 1.1). The IBI data suggest that active versus passive coping may represent the extremes on that function which relates the magnitude of cardiac change to task demand along a continuum of behavioural coping, rather than two dichotomous behavioural states.

Notably, exactly the same magnitude effect was produced on the IBI response by the instructions which preceded the tasks as by the tasks themselves. In fact, the magnitude of acceleratory change was larger during the instructions prior to the reaction and personal tempo tasks than during the tasks themselves. These data confirm IBI as a measure which is sensitive to anticipation (Gunn, et al., 1972) and suggest that behavioural states similar to those found during the task could also be generated by instructions, consistent with an interpretation of "psychological engagement" hypothesized to form one component of the coping responses (Light , 1981). In addition, larger IBI changes were associated with fewer errors on active tasks, consistent with the expectation that partially successful coping should be associated with large magnitude cardiac, and in particular HR, responses (Obrist, et al., 1978).

The sex differences obtained suggest women either had more reactive HR or were more actively engaged in the task. These data point out the need to include women in studies of active coping as it seems that women's cardiac responses are also determined by task requirements.

The relative frequency of acceleratory IBI changes was determined by task difficulty as defined by task type. Deceleratory IBI changes were only significant during the reaction time and personal tempo tasks.

In contrast to IBI, although larger changes in PTT were associated with inferior behavioural performance on all tasks except mental arithmetic, PTT appeared to be insensitive to task difficulty and PTT responses were characterized by extraordinary directional variability. All acceleratory and deceleratory changes began during pre-task instructions, maximized during task

and returned to pre-task levels during the post-task recovery (see Figure 1.4). A second experiment was designed specifically to confirm the directional variability of PTT changes in response to tasks.

4 EXPERIMENT 2

Individual Differences in the Arterial Pulse Transit Time
Response to Problem Solving at Two Levels of Difficulty

INTRODUCTION

Experiment 2 was designed to further test the directional variability of PTT in particular by manipulating task difficulty and controlling task type.

It was hypothesized that the variety of tasks used in Experiment 1 could have confounded task difficulty with task type, and that confounding might have resulted in the manifest directional variability in PTT due to novelty of task, rather than difficulty. If novelty was the determinant of directional variability, then the presentation of two problem solving tasks at different levels of difficulty should eliminate that variability. Alternatively, if the direction of PTT response formed part of an idiosyncratic response to a particular task for each subject then the use of two problem solving tasks would allow the direction of the response during those two tasks to be compared, and further, would allow the direction of PTT response during those two tasks to also be compared with the direction of the response during the problem solving task in Experiment 1.

In addition, it was noted that only acceleratory IBI changes were significant during the problem solving and mental arithmetic tasks in Experiment 1, but that both acceleratory and deceleratory IBI changes were

significant during reaction time and personal tempo. If task difficulty, not task type, was the determinant of the frequency of occurrence of IBI acceleratory changes, then problem solving tasks at different levels of difficulty should not only generate different magnitudes of IBI changes, but should also generate different numbers of IBI accelerators and decelerators.

Finally, it was also considered important to replicate the demonstrated relationship between magnitude of PTT change and task performance measures as a manifest relationship between task parameters and PTT response change.

Experienced women and men subjects were presented with a problem solving task at two levels of difficulty. One of the tasks was the problem solving task used in Experiment 1 and hence was familiar to all subjects, and the other was a harder, but otherwise equivalent task.

As in Experiment 1, IBI was recorded concurrently with PTT. In addition, measures of systolic (SBP) and diastolic (DBP) blood pressures were recorded.

METHOD

Subjects

Twenty undergraduate psychology students were invited to participate in Experiment 2 on the basis of their PTT response direction to the mental arithmetic and problem solving tasks in Experiment 1. Thirteen of the subjects (9 women and 4 men) had accelerated to both tasks; 7 of the subjects (3 women and 4 men) had decelerated to both tasks. However, data from one of the women accelerators was rejected due to movement artefact, leaving an N of 19, 11 of whom were women: 8 accelerators and 3 decelerators.

Apparatus and Physiological Recording

As in Experiment 1, with the additional measurement of blood pressure as detailed below.

Systolic (SBP) and diastolic (DBP) blood pressures were measured using a digital sphygmomanometer (Nissei DS-103). Readings were taken from the control room without the experimenter entering the experimental room. Blood pressure was recorded during the 1st, 5th and 10th minutes of the initial baseline period. A pre-task baseline level was recorded, then a reading taken during the pre-task instruction period prior to each task and also during each of the 2nd and 4th minutes of the task and post-task periods for both tasks.

PROCEDURE

The procedure was essentially identical to that used in Experiment 1, with the following changes.

Subjects each took part in only one session at which two problem solving tasks were completed. As well as the beat-by-beat recording of PTT and IBI, SBP and DBP responses were recorded at the time-points described above.

Two problem solving tasks were used. Both were based on the Nufferno Speed Tests (Furneaux, 1956) and used letter sequences which required the subject to provide the next two letters of the sequence. Presentation of the tasks, and the scoring of the behavioural responses were identical to those used in Experiment 1.

All subjects had previously been exposed to the easier of the two tasks. The harder task involved items which were judged by the experimenter to be more difficult than those in the easier task. Following the completion of the experiment, the relative difficulty of the tasks was evaluated by comparing the performance on each task. It was found that during the easier task more problems were solved correctly (49% versus 37%, p<.0001), and fewer errors of omission (41% versus 53%, p<.0001) were made. There was no difference in the number of errors of commission across tasks (10% of the total number of problems presented in each task).

RESULTS

The median value for PTT and IBI over every minute of the experimental session was obtained. SBP and DBP were recorded three times during the initial baseline, once during each pre-task baseline, once during each pre-task instruction period and twice each during every task and post-task period. These data formed the bases of all the reported analyses. However, during the easier task, blood pressure data from 2 subjects were lost and during the harder task blood pressure data from 3 subjects were lost due to equipment malfunction or movement artefact.

Analyses of Absolute Levels

Absolute levels of cardiovascular activity during each experimental condition were analysed in a manner consistent with that described in Experiment 1. Analyses of absolute levels of cardiovascular activity during the instruction, task and post-task periods are presented in Appendix 3.

Initial Baseline

The absolute levels of PTT and IBI recorded during the initial baseline period were analysed using a Sex (2) X Minute (10) ANOVA with repeated measures on the last factor. No significant effects were found.

The three measures of SBP and DBP taken during the initial baseline were analysed using a simple ANOVA with a factor of Sex (2). No significant effects were found.

Pre-task Baseline Periods

Absolute levels of IBI, PTT, SBP and DBP during pretask baseline were analysed using a Sex (2) X Task (2) ANOVA with repeated measures. No significant effects were found for any response.

Analyses of Response Change

Response change from pre-task baseline was calculated for IBI and PTT during each pre-task instruction period, and for each minute of all task and post-task periods. IBI and PTT response changes for men and women over each condition are shown in Figure B, Appendix 1. Response change from pre-task baseline was calculated for each of SBP and DBP during each pre-task instruction period and for both of the readings taken for each measure during every task and post-task period. SBP and DBP response changes for men and women over each condition are shown in Figure C, Appendix 1. Post-task recovery data are presented in Appendix 4.

The analyses to be described were performed on the actual change scores in each case, but scores were converted to mean percentage change from pre-task baseline for use in figures and tables.

Instruction Periods

The change scores during the instruction periods were analysed using a Sex (2) X Task (2) ANOVA with repeated measures on the last factor. A secondary analysis which substituted a factor of Task Order for Task was also performed.

As in Experiment 1, instruction generated changes in IBI were sensitive to the imminent task (Task Effect, $F(1/17)=25.07$, $p<.007$) with larger magnitude changes occurring in anticipation of the harder task (-12ms vs -63ms).

A Main Effect of Sex ($F(1/17)=4.70$, $p<.04$) was found for the instruction generated changes in IBI. Women had larger acceleratory changes than men (-55ms vs -14ms).

A Main Effect of Sex ($F(1/17)=18.19$, $P<.0006$) was also found for the instruction generated changes in DBP. Women had no change in DBP, the men had a decrease (+0.26mmHg vs -6.4mmHg).

The differences between the sexes are shown in Table 2.1.

There were no other significant effects or interactions.

Task Periods

The IBI and PTT change scores during task periods were analysed using a Sex (2) X Task (2) X Minute (4) repeated measures ANOVA. The SBP and DBP change scores during task periods were analysed using a Sex (2) X Task (2) repeated measures ANOVA. Secondary analyses which substituted Task Order for Task were also performed.

There was a Main Effect of Minute $(F(3/551)=5.37$, $p<.003)$ for task-generated change in IBI. Follow-up means tests showed that the response peaked on minute 1 of the task period and then stabilized from minute 2 over the remaining minutes.

Changes in the first measure of SBP recorded during each task were also sensitive to the differences in task demands, (Task Effects, $F(1/13)=7.05$, $p<.02$) with larger increases during the more difficult task.

There were no other significant effects or interactions.

Behavioural Task Performance

The behavioural response made to each problem solving item by each subject was scored as either correct, an error of omission or an error of commission. The overall score for each of the three measures was then converted to a percentage of the total number of items presented in that task for that subject.

TABLE 2.1

The Sex Differences (p<0.02) found for instruction-generated changes in Interbeat interval (IBI) and diastolic blood pressure (DBP). All changes shown as percentage changes from pre-task baseline levels.

	Men (n=11)	Women (n= 8)
Response	% Change	% Change
IBI	-1.7	-6.7
DBP	-9.1	+0.4

Relationship Between Performance & Cardiovascular Responses

Linear regression analyses were used to test the relationships between magnitude of change in each cardiovascular response during a task and behavioural performance on that task. A larger change in PTT during the easier task was associated with more errors of omission $(F(1/17)=4.916, p<.04)$ and fewer problems solved correctly $(F(1/17)=4.734, p<.04)$.

During the harder task, no relationship was found between PTT change and task performance, nor were there any other significant relationships between task-generated cardiovascular response change and behavioural task performance.

Relationship Between Task Performance and Sex

Behavioural measures of task performance were initially analysed using a simple ANOVA with a factor of Sex (2). Women were found to make more correct responses to both the easier (Sex Effect, $F(1/17)=6.7, p<.02$) and the harder tasks (Sex Effect, $F(1/17)=8.06, p<.01$) and make fewer errors of omission than the men (Sex Effect, easier task, $F(1/17)=6.91, p<.02$; harder task, $F(1/17)=5.51, p<.03$). The percentages of problems answered correctly and errors of omission for the women and the men are presented in Table 2.2 along with their mean scores from Experiment 1, and those of all the subjects in Experiment 1.

TABLE 2.2

The Sex Differences in task performance found in Experiment 2. Also shown are the task performance scores from Experiment 1 for the subgroup who participated in Experiment 2, and for all subjects in Experiment 1.

EXPERIMENT 2			
Problems	Men (n=11)		Women(n=8)
% Correct	39	*	57
% Omitted	53	*	33
% Wrong	8		10

EXPERIMENT 1 (SUBGROUP)		
Problems	Men (n=11)	Women (n=8)
% Correct	26	48
% Omitted	66	44
% Wrong	8	8

EXPERIMENT 1 (ALL SUBJECTS)		
Problems	Men (n=19)	Women (n=31)
% Correct	35	40
% Omitted	56	52
% Wrong	9	8

* p<.05 or better

Direction of Response Changes

As in Experiment 1, subjects were firstly classified as accelerators and decelerators on each task and for each cardiovascular response separately, and then secondly, the pattern of changes in the other concurrently measured responses was considered. The method of classifying subjects as accelerators or decelerators was to find the mean of a task-generated change-score, and then, if the mean was less than pre-task baseline, to classify the subject as an accelerator, but if the mean was greater than pre-task baseline, to classify the subject as a decelerator. That method of classifying each subject was firstly applied to IBI for each of the two tasks, see Table 2.3; secondly to PTT, see Table 2.4; thirdly to SBP, see Table 2.5; and fourthly to DBP, see Table 2.6.

Each table also presents details of the concurrently measured other cardiovascular change scores, and the mean change scores for all the groupings thus formed were tested for the signficance of their deviation from baseline using t-tests for paired means comparisons. The signficance of each response is indicated in the tables. The data from these groupings were also tested for differences between accelerators and decelerators in sex, pre-task baseline levels of cardiovascular activity and in task performance measures. No differences were found.

The response changes within tasks for each of the groupings are shown in

Table 2.3

Interbeat Interval (IBI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup. The concurrently measured Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds. Systolic (SBP) and Diastolic (DBP) Blood Pressure changes are shown in mmHg. The significance of deviation from pre-task baseline for each change is indicated.

		Acceleratory		Concurrently Measured	
Task	N	IBI	PTT	SBP	DBP
Easy	18	-49*	-4.5	-3.2	-1.9
Hard	16	-68*	-0.9	-8.2	-2.8

		Deceleratory		Concurrently Measured	
Task	N	IBI	PTT	SBP	DBP
Easy	1	+14	+0.7	+3.5	-8.5
Hard	3	+35	+3.0	-3.9	-2.5

* p<.05 or better
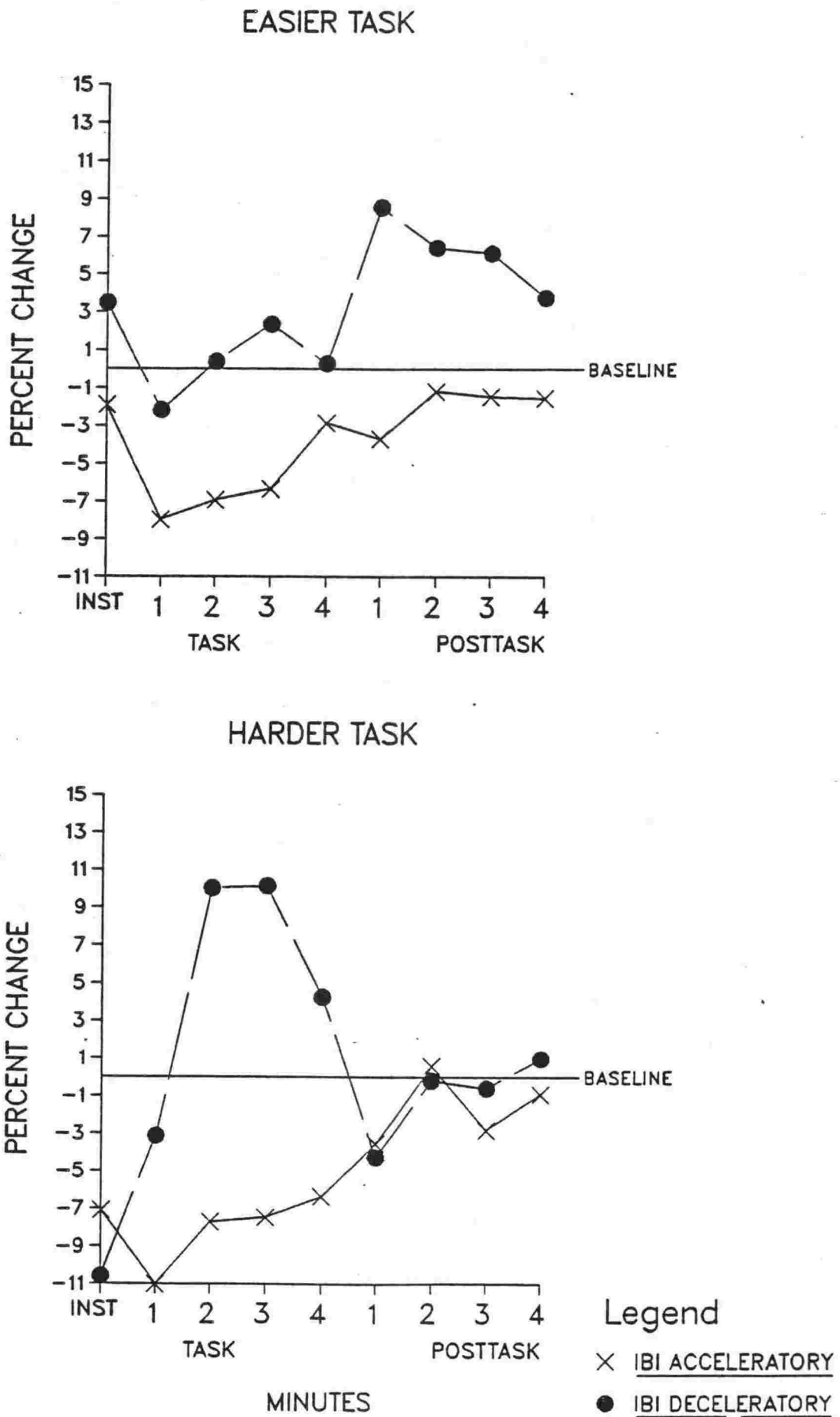
Figure 2.1 Minute—by—minute Interbeat Interval acceleratory and deceleratory responses, Experiment 2.

Table 2.4

Arterial Pulse Transit Time (PTT) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup. The concurrently measured Interbeat Interval (IBI) changes are also shown in milliseconds. Systolic (SBP) and Diastolic (DBP) changes are shown in mmHg. The significance of deviation from pre-task baseline for each change is indicated.

		Acceleratory		Concurrently Measured	
Task	N	PTT	IBI	SBP	DBP
Easy	9	-5.3*	-44*	-6	-4.5
Hard	10	-6.0*	-55*	-9.2	-5.2
		Deceleratory		Concurrently Measured	
Task	N	PTT	IBI	SBP	DBP
Easy	10	+12.9	-48*	- 0.2	-0.1
Hard	9	+ 6.2*	-47	-17.3	-8.0

*   p<.05 or better
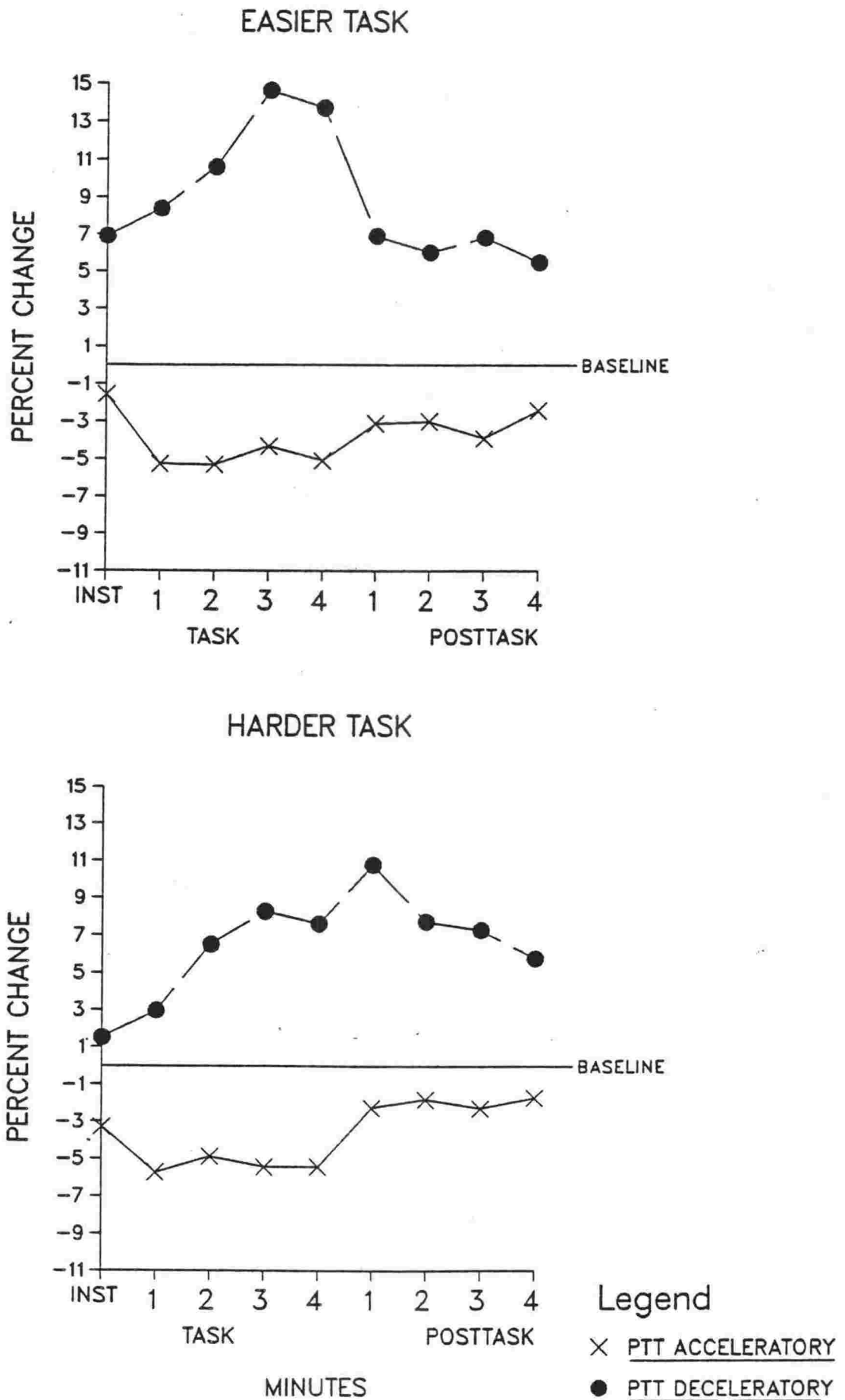
Figure 2.2 Minute—by—minute Arterial Pulse Transit Time acceleratory and deceleratory responses, Experiment 2.

Table 2.5

Systolic Blood Pressure (SBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup. The concurrently measured Diastolic Blood Pressure (DBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are shown in milliseconds. The significance of deviation from pre-task baseline for each change is indicated.

Task	N	Acceleratory SBP	IBI	Concurrently Measured PTT	DBP
Easy	9	+4.1*	-43*	+8.4	+0.7
Hard	10	+5.1*	-65*	+1.4	+4:3*

Task	N	Deceleratory SBP	IBI	Concurrently Measured PTT	DBP
Easy	10	-5.7	-29*	-0.3	-4.4
Hard	9	-2.5	-38	-0.9	+1.2

* p<.05 or better
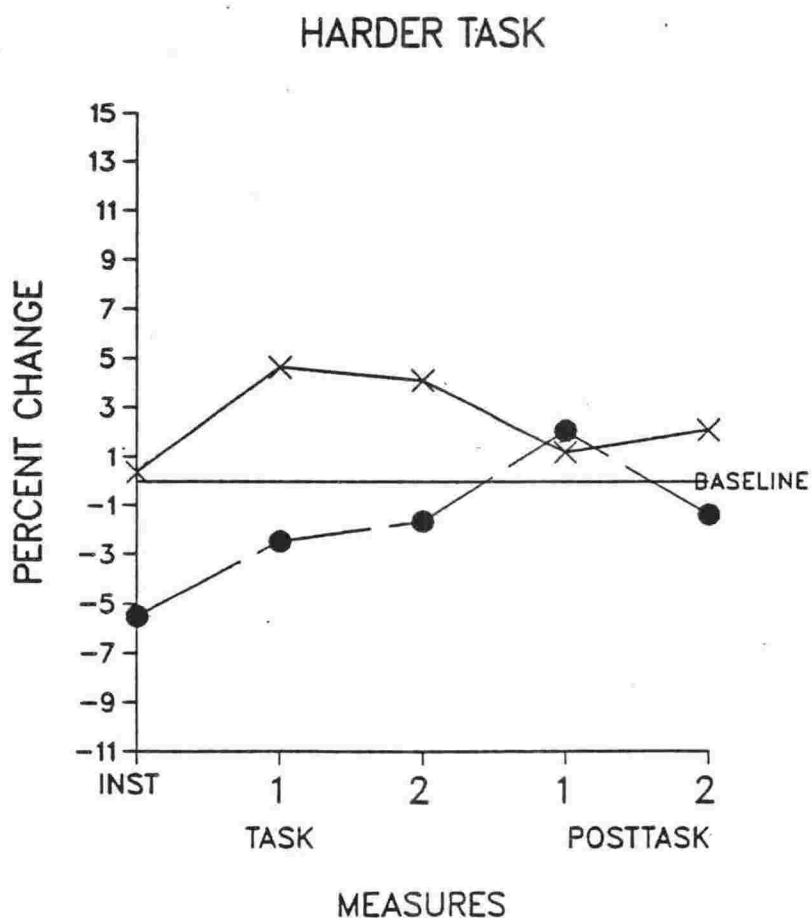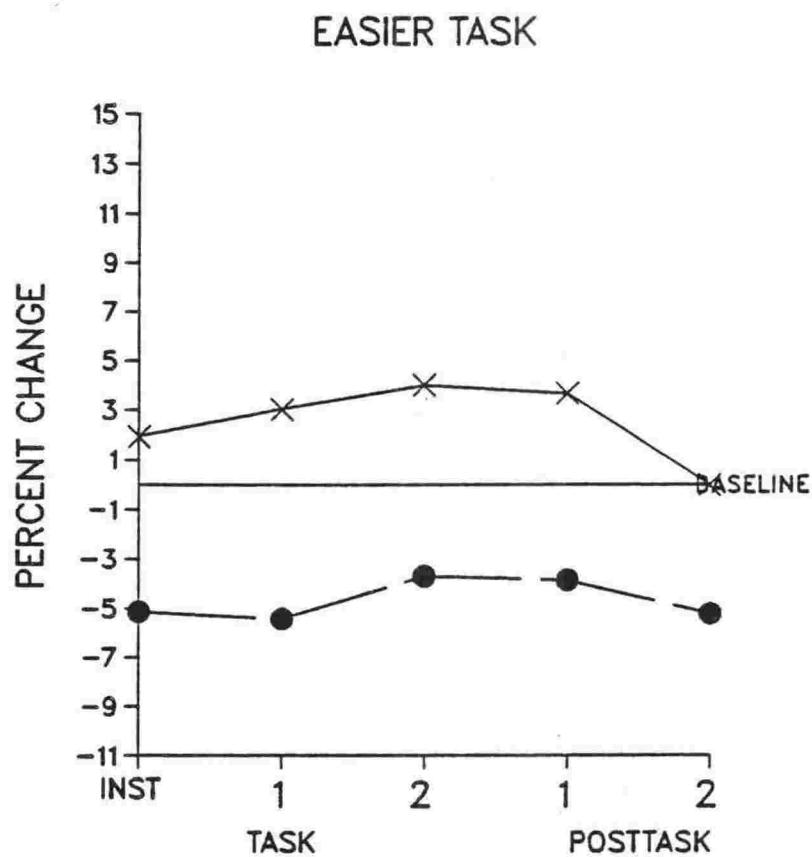
Figure 2.3 Repeated recording of Systolic Blood Pressure acceleratory and deceleratory responses, Experiment 2.

Table 2.6

Diastolic Blood Pressure (DBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup. The concurrently measured Systolic Blood Pressure (SBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are shown in milliseconds. The significance of deviation from pre-task baseline for each change is indicated.

		Acceleratory		Concurrently Measured		
Task	N	DBP	IBI	PTT	SBP
Easy	9	+4.5*	-51*	+11.6	+1.6
Hard	10	+7.3*	-76*	+ 1.2	+4.4*

		Deceleratory		Concurrently Measured		
Task	N	DBP	IBI	PTT	SBP
Easy	10	-5.4	-40*	+2.0	-1.6
Hard	9	-3.4	-25	+0.4	+2.8

* p<.05 or better
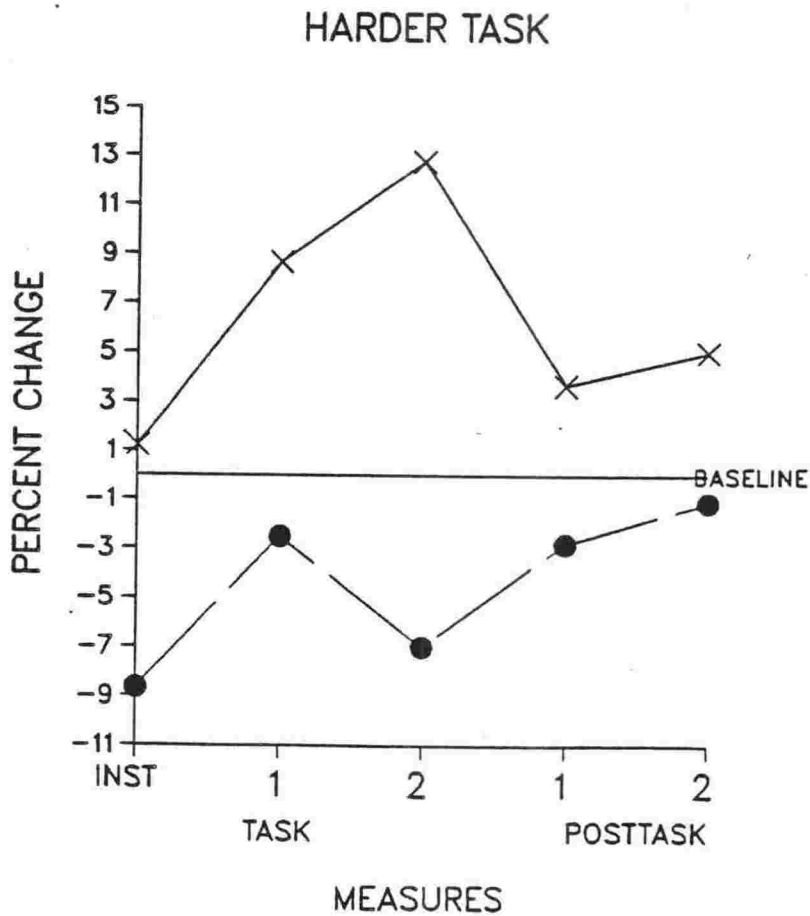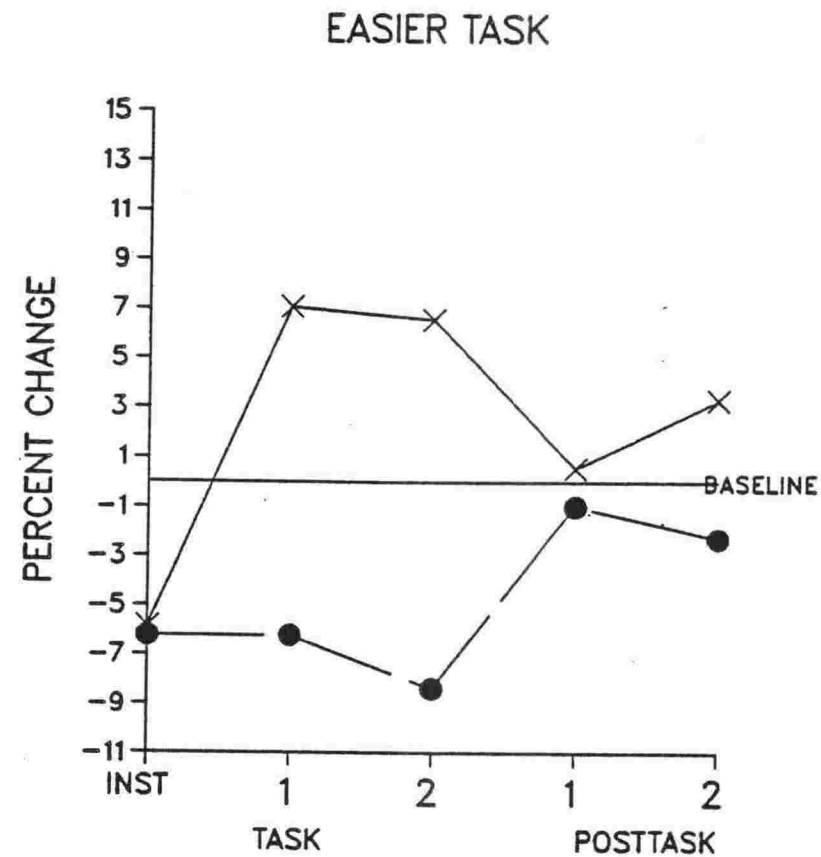
Figure 2.4 Repeated recording of Diastolic Blood Pressure acceleratory and deceleratory responses, Experiment 2.

Individual Response Stereotypy

Inspection of the data revealed that 14 subjects had PTT responses which were consistent in direction across both tasks in Experiment 2. Subjects were then categorized as either accelerators or decelerators on the basis of their PTT response direction during the problem solving task in Experiment 1 and assigned to a group accordingly. During Experiment 2, only 6 subjects made PTT responses in a direction consistent with that made during the problem solving task in Experiment 1. Both absolute levels of cardiovascular activity and response change from pre-task baseline were analysed using a Group (2) X Sex (2) ANOVA for unbalanced data in a test for differences between previous accelerators and decelerators. No differences were found.

DISCUSSION

The results from Experiment 2 confirm the findings from Experiment 1 which identify PTT as characterized by unusual directional variability. Deceleratory PTT changes were observed during conditions of greatest stress. In Experiment 1, deceleratory changes dominated responding during the first task on the first session, which is probably the most stressful laboratory exposure and task (Obrist, 1981); in Experiment 2, the deceleratory PTT response was significant during the harder task but not during the easier task. Thus the results suggest that both acceleratory and deceleratory PTT changes are generated by the same tasks, and are equivalent responses. The acceleratory changes can be described as having a shorter latency, smaller variance and more rapid decline than the deceleratory change.

However, all attempts to identify the determinants of the direction of PTT change were unsuccessful. Accelerators and decelerators did not differ in baseline levels of PTT, in sex or in task performance. In addition, each subject's response direction during the tasks in Experiment 2 was not predicted by his/her response direction to the problem solving task in Experiment 1. Thus further investigation into the determinants of the direction of PTT change was indicated.

Women had larger acceleratory IBI changes than men, and also had superior task performance although the peformance data from Experiment 1 suggest that the selection of subjects according to PTT response direction may have inadvertently biased the performance data by selecting men who had relatively poor performance in comparison to the performance of the other men in Experiment 1, and by selecting women who had relatively good performance in comparison to the overall performance by women in Experiment 1.

Notably, no deceleratory IBI changes were significant. However, the magnitude of IBI change was not differentiated by the tasks, although behavioural performance identified one as more difficult than the other and further, as in Experiment 1, the magnitude of IBI change was differentiated by the task instructions with a larger change during the pre-task baseline before the harder task than during the one before the easier task. It could be the case that the second exposure to the laboratory situation may have resulted in non-reactive IBI (Obrist, 1981; Glaser, 1966). If that was the case, then firstly, the application of the active/passive coping concept of stress-related cardiovascular dysfunction would seem inappropriate as such dysfunction is progressive over a relatively long period, and any mediating variable should be

demonstrable over repeated occasions. Secondly, the active/passive hypothesis as currently formulated would be incomplete as the magnitude of cardiac change would not be determined by task difficulty, but by a concatenation of novelty and difficulty. Alternatively, task variety, novelty and difficulty could have been confounded in Experiment 1, with the possiblity that task difficulty could be an insignificant factor in determining the magnitude of cardiac and vascular response changes. The relationship between task difficulty and cardiovascular changes under non-avoidance conditions was further investigated in Experiment 3.

5 EXPERIMENT 3

Individual Differences in Arterial Pulse Transit Time

Changes in Response to Familiar Psychological Events

INTRODUCTION

In Experiments 1 and 2, the directional variability in PTT change had been found not to be associated with baseline level of PTT (Wilder, 1958, 1962) or with subject sex. Nor was the direction of PTT change part of an individual stereotypic response (Malmo & Shagass, 1949; Engel, 1972). However, the possibility that the direction of PTT could be associated with a predisposing psychological variable had not been explored.

The purpose of Experiment 3 was to begin to explore any possible relationship between scores on psychological measurement scales with cardiovascular responses. In particular, it was to determine whether acceleratory and deceleratory PTT change could be lawfully related to psychological measures.

Hare (1972, 1973) has reported that when faced with an aversive stimulus, anxious subjects are more likely to have acceleratory vascular (vasoconstriction) responses than non-anxious subjects who are more likely to have deceleratory vascular (vasodilation) responses. Similarly, Kelly & Martin (1969) found that subjects who were hospitalized as neurotic scored more highly on the neuroticism scale of the Maudsley Personality Inventory and also had

faster resting levels of, but smaller magnitude changes in, forearm blood flow than non-hospitalized normal control subjects had. Further, Teichner (1965) demonstrated that subjects differed in the latency and magnitude of their vasodilation responses to aversive stimuli. He postulated that subjects who had more rapid and larger responses were distinguished from subjects who had slower and smaller responses by their chronic levels of high arousal. In summary, the literature which reports investigations into the relationships between vascular responding and psychological measures has identified subjective levels of anxiety, neuroticism and arousal as being of particular relevance.

In addition, the recent identification of the coronary-prone personality (Jenkins, 1975) has generated a body of research directed toward identifying any distinctive cardiovascular response patterns which can be linked to the Type A behaviour patterns (e.g. Dembroski, et al., 1978; MacDougall, et al., 1981; Manuck & Garland, 1979).

Thus the measures selected for initial investigation in this experiment were the State-Trait Anxiety Inventory (STAI, Spielberger, et al., 1970) comprising the State and the Trait Anxiety Scales, the Eysenck Personality Inventory (EPI, Eysenck, 1976) comprising the Extraversion, Neuroticism and Lie Scales, the Stress-Arousal Checklist (SACL, MacKay, et al., 1975) comprising the Stress and Arousal Scales, and the Jenkins Activity Survey (JAS, Jenkins, Rosenman & Friedman, 1967),

The STAI and the EPI were selected because each has a base in a theory which explicitly proposes that higher levels of autonomic activity underlie higher scores in anxiety (Spielberger, 1966) or neuroticism (Eysenck, 1967),

Zealand subject sample similar to the one participating in the current research (McCormick, 1983) and the JAS was selected because of its direct relevance to cardiovascular functioning.

It should be noted that the interest in these psychological variables in the present research was exclusively in their possible relationship with the direction of PTT change during tasks. Therefore, subjects were not grouped according to scale scores and then tested for differences in cardiovascular response patterns. Instead, it was intended that once the accelerators and decelerators during each task had been identified they would be tested for differences in each scale score. The broader question of whether individuals who differ in their scores on any particular psychological measure also differ in their cardiovascular response patterns and if so, how, was considered to be beyond the scope of the present research.

Secondly, Experiment 2 had confirmed directional variability as a characteristic of the PTT response, but had raised the question of whether the magnitude of IBI change is determined by task difficulty, by a combination of task difficulty and task type, or by a combination of task difficulty, type and novelty. Experiment 3 was also designed to answer that question.

Three problem solving tasks similar to those used in the two previous experiments were developed, one at each of three levels of difficulty: easy, hard and impossible. The personal tempo was also included again as a comparison task with no a priori associated demands.

The cardiovascular responses measured were identical to those measured in Experiment 2.

The cardiovascular responses measured were identical to those measured in Experiment 2.

METHOD

Subjects

Subjects who had participated in Experiment 1 were invited to return. All those who had participated in Experiment 2, and 11 additional individuals volunteered. Of the 30 subjects, 14 were women.

Apparatus and Physiological Recording

The apparatus and physiological measurements were identical to those used in Experiment 2 with the exception that blood pressures were only measured during minutes 1 and 4 of the initial baseline, and once (on minute 2) of each task and post-task recovery period. As in Experiment 2, blood pressures were recorded once during each pre-task baseline and instruction period.

PROCEDURE

The procedure was essentially identical to that used in Experiment 2 except for the following changes. Firstly, as the initial baseline data in Experiment 2 had shown no change over the 10-minute period, the initial baseline in the current experiment was reduced to 5 minutes. Secondly, as the task response had peaked by minute 2 in both experiments 1 and 2, task duration was reduced to 3 minutes in the current experiment, and the post-task recovery period was correspondingly shortened.

Three problem solving tasks and one personal tempo task were used. All the problem solving tasks were based on the Nufferno Speed and Power Tests (Furneaux, 1956) and used letter sequences which required the subject to provide a single letter to continue or complete the sequence. Subjects completed all four tasks in a single session which began with a 5-minute initial baseline and was followed by 4 cycles of 1-minute pre-task baseline, 1-minute instruction, 3-minute task and 3-minute post-task recovery period. Task order was counterbalanced across the subjects. Presentation of the tasks and scoring of the behavioural responses were identical to those described in Experiment 1.

The problem solving tasks were graded as either easy, hard or impossible either with items taken directly from the Nufferno tests or with similar items constructed by the experimenter and assigned to tasks according to their complexity. Following completion of the experiment, the actual relative difficulty of the tasks was evaluated by paired comparisons of the behavioural performance scores across tasks for the entire subject population. Initially, the easy and hard tasks were compared. During the easy task, more problems were solved correctly (77.6% vs 30.8%, p<.0001), there were fewer errors of omission (19.6% vs 60.78%, p<.0001) and fewer incorrect responses (2.78% vs 8.5%, p<.004) than during the hard task. Then the hard and impossible tasks were compared. During the hard task there were more problems solved correctly (30.8% vs 5.3%, p<.0001) and fewer errors of omission (60.8% vs 82.1%, p<.0001) than during the impossible task.

Analyses of Absolute Levels

Absolute levels of IBI, PTT, SBP and DBP were analysed as in the previous experiments. The absolute levels of cardiovascular activity during instruction, task and post-task periods are presented in Appendix 3.

Initial Baseline

The absolute levels of IBI and PTT during the initial baseline were analysed using a Sex (2) x Minute (5) ANOVA with repeated measures on the last factor. No significant effects were found.

The measures of SBP and DBP taken during the initial baseline were analysed using a simple ANOVA with a factor of Sex (2). Main Effects of Sex were found for both measures of SBP (first measure, $F(1/28)=9.63$, $p<.004$; second measure, $F(1/28)=11.74$, $p<.002$). Women had lower SBP than men (See Table 3.1).

Pre⁻task Baseline Periods

The absolute levels of IBI, PTT, SBP and DBP during the pre-task baseline were analysed using a Sex (2) x Tasks (4) ANOVA with repeated measures on the last factor. A secondary analysis which substituted a factor of Task Order for Task was also performed.

A Main Effect of Task was found for levels of PTT during pre-task baseline ($F(3/84)=4.44$, $p<.0006$). PTT was slower prior to the hard task (103.8ms) than prior to the easy, impossible or personal tempo tasks (99.7ms, 98.3ms, 98.5ms).

A Main Effect of Task was found for levels of PTT during pre-task baseline (F(3/84)=4.44, p<.0006). PTT was slower prior to the hard task (103.8ms) than prior to the easy, impossible or personal tempo tasks (99.7ms, 98.3ms, 98.5ms).

A Main Effect of Sex was found for SBP (F(1/28)=12.04, p<.004). Women had lower SBP than men (see Table 3.1).

Analyses of Response Change

Response change from the immediately preceding pre-task baseline was calculated for every minute median response and for every blood pressure measure during the instruction and task periods. Response changes by men and women during each experimental condition are shown in Figure D (IBI and PTT) and Figure E (SBP and DBP) in Appendix 1. Analyses of the responses during the post-task periods are given in Appendix 4.

Instruction Periods

The IBI, PTT, SBP and DBP change scores during the instruction period were analysed using a Sex (2) x Task (4) ANOVA with repeated measures on the last factor. A secondary analysis which substituted a factor of Task Order for Task was also performed. In addition, because of the Task Effect found for level of PTT during the pre-task baseline, an analysis of covariance, with pre-task baseline level as the covariate was performed on the PTT data.

A Main Effect of Sex (F(1/28)=4.29, p<.05) was found for instruction-generated changes in PTT. The men had an acceleratory change (-0.562ms) and the women had a deceleratory change (+1.723ms). No other effects or interactions were significant.

Task Periods

The IBI and PTT change scores during task periods were analysed using a Sex (2) x Task (4) x Minute (3) ANOVA with repeated measures on the last two factors. Secondary analyses which substituted Task Order for Task were also performed. In addition, because of the Task Effect found for level of PTT during the pre-task baseline, an analysis of covariance, with pre-task baseline level as the covariate was performed on the PTT data.

There was a Main Effect of Tasks ($F(3/84)=9.58$, $p<.0001$) for task-generated change in IBI. Follow-up REGWF means test showed that magnitude of acceleratory change during tasks was determined by task difficulty in the following descending order: hard (-70ms), easy (-50ms), impossible (-39ms) and personal tempo (-8ms). The effect is shown as percentage change in IBI as a function of task difficulty in Figure 3.1.

There was also a Task x Minute Interaction ($F(6/168)=2.79$, $p<.01$) found for task-generated changes in IBI. In particular, acceleratory changes during the easy and hard tasks were sustained, but those during the impossible task were not. Changes during the personal tempo task were progressive over minutes. That interaction is shown in Figure 3.2.

There were no significant effects or interactions for task-generated changes in PTT.

The SBP and DBP change scores during task periods were analysed using a Sex (2) x Task (4) ANOVA with repeated measures on the last factor. Secondary analyses which substituted Task Order for Task were also performed. No significant effects or interactions were found.

Behavioural Task Performance

The behavioural response made to each problem solving item by each subject was scored as either correct, an error of omission (failed to answer) or an error of commission (answered incorrectly). The overall score for each of the three measures for each task was then converted to a percentage of the total number of items presented in that task for that subject. These data were used in the analyses.

Performance on the personal tempo task was recorded as both the number of button presses and the mean latency between responses for each subject.

Relationship Between Performance & Cardiovascular Responses

A preliminary Pearson Product Moment correlation analysis showed no significant relationship on any of the four tasks between the measures of behavioural performance and their associated cardiovascular responses. Therefore no multivariate linear regression analyses were performed.

TABLE 3.1

Systolic (SBP) and Diastolic (DBP) Blood Pressure levels in mmHg for men and women during the initial baseline and pre-task baseline. A Main Effect of Sex was found for all SBP comparisons.

		Men (n=16)		Women (n=14)
Initial Baseline 1	SBP	121.75	*	110.43
	DBP	68.75		74.14
Initial Baseline 2	SBP	121.25	*	109.29
	DBP	68.50	*	71.43
Pretask Baseline	SBP	120.25	*	107.98
	DBP	67.70		69.80

* $p < .05$ or better

MEAN TASK-GENERATED IBI CHANGE
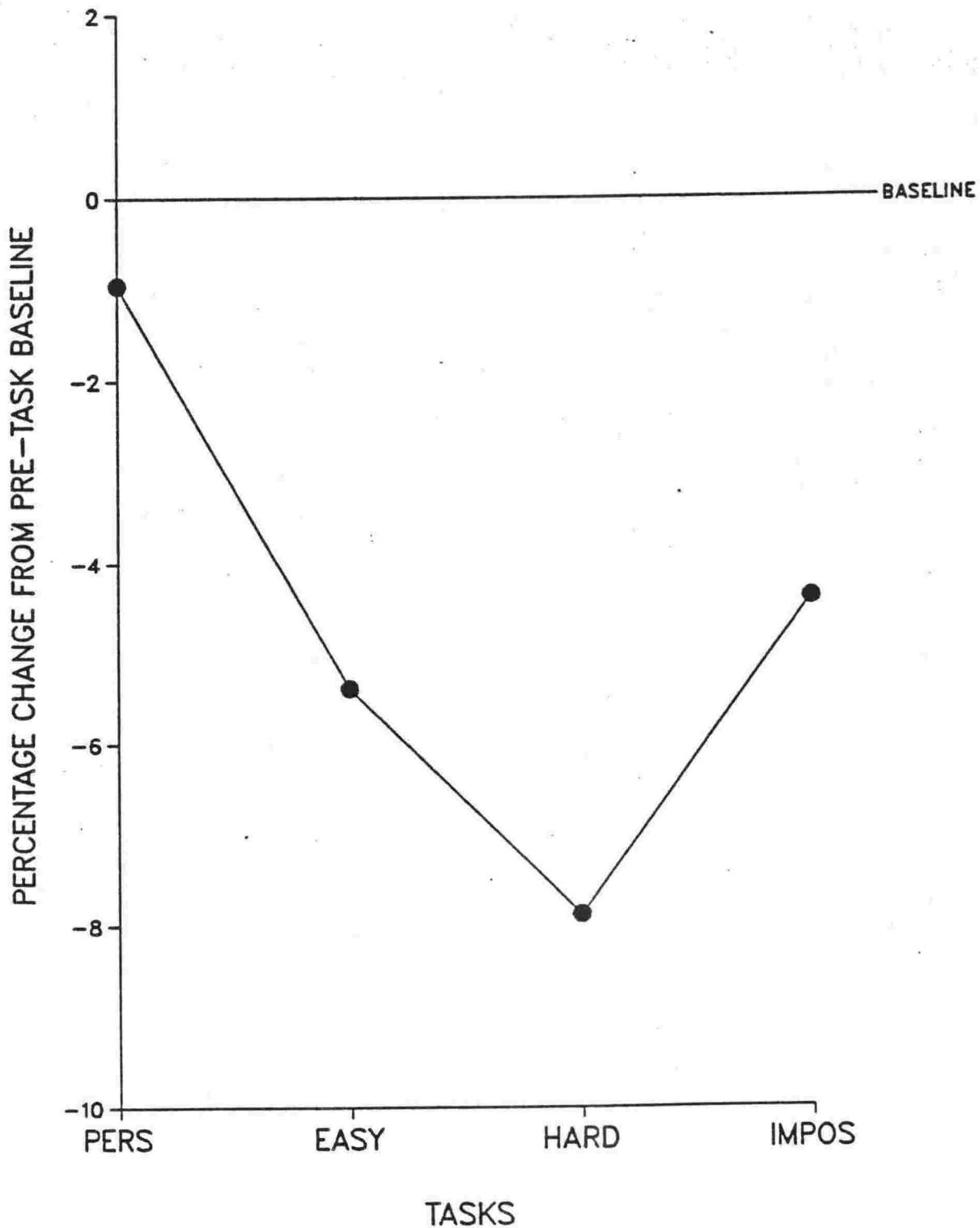
Figure 3.1 Percent change in Interbeat Interval during tasks
(Pers=personal tempo; Easy=easy problems;
Hard=hard problems; Impos=impossible problems.)
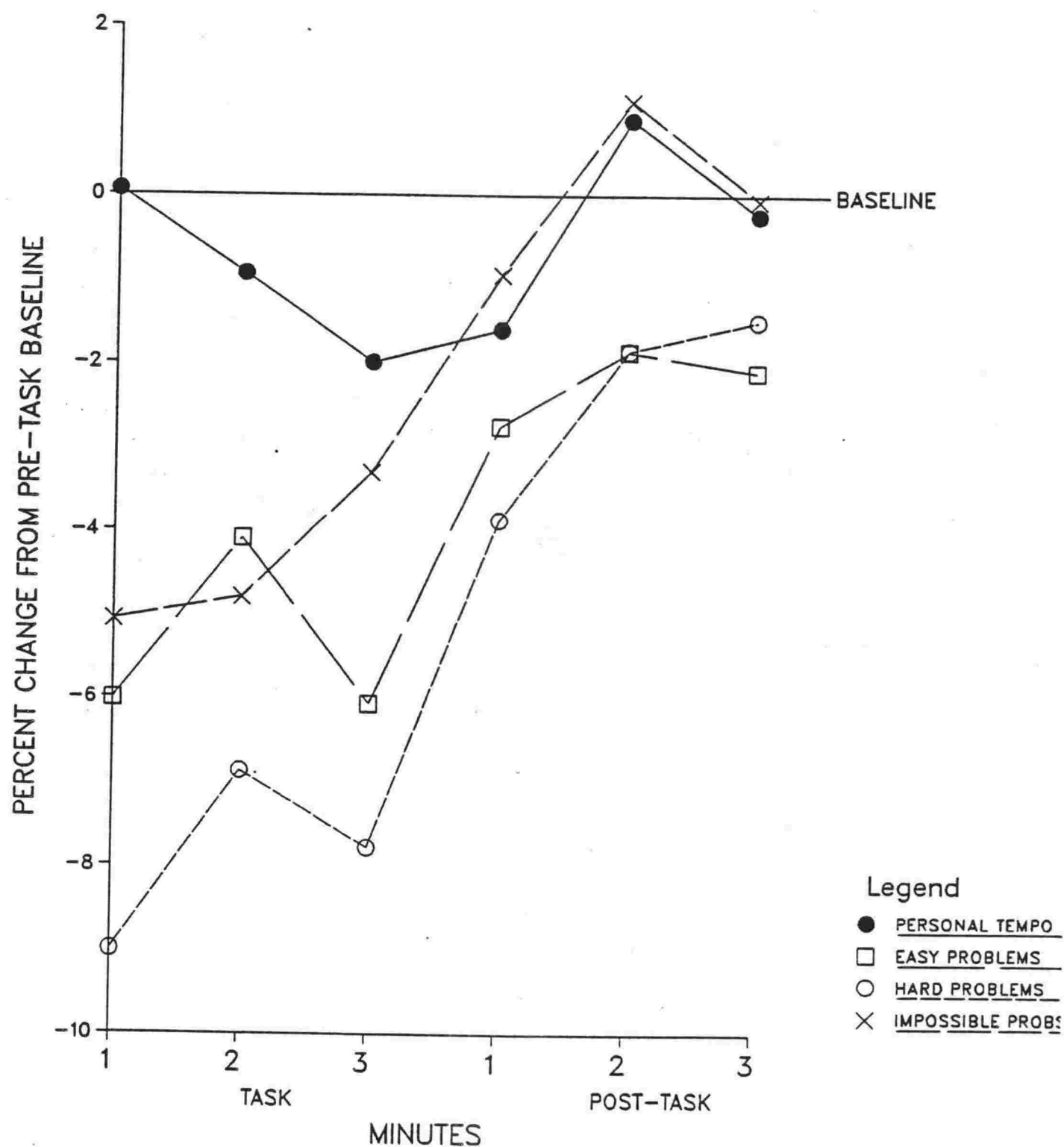
Figure 3.2 The Interbeat Interval Response showing the Task X Minutes Interaction during task.

TABLE 3.2

Behavioural task performance for men and women. Problem solving performance is shown as percentage of total problems presented. Personal tempo button presses are shown as mean number, and latency between presses is given in milliseconds.

		Men (n=16)		Women (n=14)
Easy Task	Correct	75.75		79.70
	Omitted	21.76		17.21
	Wrong	2.49		3.09
Hard Task	Correct	20.17	*	42.92
	Omitted	71.95	*	48.02
	Wrong	7.88		9.06
Imp. Task	Correct	4.03		6.81
	Omitted	87.74		75.73
	Wrong	8.22		17.99
Pers Temp	Presses	11.70	*	18.30
	Latency	37.02		16.03

* $p < .05$ or better

Relationship Between Task Performance and Sex

Behavioural measures of task performance were analysed using a simple ANOVA with a factor of Sex (2). During the hard task, women were found to make more correct responses (Sex Effect, $F(1/28)=10.76$, $p<.003$) and fewer errors of omission ($F(1/28)=7.85$, $p<.009$). There was no difference between the men and the women in performance on either the easy or the impossible tasks. But women made more button presses than men during the personal tempo task ($F(1/28)=4.22$, $p<.05$). The performance data for men and women are shown in Table 3.2.

Direction of Response Change

As previously described in Experiments 1 and 2, subjects were re-classified as accelerators or decelerators for each response on each task. The categorizations based on the IBI response are shown in Table 3.3, those based on the PTT responses are shown in Table 3.4, those based on the SBP responses are shown in Table 3.5, and those based on the DBP responses are shown in Table 3.6. The minute-by-minute IBI responses are shown in Figure 3.3; those for PTT are shown in Figure 3.4. As only one measure of SBP and DBP were recorded during task periods, those responses are not graphed.

The data from the groupings thus formed were also tested for differences between accelerators and decelerators in sex, baseline levels, task performance and also for differences in their scores on each of the psychological measures used. No differences were found.

Table 3.3

Interbeat Interval (IBI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup. The concurrently measured Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds, and the concurrently measured Systolic (IBI) and Diastolic (DBP) Blood Pressure changes are shown in mmHg. The significance of deviation from pre-task baseline for each change is indicated.

(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory IBI	Concurrently Measured PTT	SBP	DBP
Easy	25	-66*	-1.6*	+2.04*	+1.96
Hard	26	-83*	-1.92	+1.4	+2.85*
Impos	22	-65*	-0.80	+0.73	+2.45*
Pers	16	-33*	-0.35	+0.81	+1.19

Task	N	Deceleratory PTT	Concurrently Measured IBI	SBP	DBP
Easy	5	+27	+4.40	+0.2	-1.2
Hard	4	+17	+1.4	+2.25	-1.25
Impos	8	+33	+3.25	-3.38	+5.5
Pers	14	+19*	+0.90	-2.29	-0.50

* p<.05 or better
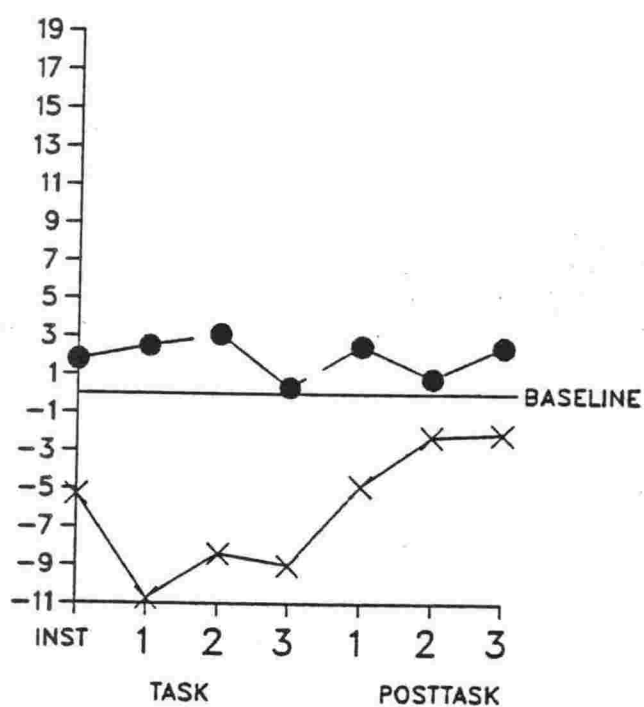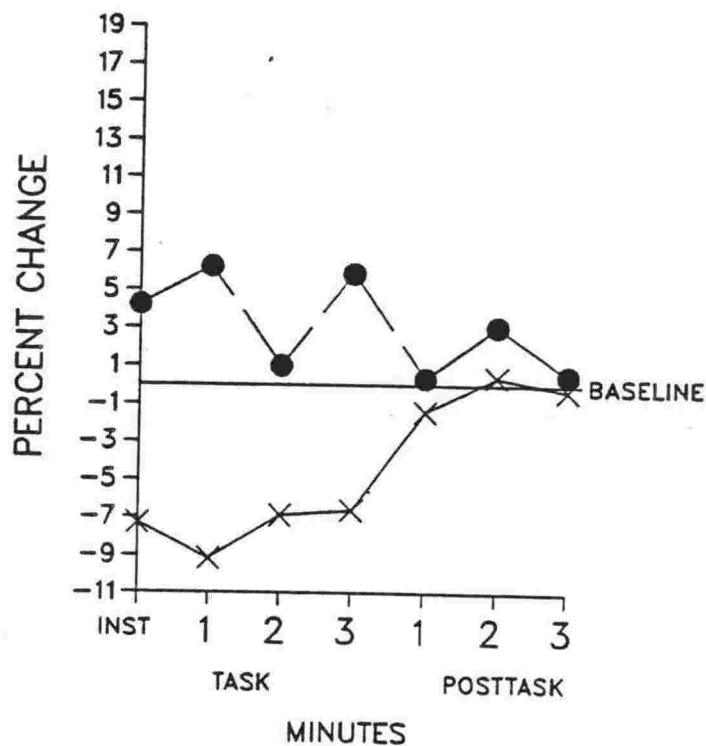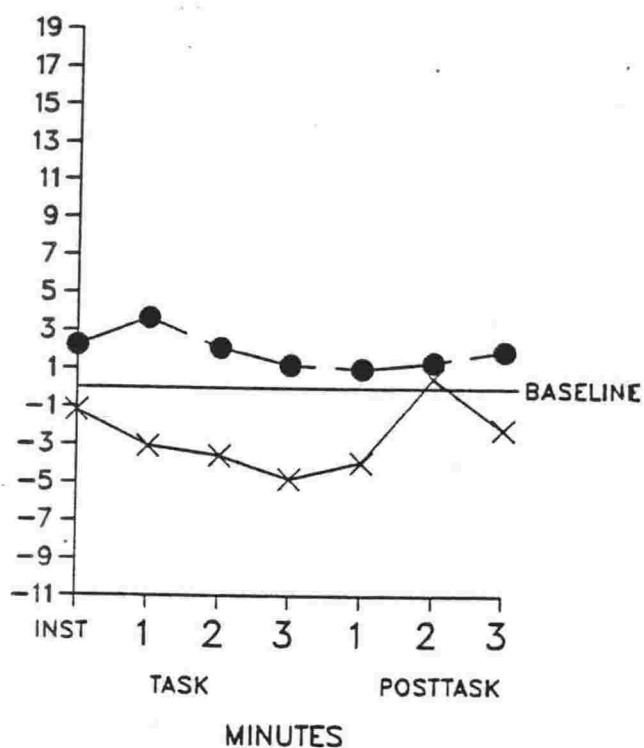
Figure 3.3 Minute—by—minute Interbeat Interval acceleratory and deceleratory responses, Experiment 3.

Table 3.4

Arterial Pulse Transit Time (PTT) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup. The concurrently measured Interbeat Interval (IBI) changes are also shown in milliseconds, and the concurrently measured Systolic (IBI) and Diastolic (DBP) Blood Pressure changes are shown in mmHg. The significance of deviation from pre-task baseline for each change is indicated.

(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory PTT	Concurrently Measured IBI	SBP	DBP
Easy	19	-3.11*	-52*	+0.84	+3.79*
Hard	18	-4.98*	-86*	+2.44*	+3.83*
Impos	11	-4.18*	-39	+1.09	+2.57
Pers	14	-2.50*	+ 3	-1.50	+1.57

Task	N	Deceleratory PTT	Concurrently Measured IBI	SBP	DBP
Easy	11	+3.68*	-47	+3.27	-2.63
Hard	12	+3.79*	-47*	+0.17	+0.05
Impos	19	+3.70*	-39*	-1.21	+2.11*
Pers	16	+3.11*	-13	+0.13	-0.63

* p<.05 or better
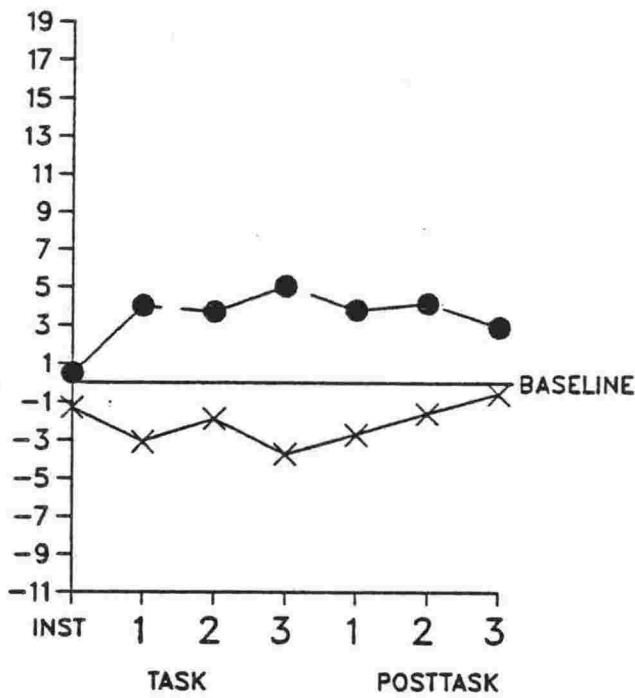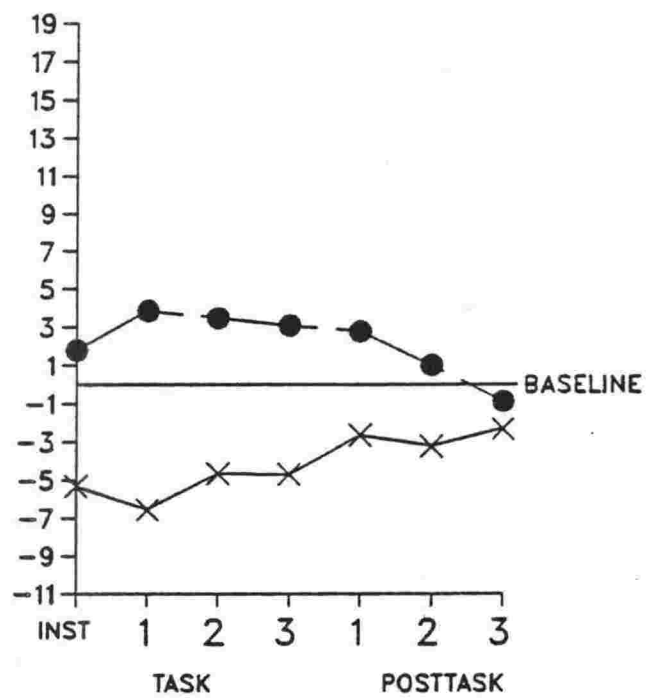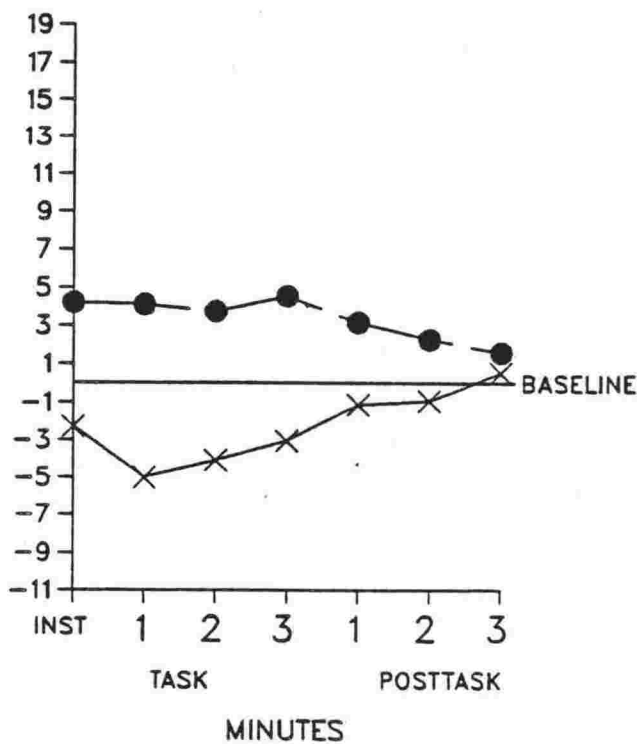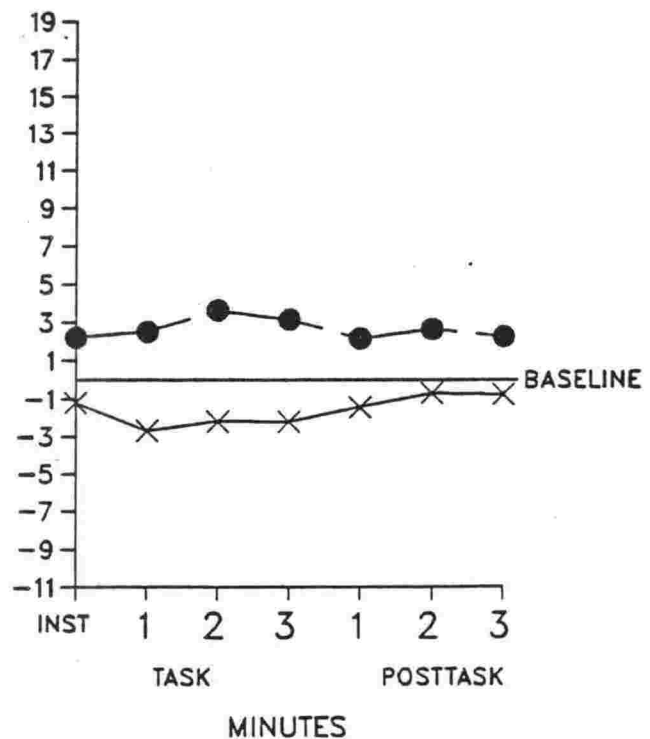
Figure 3.4 Minute—by—minute Arterial Pulse Transit Time acceleratory and deceleratory responses, Experiment 3.

Table 3.5

Systolic Blood Pressure (SBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup. The concurrently measured Diastolic Blood Pressure (DBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are shown in milliseconds. The significance of deviation from pre-task baseline for each change is indicated.

(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory SBP	IBI	Concurrently Measured PTT	DBP
Easy	18	+5.17*	-66*	-0.12	+2.83
Hard	19	+4.53*	-67*	-2.66	+3.68*
Impos	12	+5.25*	-51*	-0.27	+4.33*
Pers	12	+5.17*	-22	-0.57	+1.25

Task	N	Deceleratory SBP	IBI	Concurrently Measured PTT	DBP
Easy	10	-4.10*	- 1	-0.12	-0.60
Hard	10	-4.00*	-76*	+1.20	-0.10
Impos	16	-4.63*	-19	+1.66	+3.38
Pers	17	-4.76*	+ 2	+1.10	-0.12

* p<.05 or better

Table 3.6

Diastolic Blood Pressure (DBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup. The concurrently measured Systolic Blood Pressure (SBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are shown in milliseconds. The significance of deviation from pre-task baseline for each change is indicated.
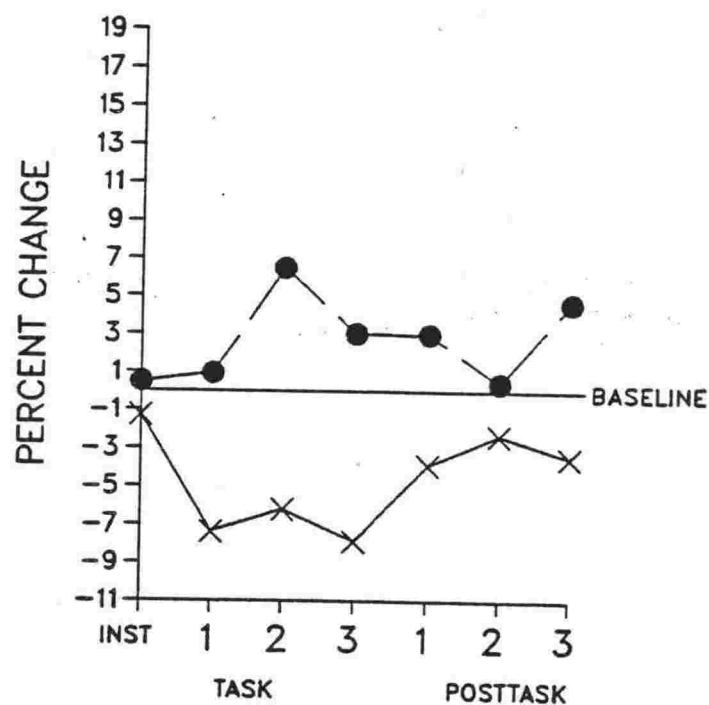
(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory DBP	Concurrently Measured IBI	PTT	SBP
Easy	16	+6.31*	-43*	-2.71*	+1.81
Hard	18	+6.06*	-89*	-3.48*	+3.17*
Impos	18	+6.67*	-29	+0.88	+1.56
Pers	15	+5.07*	-16	-0.23	+1.27

Task	N	Deceleratory SBP	Concurrently Measured IBI	PTT	DBP
Easy	11	-5.27*	-39	+1.82	+1.27
Hard	9	-4.44*	-45*	+2.25	-0.67
Impos	8	-2.75*	-76*	+1.02	-3.88
Pers	14	-4.57*	- 2	+1.11	-2.50

* p<.05 or better

DISCUSSION

Once again directional variability was the dominant characteristic of the PTT changes during task. The attempt to account for the direction of PTT change in terms of scores on the psychological scales was unsuccessful. Accelerators and decelerators did not differ in their scores on any of the psychological scales used. As noted in the introduction, the scores from the psychological scales were used in a highly specific attempt to account for the directional variability of the PTT change. The broader question of whether, and how, individuals who differ in a particular psychological attribute also differ in their cardiovascular response patterns was not addressed in the present experiment, but could provide a focus for future research.

Task difficulty had no demonstrable effect on the PTT changes, and again all acceleratory and deceleratory changes during tasks were significant deviations from pretask baseline level. Accelerators and decelerators on each task did not differ in their task performance, or in their pre-task baseline levels of activity and the direction of PTT change during task was not related to subject sex, although change during instructions was. Thus, at the end of Experiment 3, the determinants of the direction of PTT change remain to be identified.

However, the results from Experiment 3 confirmed that the magnitude of IBI change is determined primarily by task difficulty. The largest magnitude accelerations occurred during the hard task, with progressively smaller accelerations during the easy task, the impossible task and the personal tempo task.

In Experiment 2, the magnitude of acceleratory IBI change was not determined by task difficulty, specifically by tasks associated with 49% success and 37% success. In the present experiment, the magnitude of acceleratory IBI change was determined by task difficulty, specifically by tasks associated with 100% success, 76% success, 20% success and 5% success. It seems likely that there may have been an insufficient difference between the difficulty levels of the tasks used in Experiment 2 to produce a difference in IBI change.

Consistent with Light & Obrist (1983), IBI responses during the easy task in the present experiment paralleled those which occurred during the hard task, not those which occurred during the impossible task. That finding suggests that, firstly, the lack of effect for HR in the Light & Obrist (1983) study was likely to have been an artefact of the selected baseline, and secondly, the effect of task contingency on the manifest IBI response remains to be clarified. The study by Light & Obrist (1980a) showed that the avoidance contingency was an important determinant of the manifest IBI changes associated with active and passive coping. The evidence from Experiment 1 in the present study suggests that task type is also important. The present data could be consistent with an interaction between task type and contingencies. It may be that, when avoidance contingencies are not used, similar tasks with either easy or hard demands generate active coping, while impossible tasks and those with a *priori* no demands attached to them generate passive coping. Alternatively, it may be that if problem solving tasks were associated with contingent electric shocks, a hard task would generate active coping while both easy and impossible tasks would generate passive coping. Hence the contributions both of task type and task consequence or contingency, as distinct from task difficulty, to the

generated behavioural state as indicated by the IBI change, remain to be specified.

As with PTT, all acceleratory (increasing) and deceleratory (decreasing) changes by the groupings in both SBP and DBP were significant deviations from baseline. When considered in conjunction with the results from Experiment 2, it seems possible that novelty may increase the frequency of acceleratory blood pressure changes. The prevalence of deceleratory BP changes in Experiment 3 serves to emphasize the importance of documenting the tonic acceleratory and deceleratory cardiovascular responses which occur in the psychophysiological context. Identification of the determinants of response direction for tonic PTT changes in particular, but possibly for other tonic cardiovascular changes in general, emerges from these experiments as a primary focus for investigation.

6 EXPLORATION OF DIRECTIONAL VARIABILITY

The main findings at the end of Experiments 1, 2 and 3 can be summarized as follows: 1) the magnitude of acceleratory IBI changes during tasks is primarily determined by task difficulty; 2) the frequency of acceleratory IBI changes during tasks is primarily determined by task type; 3) the magnitude and frequency of acceleratory blood pressure changes are determined by task difficulty and novelty; 4) women had lower SBP than men (see also Appendix 3), but there were no fundamental sex differences in task-generated cardiovascular changes; 5) unexplained directional variability was the predominant characteristic of task-generated PTT changes.

In particular, PTT directional variability had the following major characteristics: firstly, with one exception (deceleratory change during the easier task in Experiment 2), all acceleratory and deceleratory PTT changes during tasks were significant deviations from pre-task baseline; secondly, comparisons between accelerators and decelerators for differences in pre-task baseline levels, sex, behavioural performance and scores on psychological measures were consistently negative; thirdly, all significant effects involving the PTT response (session, order, task and sex) reflected a difference in the direction of change, or in the frequency of acceleratory versus deceleratory changes, rather than the magnitude of the change; fourthly, over the course of the research to the present point, no subject had made PTT changes in only one direction.

Two alternative physiological mechanisms for the responses were considered. One alternative was that acceleratory and deceleratory PTT changes reflect increases and decreases in sympathetic neural influences on the vasculature respectively, in which case the systemic PTT response must be described as being dominated by changes in the peripheral vascular beds in particular (Berne & Levey, 1977; Guyton, 1981). The second alternative was that both changes reflect increases in sympathetic influences on the vasculature, with acceleratory changes dominated by neurally mediated vasoconstriction and deceleratory changes dominated by humorally mediated vasodilation in some vascular beds, thus allowing for contributing influences from throughout the systemic vasculatures. The first alternative requires that the changes in opposing directions must be fundamentally distinct responses; the second alternative suggests they reflect a difference in the dominant influence, in terms either of the site or the degree of task-generated response. Given the equivalence of the responses in every respect except their direction, and the equivalence of all the associated behavioural and other physiological and psychological measures, the second alternative was considered to be the more appropriate.

DIRECTIONAL VARIABILITY AND BEHAVIOURAL STATE

Identification of the determinants of PTT response direction during tasks emerged from Experiments 1, 2 and 3 as the unresolved issue of most importance. Examination of the possible physiological mechanisms (elaborated in Chapter 10) suggested that the acceleratory and deceleratory changes could reflect differing degrees of responding, rather than fundamentally distinct responses. It was hypothesized that, if that was the case, then the behavioural state of

the subject prior to participating in the task could be contributing to the degree of responsivity during the task, that is, that there was an interaction between the subject's predisposition to respond and task demands in determining manifest response. Extreme examples of such an interaction have been reported as rigid individual response patterns or stereotypy (Malmo, 1959; Duffy, 1972; Lawler, 1980) with relevance to individual differences in autonomic reactivity and stress-related physiological dysfunction (Malmo & Shagrass, 1949; Engel & Bickford, 1961; Light & Obrist, 1983; Redman & Dutch, 1984b).

As described in Chapter 2, five studies have reported acceleratory and deceleratory changes in tonic responding, but none provide an account of directional variability which is adequate for the observed PTT response.

However, the concept of activation which describes a continuum of alertness or activity with a lower extreme in deep sleep and a higher extreme in excitement (Duffy, 1962) has similarities with the specific behavioural states of coping proposed by Obrist (Obrist, 1981). The main difference between the concept of active and passive coping and that of activation is that the former is more specific, particularly directed toward identifying links between cardiac responses and hypertension (Obrist, 1981). It had been noted that, in Experiment 3, PTT accelerators had larger magnitude acceleratory IBI changes than PTT decelerators during both the hard and the easy problem solving tasks. It therefore seemed likely that more reactive HR responses under particular conditions were associated with acceleratory PTT changes. But it was considered that to focus on cardiac reactivity under particular conditions would be premature at the present stage, since there was also evidence of an association between HR reactivity and the deceleratory PTT response during

mental arithmetic in Experiment 1. To focus on a possible association between HR reactivity and PTT response direction was considered likely to divert attention from environmental and behavioural processes and detract from the understanding of the PTT response by making it another subsidiary to the better documented HR response, and further restrict understanding of relationships between cardiovascular and behavioural processes. In addition, as stated in Experiment 3, the IBI data from Experiments 1, 2 and 3 are best described by a relationship between task difficulty and magnitude of change along a continuum of behavioural coping, and that continuum is also consistent with the concept of a continuum of activation.

Three approaches to activation have been reviewed by Malmo (1959). One emphasizes neurological processes, in particular the contribution of the ascending reticular activating system (ARAS) and other central nervous system processes as the initiating and mediating processes (e.g. Lindsley, 1951; Hebb, 1955); the second emphasizes behavioural intensity or energetics, measured by recording muscle tension or palmar conductance and level of behavioural performance, and advocates that the lawful relation between activation and level of performance could be described by a curvilinear function, in particular the inverted-U curve (Duffy, 1972); and the third emphasizes the congruence between undirected general drive as described in Hullian learning theory and the principle of activation (Malmo, 1959), with physiological measures, particularly HR, used to gauge activation.

It is that third alternative which is considered to be particularly applicable to the current research. In that approach, a high HR is seen to reflect high activation or high general drive (Duffy, 1962; Malmo, 1959), with

an increase in general drive shown in monotonic increases in HR, and a decrease in general drive shown in decreases in HR. Animal studies have suggested that activation level results from the interaction of environmental stimulating factors acting on the internal physiological conditions produced by relative deprivation. Longer periods of deprivation (of sleep, food or water) are associated with higher levels of HR when appropriate stimulation occurs, possibly because central nervous and humoral processes which mediate the HR changes may be sensitized by deprivation and thus respond to external stimulation more rapidly and to a greater degree (Malmo, 1959). Notably, an increase or decrease in HR from one condition to another is considered to reflect an increase or decrease in activation between conditions, but an individual with a higher HR is considered to be not necessarily more activated than an individual with a lower HR.

RESTING HR LEVEL AND DIRECTION OF PTT CHANGE

In the preceding section it was hypothesized that if acceleratory and deceleratory PTT changes were equivalent responses which differ in degree or site of change and are not fundamentally different in mechanism, then the individual's behavioural state prior to the task could be one determinant of the direction of change. Further it was suggested that the individual's behavioural state prior to task could be measured by his/her relative level of resting HR (or IBI) and that the relative level of HR from one baseline to another should predict the direction of the task-generated PTT change following the latter baseline.

It should be noted that the hypothesis does not imply a dependence of PTT on HR, not does it predict a co-variation in task-generated PTT and HR responses. It is an argument which suggests central mediation of an integrated physiology, part of which is the cardiovascular system. It is suggested to be consistent with a concept of an integrated cardiovascular system which is sensitive to the interaction between the individual's relative state and the environmental demands of the moment.

The hypothesis also receives support from the IBI changes in the experiments in the present research. IBI was found to be reactive both in anticipation of, and in response to, the tasks presented. Those responses were consistent with an account in terms of individual engagement level or behavioural state (Obrist, 1976, 1981)

For any individual an increase in relative resting HR is associated with a relative increase in sympathetic influence on the myocardium, whether that is achieved directly or through a decrease in vagal activity, and hence reflects an increase in resting sympathetic tone, consistent with an account of a more active behavioural state. In that case, the effect of any task is superimposed on an already relatively high sympathetic tone, and an increase in HR is likely to be associated with an increase in cardiac output, through an accompanying increase or no change in stroke volume, and also to be associated with a more rapid, predominantly neural response which may be particularly marked in the peripheral vasculature (Hare 1972, 1973), resulting in an over-riding vasoconstriction response and an acceleratory change in PTT.

Conversely, for any individual a decrease in relative resting HR reflects a decrease in resting sympathetic tone, consistent with a less active behavioural

state. As in the previous case, the effect of any task is superimposed on the existing level of sympathetic tone. If the existing level of sympathetic tone is low, an increase in HR may be associated with no change in cardiac output, due to a decrease in stroke volume, and may also be associated with a less rapid or dominant neural response which results in the humorally mediated vasodilation in some vascular beds producing an over-riding vasodilatory response and a deceleratory change in PTT. That hypothesis was tested in Experiments 4 and 5.

In Experiments 4 and 5 it was decided to convert the recorded PTT into PWV in order to minimize the effect of between subject differences in height. That conversion is detailed in the next section.

CONVERSION OF PTT TO PWV

PTT is the time taken for the pressure pulse to travel between two arterial sites, one more distal than the other. The value obtained will reflect not only the speed with which the pulse travelled, but also the distance which the pulse travelled, in particular the length of the arterial segment over which recording takes place. The advantage of reporting PTT lies in the fact that it is the actual value recorded with no error introduced by conversion; the major disadvantage of reporting PTT is that it does not control for the length of artery over which measurement occurred. Given that, in the present study, the recorded values were obtained over the systemic vasculature and that subjects can have quite large differences in height, it was decided to convert the recorded PTT into a measure of PWV.

As discussed in Chapter 1, the most important consideration when converting PTT to PWV in *in vivo* studies is the accurate calculation of the length of artery over which measurement is recorded. When measured over a short peripheral arterial segment on the same arterial branch (e.g. Dutch & Redman, 1983), the actual distance between transducers can be an acceptable measure of arterial length which can then be divided by the recorded PTT to give PWV as shown in equation (6) in Chapter 1. But when, as in the present research, measurement spans arterial segments on different branches a simple measurement of distance between transducers is not appropriate because of the nature of pulse travel. In the present research, for example, the pulse does not travel from the ear to the foot, as would be implied in a transducer-to-transducer measurement, but a single pulse begins from a central location and fragments at arterial bifurcations so that it travels toward the ear and the foot simultaneously.

Systemic pulse transit time begins at the aortic arch when the pulse has been ejected from the left ventricle (at the onset of the second heart sound). The pulse disperses through the major arterial branches towards the peripheral vascular beds in all directions as each pulse fragments at arterial branches and the vessels are fed simultaneously. In the present research, some proportion of each pressure pulse travelled down the abdominal aorta, and on through the iliac, femoral and tibia arteries to the dorsalis pedis arterial site where it was detected. Simultaneously, a proportion of the same pressure pulse travelled up through the innominate and carotid arteries to the auricular arterial site where it was detected. Thus the same pulse was detected at different sites on distinct arterial segments. But the time from leaving the aortic arch until the detection of the proximal pulse at the ear was common

travel time for both portions of the pulse, and the obtained value of PTT reflects the compliance of the arterial walls from the abdominal aorta to the dorsalis pedis artery. The common travel time is not part of the recorded PTT, and thus the distance travelled in that time cannot be included in the calculated PWV.

In order to convert PTT into PWV a noninvasive calculation of the length of the arterial segment over which the pulse travels (from the abdominal aorta to the dorsalis pedis artery) must be made. First it is necessary to know the distance travelled during the common travel time, i.e. that from the aortic arch to the ear detection site. That distance can be measured, as the aortic arch is sited behind the ridge at the centrepoint of the manubrium sternum (at the base of the throat). That ridge can be felt on most indiviuals, and certainly the notch at the top of the manubrium sternum can be located on all individuals with the ridge 1 inch below the notch (Guyton, 1981). Thus the length of the arterial segment travelled by the pulse to the proximal site (L_p) was measured from the ridge on the manubrium sternum to the detection site on the right ear.

Similarly, the full length of the arterial segment travelled by the pulse to the distal site (L_d) can be measured from the ridge on the manubrium sternum to the dorsalis pedis pulse detection site on the right foot. Then the length of the arterial segment can be calculated as the arithmetic difference between the two sites (Avolio, et al., 1983):

$$L_d - L_p = L_a \qquad (12)$$

That calculated value of L_a can then be divided by the recorded value of PTT according to equation (6) in Chapter 1 to obtain a measure of PWV which

represents the propagation rate of the pulse from the abdominal aorta to the dorsalis pedis detection site.

It should be noted that the calculation requires the assumption that the pulse travels at the same speed toward both detection sites. That assumption has not been proven, but is considered appropriate under the circumstances of the present research. Firstly, the distance to the ear is short and thus any error introduced is minimal. Secondly, for most of the common travel time the pulse is travelling through large vessels in both directions. Thus any error introduced by that assumption is not only very small, with no impact on task generated changes, but it does not have any implications for between subject comparisons since the same assumption is made in every case. Finally, any related error must generate conservative conclusions in that, as the speed toward the ear must be either the same or faster than that toward the foot during the common travel time, the length of the abdominal aorta included in the subsequent calculation, and hence the associated effect on cardiac load, can only be underestimated (Avolio, et al., 1983).

7 EXPERIMENT 4

PWV Changes During an Extended Experimental Session

and the Effects of Caffeine on the Cardiovascular Responses

INTRODUCTION

In the experiment to be reported, PTT was converted to PWV as detailed in the previous chapter in order to control for differences in subject height and thus control for the contribution made to the obtained absolute PTT value by the length of the arterial segment over which measurement was recorded.

An initial test of the hypothesis proposed in the previous chapter was designed. Resting levels of IBI during initial baseline periods were used as an indication of initial level of activation, or internal physiological conditions (Malmo, 1959), prior to participating in the tasks. It was predicted that an increase in IBI from one initial baseline to the next would be associated with an acceleratory PWV response during task, and that a decrease in IBI from one initial baseline to the next would be associated with a deceleratory PWV response during task (Duffy, 1972).

In Experiment 4 there were two direct manipulations of the theoretical activation, or level of behavioural state, firstly by using an extended experimental session, and secondly by giving some subjects caffeine.

The experimental session lasted between 2-3 hours and was divided into two identical parts separated by a short rest period, and with all four tasks from Experiment 3 in both parts. It was expected that IBI would be lower in the initial baseline of the second part of the experimental session than in the first part, and therefore deceleratory PWV change would be more likely to occur in part 2.

Caffeine consumption is typically reported to be associated with an increase in HR, or IBI, (Thorp & Cobbin, 1967) but has been hypothesized to either raise or lower activation level under stress depending on personal initial level of activation (Revelle, Amaral & Turriff, 1976). Consistent with the latter suggestion, recent psychophysiological studies have reported either no change or a small decrease in HR during stress following consumption of a moderate amount of caffeine (Lane, 1983; Lane, et al., 1985). It was expected that, if consumed in the interval between session parts, caffeine should reverse the effect of the extended session and raise activation. Therefore subjects who consumed caffeine should have an increase in HR in the initial baseline of part 2 of the experimental session relative to the initial baseline in part 1, and for those subjects, an acceleratory PWV change during tasks in part 2 should be more likely to occur.

The possibility that the administration of the psychological scales in Experiment 3 had been inappropriately timed was also considered. If the individual's state can change from moment to moment (Spielberger, 1972) it could be that the score obtained following task completion may be more likely to demonstrate a relationship with the direction of response during the task. That hypothesis was tested by including the psychological scales which were

introduced in Experiment 3 in the present experiment but changing the time of administration: in Experiment 3 subjects completed the scales prior to the beginning of data recording; in Experiment 4 subjects completed the scales during the interval following part 1 of the experimental session. It should be noted that the intention of the use of the scales remained solely as the identification of any possible relationship between the scores on the scales and the direction of PWV change during task.

As in Experiments 2 and 3 PWV was the response of primary interest, IBI was measured as the cardiac response and SBP and DBP were measured concurrently. RPI data were also recorded and are presented in Appendix 2.

METHOD

Subjects

The thirty undergraduate psychology students who participated in Experiment 3 all returned to participate in Experiment 4.

Apparatus and Physiological Recording

The apparatus and physiological measures were identical to those used in Experiment 3 except that PTT was converted to pulse wave velocity (PWV).

The distance from the centre of the manubrium sternum to each pulse detection site was measured. The distance to the ear site was subtracted from the distance to the foot site, and the obtained difference was divided by the recorded PTT to give PWV.

PROCEDURE

The tasks used, the initial baseline, pre-task baseline and post-task periods were identical to those in Experiment 3. As the subjects were all experienced, and the tasks all familiar, no pre-task instruction periods were necessary.

Subjects each attended one experimental session which lasted between 2 and 3 hours, selecting their own preferred times. Subjects were not controlled for habitual use of caffeine but were all requested not to consume tea, coffee, cola drinks or caffeine-containing substances for 8 hours prior to the experiment (Revelle, et al., 1980). The session was divided into 3 sections as described below.

Part 1.

On arrival at the laboratory subjects signed an informed consent form then entered the experimental room where the transducers were attached and the experiment began, proceeding exactly as in Experiment 3 with the subject completing all four tasks.

As in Experiment 3, following completion of the experiment, the relative difficulty of the tasks was evaluated by paired means comparisons of the behavioural performance scores on each task. More problems were solved correctly during the easy task than during the hard task (79.0% vs 35.8%, p<.0001) and there were fewer errors of omission during the easy task than

during the hard task (17.2% vs 56.3%, p<.0001). There were more problems solved correctly during the hard task than during the impossible task (35.8% vs 7.7%, p<.0001), there were fewer errors of omission during the hard task than during the impossible task (56.3% vs 76.8%, p<.0001) and there were also fewer problems answered incorrectly during the hard task than during the impossible task (7.9% vs 15.5%, p<.01).

Rest Period.

Following completion of the tasks, the transducers were removed, and the subject was given a cold, premixed orange flavoured drink. For half of the subjects, the drink contained 200mg of caffeine. The caffeine was obtained as commercially available 100mg pills which were dissolved in the drink. The other half of the subjects had a similarly orange flavoured drink which contained a commercially available quinine tonic water. Pilot work had shown the caffeine and tonic water drinks to be indistinguishable in taste or appearance. Subjects were assigned to either the caffeine or the no caffeine condition in order of their arrival at the laboratory balanced with regard to subject sex. There were 8 men and 7 women in each condition.

Subjects were unaware of the condition they were in: all were informed that the drink might contain caffeine. Following consumption of the drink (about 50mls), each subject completed the SACL and STAI questionnaires.

Part 2.

After at least 30 minutes and not more than 45 minutes had elapsed from the time of finishing the drink, the subject returned to the experimental room, the transducers were re-attached and the subject completed the same 4 tasks in a different order for the second time.

In part 2 of the experimental session, as in part 1 and in Experiment 3, following completion of the experiment the relative difficulty of the tasks was evaluated by paired means comparisons of the behavioural performance

Similarly, in part 2 of the experimental session, there were more problems answered correctly (82.9% vs 43.4%, p<.0001) and fewer errors of omission (13.5% vs 49.8%, p<.0001) during the easy task than during the hard task, and more problems answered correctly (43.4% vs 9.45%, p<.0001), fewer errors of omission (49.8% vs 71.1%, p<.0001), and fewer problems answered incorrectly (6.9% vs 19.5%, p<.04) during the hard task than during the impossible task.

RESULTS

The median value for IBI and PWV over every minute of the session was obtained. SBP and DBP were recorded during minutes 2 and 4 of the initial baseline periods, and once each during every pre-task baseline, task and post-task period. These values formed the bases of all the reported analyses.

Analyses of Absolute Levels

The absolute levels of cardiovascular activity were analysed as described in Experiment 1. In addition, levels during each experimental period were analysed for evidence both of the effects of caffeine (Condition), and of any differences over the parts of the extended session (Part).

Initial Baseline

The absolute levels of IBI and PWV during the initial baseline were analysed using a Sex (2) x Condition (2) x Part (2) x Minute (5) ANOVA with repeated measures on the last two factors. The absolute levels of SBP and DBP during the initial baseline were analysed using a Sex (2) x Condition (2) x Part (2) ANOVA with repeated measures on the last factor.

There was a Main Effect of Part for absolute levels of IBI (F(1/26)=32.53, p<.0001). During part 1, IBI was shorter during the initial baseline than it was during part 2 (see Table 4.1).

There was also a Main Effect of Condition (F(1/26)=4.51, p<.04 for absolute levels of IBI. Subjects who consumed caffeine had longer IBI during the initial baseline than subjects who did not (884ms vs 803ms).

There was a Main Effect of Sex (F(1/26)=5.71, p<.02) for PWV. Women had slower PWV than men (see Table 4.2).

There was a Main Effect of Minute=(F(4/104)=13.38, p<.0001) for PWV reflecting a deceleratory response which occurred over the initial baselines in both parts.

A Main Effect of Sex was found for the measure of SBP taken during minute 2 (F(1/26)=11.54, p<.002) and for that taken during minute 4 (F(1/26)=6.77, p<.02) of both initial baselines. Women had lower SBP than men (see Table 4.3).

There was a Main Effect of Part found for the second measure of SBP taken during minute 4 of both initial baselines (F(1/26)=5.03, p<.03, 114.77mmHg vs 118.37mmHg). It was lower in part 1 of the session than in part 2.

There was a Main Effect of Part found for the measure of DBP taken during minute 2 of the initial baselines (F(1/26)=19.69, p<.0001, 66.93mmHg vs 72.83mmHg) and also for the measures taken during minute 4 (F(1/26)=9.96, p<.004, 66.27mmHg vs 71.73mmHg). DBP was higher in part 2 than in part 1.

There was a Condition x Part Interaction found for the measures of DBP taken during minute 2 of the initial baselines (F(1/26)=10.14, p<.004). Subjects who had consumed caffeine had an increase in DBP, but those who had not consumed caffeine did not (see Table 4.4).

There was a Sex x Part Interaction for the measure of DBP taken during minute 4 of the initial baseline (F(1/26)=8.05, p<.009). Men had an increase in DBP but women did not (see Table 4.5).

TABLE 4.1

Interbeat Interval (IBI) levels in milliseconds during the initial baseline and pre-task baseline periods in each session part accompanied by the pulse wave velocity (PWV) percent change during tasks in each session part.

		Part 1		Part 2
Initial Baseline	IBI	815	*	872
Pretask Baseline	IBI	837	*	868
Percent Change	PWV	+ 2.07	*	− 0.90

*   p<.05 or better

TABLE 4.2

Pulse Wave Velocity (PWV) levels in m/sec for men and women during the initial baseline and pre-task baseline. A Main Effect of Sex was found for both comparisons.

		Men (n=16)		Women (n=14)
Initial Baseline	PWV	12.97	*	11.04
Pretask Baseline	PWV	12.55	*	10.62

*   p<.05 or better

TABLE 4.3

Systolic (SBP) and Diastolic (DBP) Blood Pressure levels in mmHg for men and women during the initial baseline and pre-task baseline. A Main Effect of Sex was found for all SBP comparisons.

		Men (n=16)		Women (n=14)
Initial Baseline 1	SBP	123.59	*	110.50
	DBP	68.88		71.04
Initial Baseline 2	SBP	121.50	*	110.93
	DBP	68.09		70.04
Pretask Baseline	SBP	121.78	*	108.96
	DBP	68.31		69.37

* p<.05 or better

TABLE 4.4

Diastolic Blood Pressure (DBP) levels in mmHg recorded in the first measure taken during the initial baseline and pretask baseline periods during each part of the experimental session for subjects who ingested caffeine and those who did not. The tabled data show the Condition (Caffeine/No Caffeine) X Part (Part 1/Part 2) Interaction reported in the text.

		Initial Baseline Periods	
		Caffeine	No Caffeine
Part 1	DBP	65.87	68.00
Part 2	DBP	76.00	69.67

		Pretask Baseline Periods	
		Caffeine	No Caffeine
Part 1	DBP	64.93	69.07
Part 2	DBP	72.88	68.33

* $p < .05$ or better

TABLE 4.5

Diastolic Blood Pressure (DBP) levels in mmHg recorded in the second measure taken during the initial baseline periods during each part of the experimental session for men and women.   The tabled data show the Sex X Part (Part 1/Part 2) Interaction reported in the text.

		Men (n=16)	Women (n=14)
Part 1	DBP	65.10	69.93
Part 2	DBP	72.60	73.10

*   p<.05 or better

Pre-task Baseline Periods

The absolute levels of IBI, PWV, SBP and DBP during the pre-task baseline periods were analysed using a Sex (2) X Condition (2) x Part (2) x Task (4) ANOVA with repeated measures on the last two factors. Secondary analyses which substituted Task Order for Task were also performed.

There was a Main Effect of Part for IBI ($F(1/26)=11.41$, $p<.002$). IBI was shorter during the pre-task baselines in part 1 of the session than in part 2 (see Table 4.1).

There was a Main Effect of Sex ($F(1/26)=5.96$, $p<.02$) for PWV during the pre-task baselines. Women had slower PWV than men (see Table 4.2).

There was a Main Effect of Sex ($F(1/26)=11.02$, $p<.003$) for SBP during the pre-task baselines. Women had lower SBP than men (see Table 4.3).

There was a Main Effect of Part for SBP ($F(1/26)=6.73$, $p<.02$). Pre-task baseline level of SBP was lower during part 1 of the experimental session than during part 2 (114.38mmHg vs 117.22mmHg).

There was a Main Effect of Task ($F(3/78)=4.23$, $p<.008$) for SBP. SBP was lower prior to the easy (114.93mmHg) and impossible (115.05mmHg) tasks than prior to the hard (116.38mmHg) or personal tempo (116.83mmHg) tasks.

There was a Main Effect of Part ($F(1/26)=10.39$, $p<.003$) for DBP during the pre-task baselines. DBP was lower during the pre-task baselines in part 1 of the experimental session than during those in part 2 (67mmHg vs 70.61mmHg).

There was a Condition x Part Interaction for DBP ($F(1/26)=15.04$, $p<.0006$). Subjects who had consumed caffeine had an increase in DBP, those who did not consume caffeine did not (see Table 4.4).

Analyses of Response Change

Response change from the immediately preceding baseline was calculated for every minute median response value of IBI and PWV, and for the measures of SBP and DBP from all task and post-task periods. Response changes during tasks for men and women are given in Appendix 1.

Task Periods

Response changes for all the cardiovascular measures during the task periods were analysed using a Sex (2) x Condition (2) x Part (2) x Task (4) ANOVA with repeated measures on the last 2 factors. The analyses did not include Minutes due to the large number of other variables. Secondary analyses which substituted Task Order for Task were also performed.

There was a Main Effect of Tasks ($F(3/78)=21.06$, $p<.0001$) for task-generated changes in IBI. Follow-up REGWF tests showed that the magnitude of change in IBI differentiated tasks in the following descending order: hard task (-58ms), impossible task (-52ms), easy task (-47ms), personal tempo task (-7ms). That effect is shown in Figure 4.1 where percentage IBI change is graphed as a function of task difficulty.

There was a Condition x Task Interaction ($F(3/78)=3.37$, $p<.02$) for task generated changes in IBI. Subjects who had consumed caffeine had larger

acceleratory changes during the easy and personal tempo tasks exacerbating the task-generated change. The interaction is shown in Figure 4.2

There was a Main Effect of Part ($F(1/26)=4.98$, $p<.03$) found for task-generated changes in PWV. The change in PWV was acceleratory in part 1 of the experimental session (+2.07%), and was deceleratory in part 2 (-0.90%), see Table 4.1.

There was a Main Effect of Sex ($F(1/26)=6.03$, $p<.02$) found for task-generated changes in SBP. Women had an increase (+0.79%) and men had a decrease (-0.66%).

There was a Sex x Part Interaction ($F(1/26)=4.32$, $p<.05$) found for task-generated change in SBP. Change in SBP during part 2 of the session was smaller than that which occurred during part 1 for both sexes. The interaction is shown in Figure 4.3.

There was a Sex x Part x Condition x Task Interaction ($F(3/78)=3.51$, $p<.02$) for task-generated change in DBP. That interaction is shown in Figure 4.4.

Behavioural Task Performance

Task performance measurements, except for those during personal tempo, were scored then converted to percentages as described in Experiment 3.

Pearson Product Moment correlation analyses were performed which looked at associations between behavioural performance and task-generated changes in cardiovascular responses as a function of the caffeine/no caffeine conditions. No systematic effects were found.

Task Performance, Sex, Caffeine and Session Part

Behavioural performance was also analysed using a Sex (2) x Condition (2) x Part (2) ANOVA with repeated measures on the last factor.

There was a Main Effect of Sex (F(1/26)=9.18, p<.006) found for the numbers of problems answered correctly during the hard task. Women answered more problems correctly than men (see Table 4.6).

There was a Main Effect of Sex (F(1/26)=8.92, p<.006) found for the numbers of errors of omission during the hard task. Women omitted fewer problems than men (see Table 4.6).

There was a Main Effect of Sex (F(1/26)=6.47,p<.02) found for the numbers of problems answered correctly during the impossible task. Women answered more problems correctly than men (see Table 4.6).

There was a Main Effect of Sex (F(1/26)=4.95, p<.04) for the numbers of errors of omission during the impossible task. Women omitted fewer problems than men (see Table 4.6).

There was a Main Effect of Part found for numbers of problems correctly answered during the easy and the hard tasks. More problems were correctly solved in part 2 of the experimental session than in part 1 (see Table 4.7).

There was a Main Effect of Part found for errors of omission during all three problem solving tasks. Fewer problems were omitted during part 2 of the experimental session than in part 1 (see Table 4.7).

There was a Main Effect of Part for numbers of problems answered incorrectly during the impossible task. There were more incorrect responses during part 2 than during part 1 (see Table 4.7).

There was a Condition x Part Interaction ($F(1/26)=4.73$, $p<.04$) found for the number of correct responses during the hard task. Subjects who had consumed caffeine had an increase in the number of problems solved correctly from part 1 to part 2 (from 28.9% correct to 41% correct); those who had not consumed caffeine did not (42.7% to 45.7%).

There was a Main Effect of Sex ($F(1/26)=4.27$, $p<.05$) found for the button presses made during the personal tempo task. Women made more presses than men (18.5 vs 12.6).
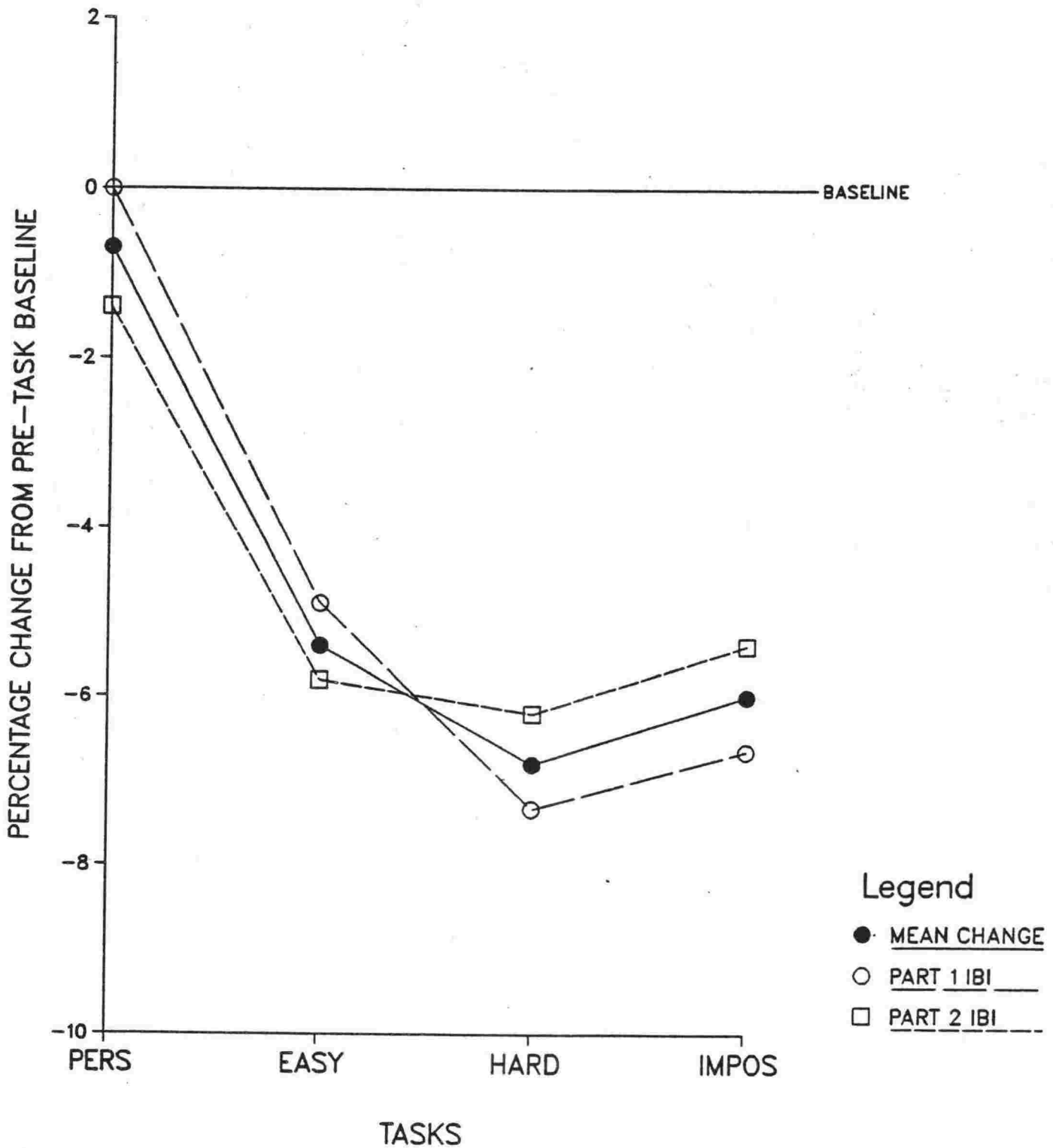
Figure 4.1 Percent change in Interbeat Interval during tasks
(Pers=personal tempo; Easy=easy problems;
Hard=hard problems; Impos=impossible problems.)
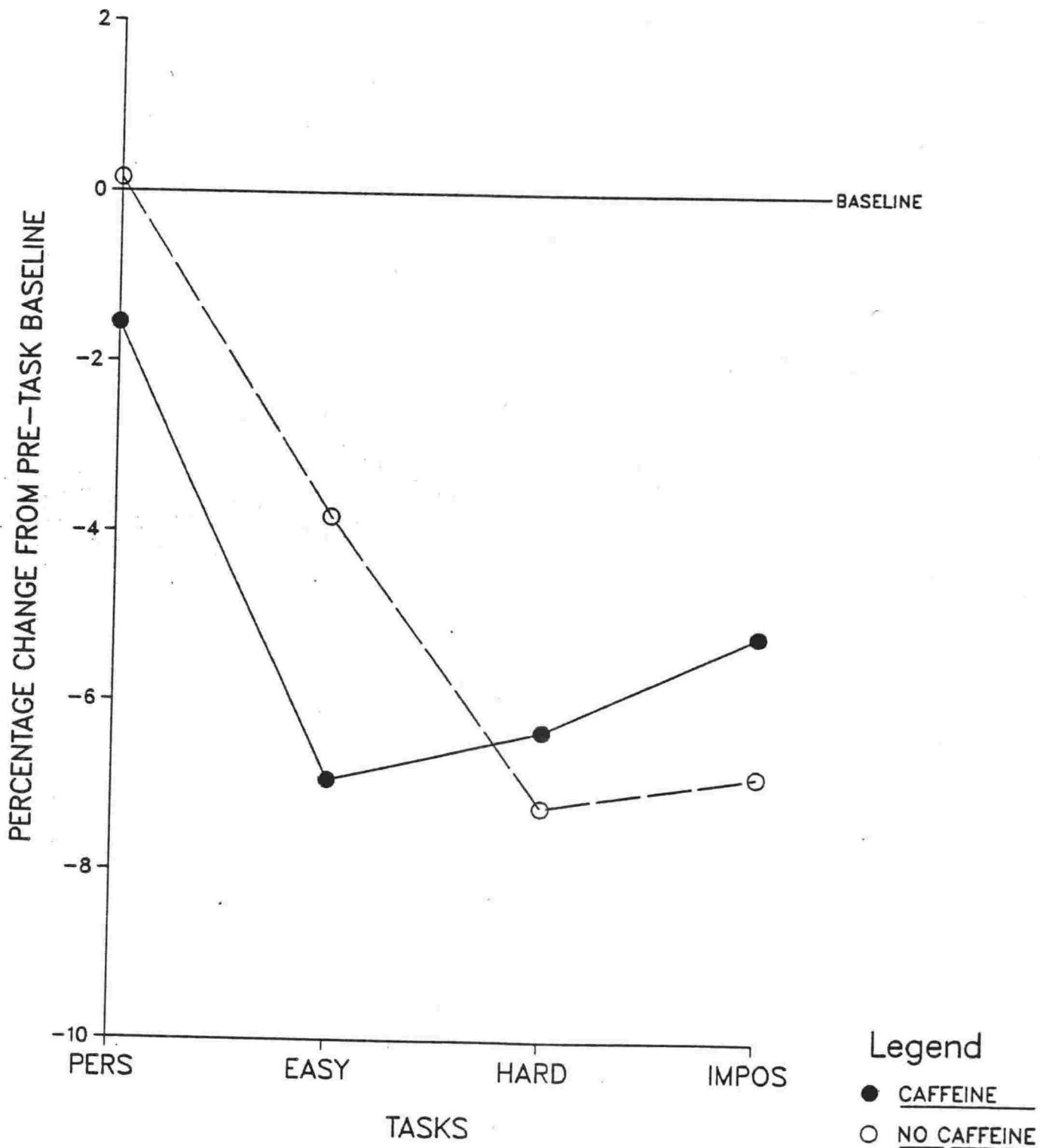
Figure 4.2 Percent change in Interbeat Interval during tasks showing Condition X Task Interaction during task. (Pers=personal tempo; Easy=easy problems; Hard=hard problems; Impos=impossible problems.)
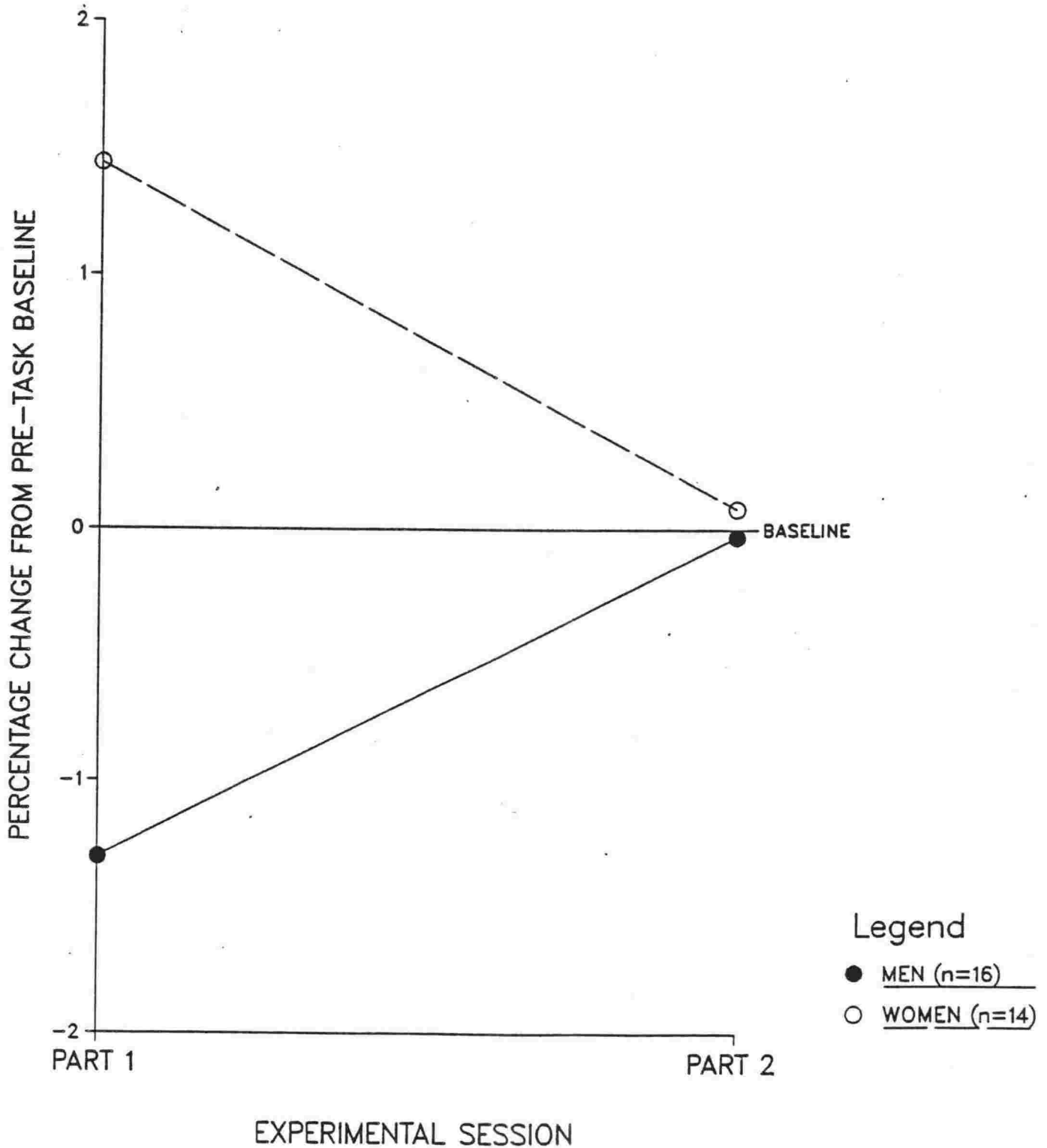
Figure 4.3 Percent change in Systolic Blood Pressure showing the Sex X Part Interaction reported in the text.
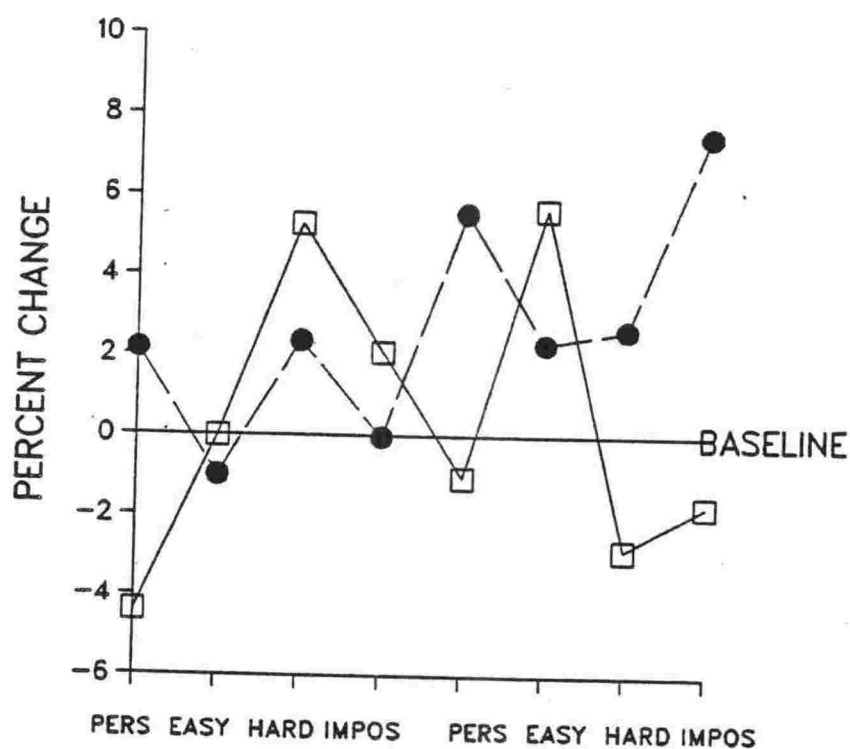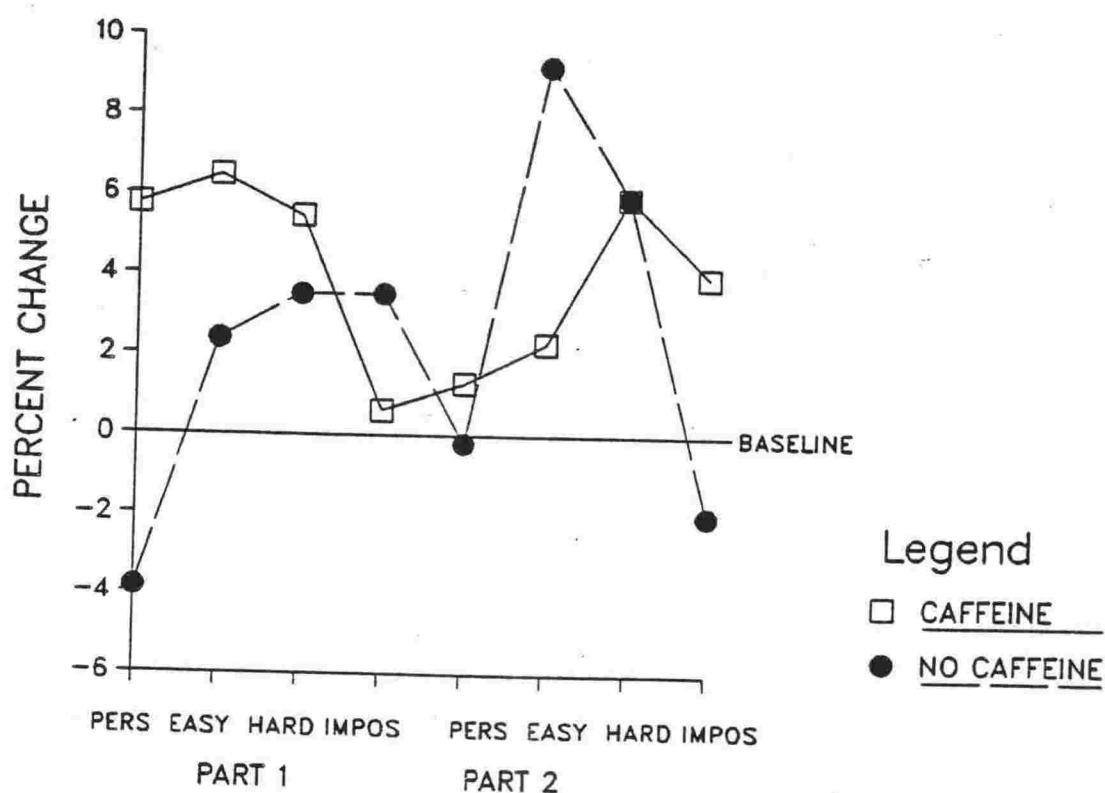
Figure 4.4 Diastolic Blood Pressure Response during task in both session parts for men and women showing the Sex X Part X Condition X Task Interaction reported in the text (PERS=personal tempo; EASY=easy problems; HARD=hard problems; IMPOS=impossible problems).

TABLE 4.6

Behavioural task performance for men and women. Problem solving performance is shown as percentage of total problems presented. Personal tempo button presses are shown as mean number, and latency between presses is given in milliseconds.

		Men (n=16)		Women (n=14)
Easy Task	Correct	80.53		81.41
	Omitted	15.97		14.62
	Wrong	3.50		3.97
Hard Task	Correct	30.59	*	49.84
	Omitted	63.50	*	41.07
	Wrong	5.91		9.09
Imp. Task	Correct	5.50	*	6.81
	Omitted	82.66	*	75.73
	Wrong	11.84		23.96
Pers. Temp	Presses	11.70	*	18.30
	Latency	37.02		16.03

* $p < .05$ or better

TABLE 4.7

Behavioural task performance in each part of the experimental session. Problem solving performance is shown as percentage of total problems presented. Personal tempo button presses are shown as mean number, and latency between presses is given in milliseconds.

		Part 1		Part 2
Easy Task	Correct	79.04	*	82.85
	Omitted	17.20	*	13.47
	Wrong	3.76		3.68
Hard Task	Correct	35.79	*	43.36
	Omitted	56.27	*	49.79
	Wrong	7.94		6.85
Imp. Task	Correct	7.69		9.45
	Omitted	76.76	*	71.10
	Wrong	15.54	*	19.45

* p<.05 or better

Direction of Response Change

As in the previous three experiments, subjects were classified as having acceleratory or deceleratory changes in each cardiovascular response on each task, and in this experiment, during each part of the experimental session. (see Tables 4.8, 4.9, 4.10, 4.11, 4.12, 4.13, 4.14 and 4.15). The concurrently measured response changes and the significance of each deviation from baseline are also given in the tables.

Minute-by-minute divergence over tasks for IBI is shown in Figures 4.5 and 4.6, and for PWV in Figures 4.7 and 4.8. As previously, accelerators and decelerators were tested for differences in sex, baseline levels of response, behavioural performance and scores on the psychological scales. No differences were found.

Table 4.8

Interbeat Interval (IBI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup in each task in part 1 of the experimental session. The concurrently measured Arterial Pulse Wave Velocity (PWV) changes are shown in m/sec, and the concurrently measured Systolic (SBP) and Diastolic (DBP) Blood Pressure changes are shown in mmHg. The significance of deviation from pre-task baseline for each change is indicated.
(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory IBI	Concurrently Measured PWV	SBP	DBP
Easy	26	-54*	+0.11	+2.42*	+1.42
Hard	28	-68*	+0.38*	-0.07	+2.00
Impos	28	-63*	+0.24	-0.64	+0.50
Pers	13	-27*	+0.07	-1.46	-2.00

Task	N	Deceleratory IBI	Concurrently Measured PWV	SBP	DBP
Easy	4	+25	+0.16	-6.00	-3.50
Hard	2	+21	+0.49	-0.50	+7.00
Impos	2	+22	+0.44	-0.02	+5.00
Pers	17	+21*	+0.04	-1.29	+0.61

* p<.05 or better
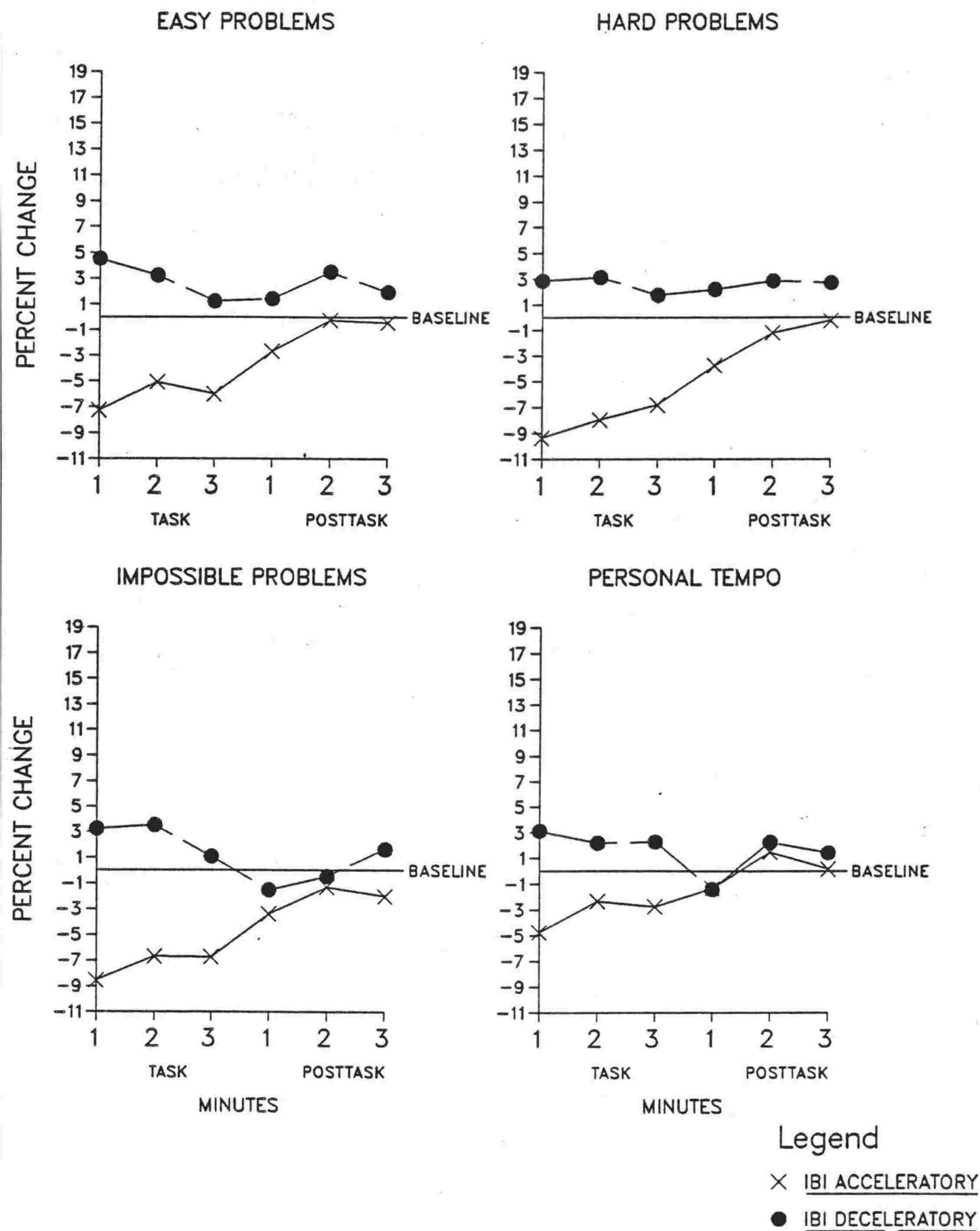
Figure 4.5 Minute—by—minute Interbeat Interval acceleratory and deceleratory responses, Part 1, Experiment 4.

Table 4.9

Interbeat Interval (IBI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup in each task during part 2 of the experimental session. The concurrently measured Arterial Pulse Wave Velocity (PWV) changes are shown in m/sec, and the concurrently measured Systolic (SBP) and Diastolic (DBP) Blood Pressure changes are shown in mmHg. The significance of deviation from pre-task baseline for each change is indicated. (Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory IBI	Concurrently Measured PWV	SBP	DBP
Easy	26	-62*	+0.14	+0.54	+2.53
Hard	26	-66*	-0.07	+0.31	+2.35
Impos	26	-62*	-0.23	-0.15	+1.42
Pers	18	-37*	-0.16	-0.50	+2.11

| Task	N	Deceleratory IBI	Concurrently Measured PWV	SBP	DBP
Easy	4	+18	-0.87*	-1.00	+6.00
Hard	4	+34	-0.70*	-1.50	-3.00
Impos	4	+27	+0.55*	+2.00*	-0.50
Pers	12	+22*	-0.17	-1.17	-1.42

* p<.05 or better
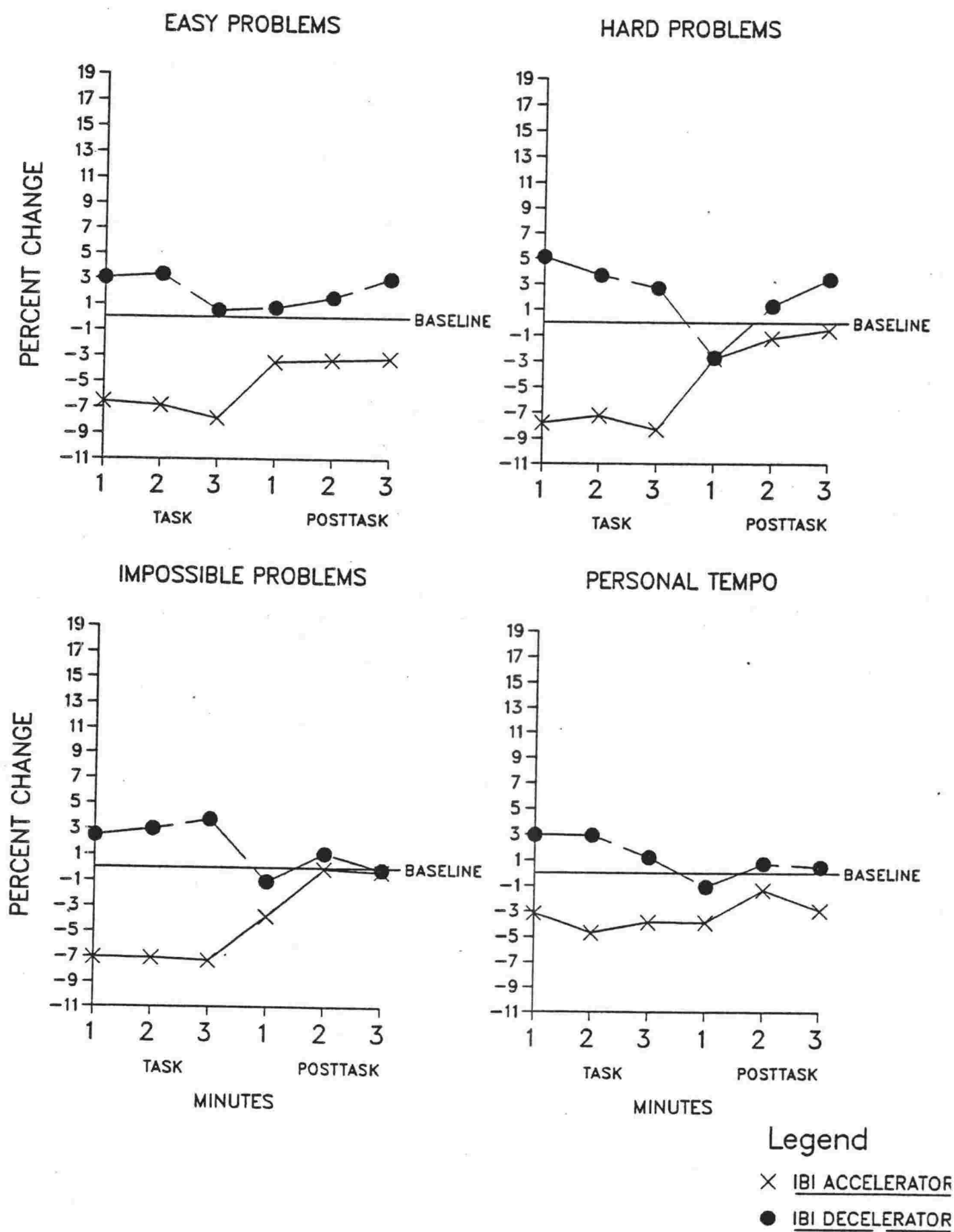
Figure 4.6 Minute—by—minute Interbeat Interval
acceleratory and deceleratory responses,

Part 2, Experiment 4.

Table 4.10

Arterial Pulse Wave Velocity (PWV) Response Changes (in m/sec) from pre-task baseline levels for each subgroup in each task during part 1 of the experimental session. The concurrently measured Interbeat Interval (IBI) changes are shown in milliseconds, and the concurrently measured Systolic (SBP) and Diastolic (DBP) Blood Pressure changes are shown in mmHg. The significance of deviation from pre-task baseline for each change is indicated.
(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory PWV	Concurrently Measured IBI	SBP	DBP
Easy	19	+0.34*	-32*	+0.76	+1.82
Hard	18	+0.66*	-55*	+0.33	+2.23
Impos	11	+0.60*	-49*	-0.48	+0.24
Pers	14	+0.35*	+ 7	-0.76	+0.41

| Task	N	Deceleratory PWV	Concurrently Measured IBI	SBP	DBP
Easy	11	-0.18*	-57*	+2.00	-0.62
Hard	12	-0.38*	-85*	-1.25	2.625
Impos	19	-0.56*	-76*	-0.89	+2.11
Pers	16	-0.34*	- 8	-2.15	-1.54

* $p < .05$ or better
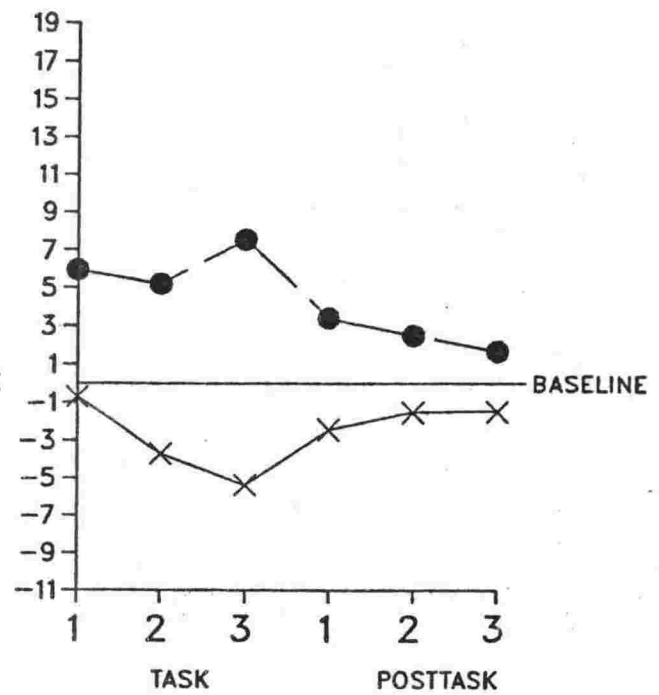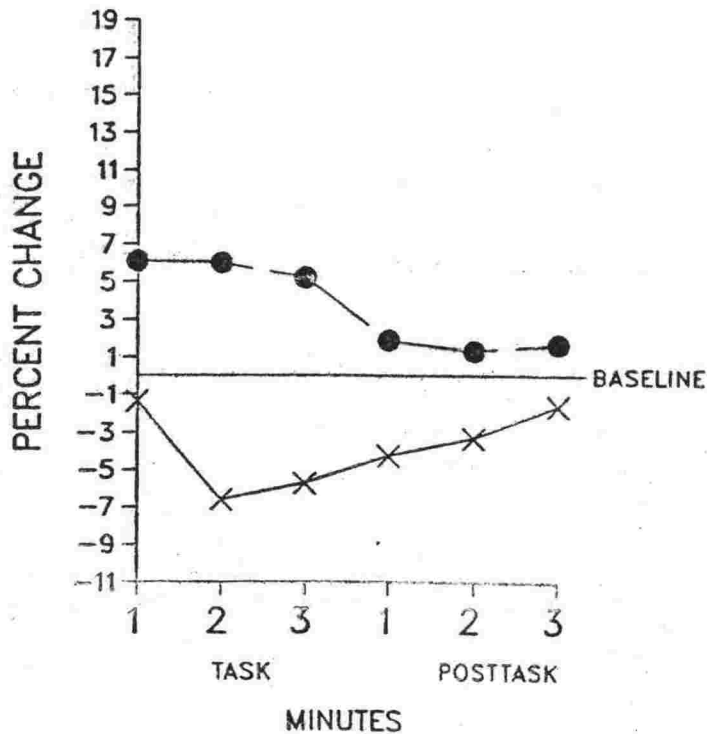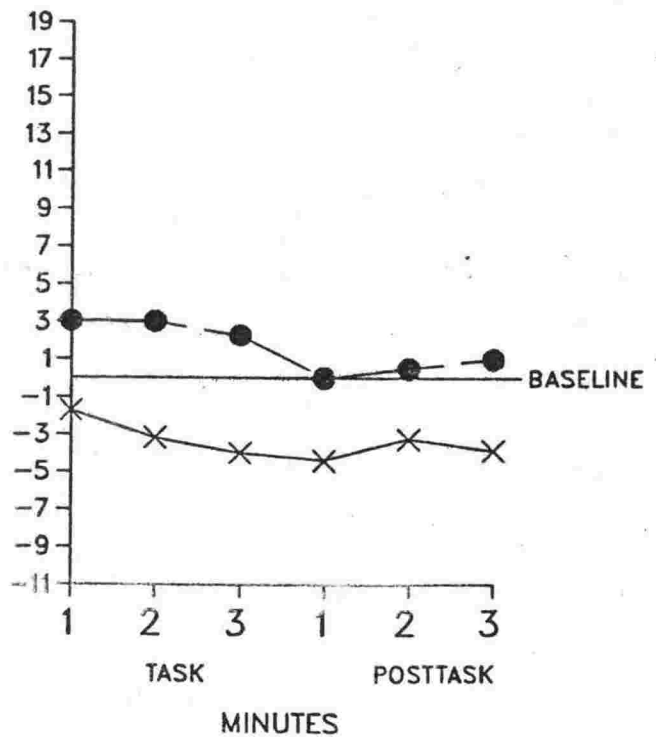
Figure 4.7 Minute–by–minute Pulse Wave Velocity acceleratory and deceleratory responses, Part 1, Experiment 4.

Table 4.11

Arterial Pulse Wave Velocity (PWV) Response Changes (in m/sec) from pre-task baseline levels for each subgroup in each task during part 2 of the experimental session. The concurrently measured Interbeat Interval (IBI) changes are shown in milliseconds, and the concurrently measured Systolic (SBP) and Diastolic (DBP) Blood Pressure changes are shown in mmHg. The significance of deviation from p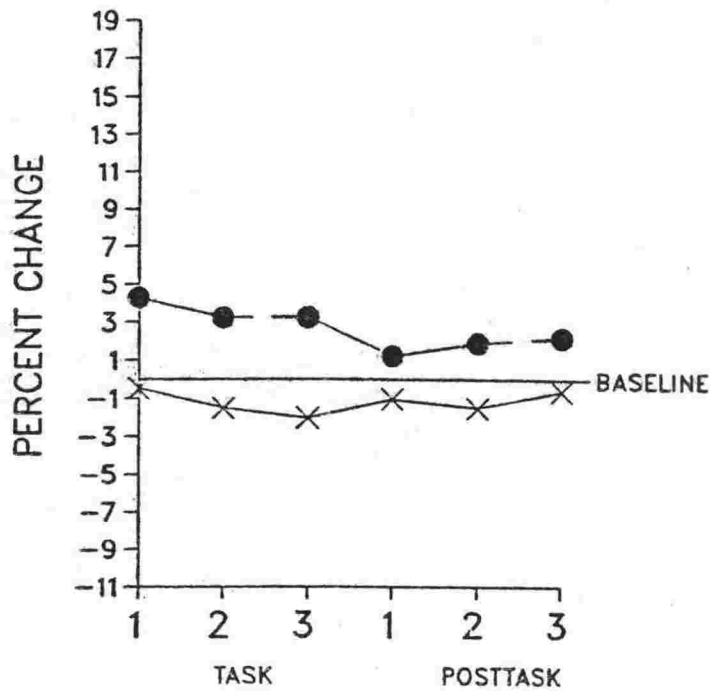re-task baseline for each change is indicated. (Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory PWV	Concurrently Measured IBI	SBP	DBP
Easy	14	+0.60*	-64*	+0.47	+4.33*
Hard	17	+0.54*	-59*	+1.24	+3.88
Impos	11	+0.59*	-64*	-0.55	+1.75
Pers	13	+0.24*	-17	-0.08	+2.69

Task	N	Deceleratory PWV	Concurrently Measured IBI	SBP	DBP
Easy	16	-0.59*	-39*	+0.02	+1.70
Hard	13	-1.06*	-44*	-1.46	-1.31
Impos	19	-0.77*	-42*	+0.53	+0.84
Pers	17	-0.47*	-11	-1.29	-0.82

* p<.05 or better
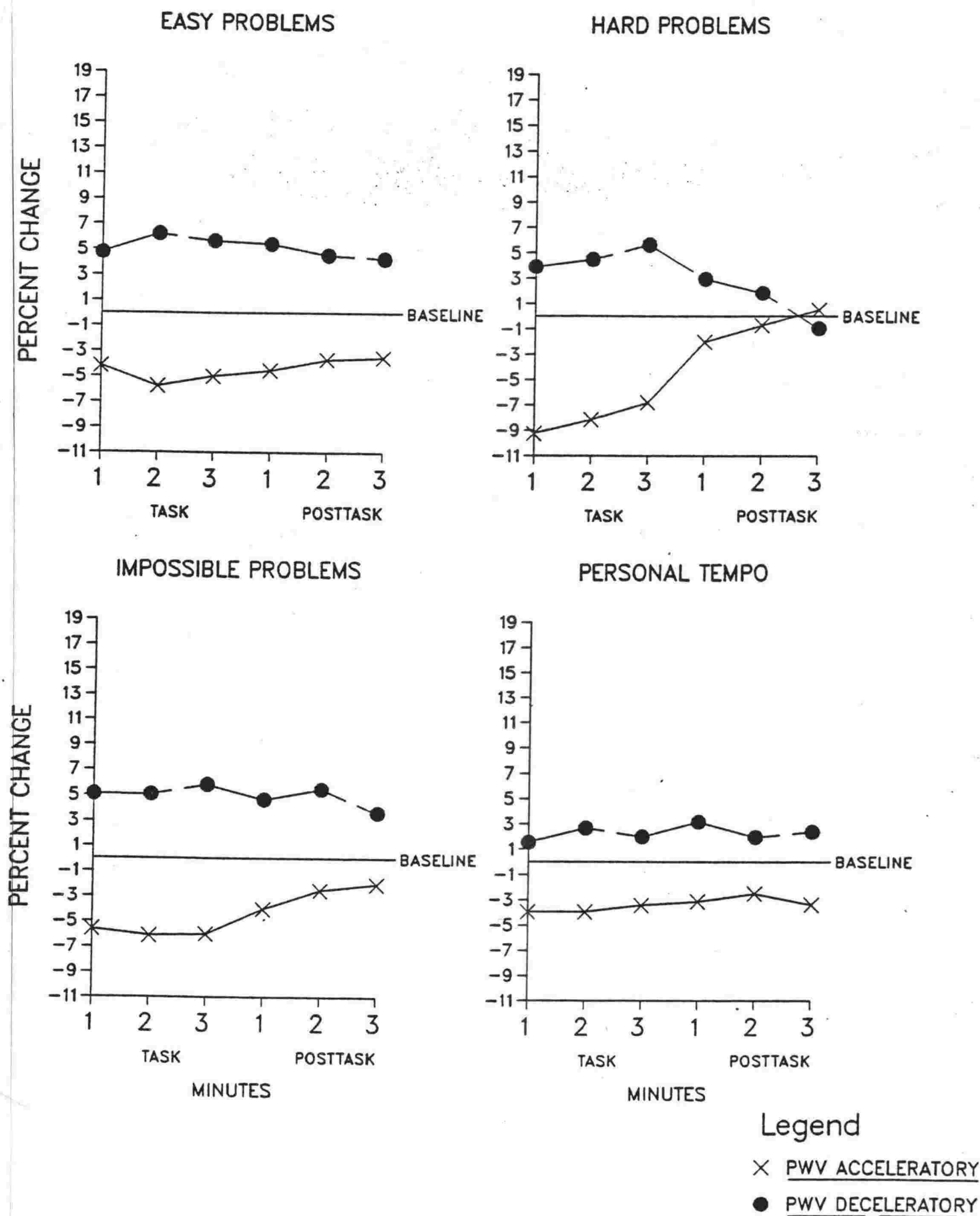
Figure 4.8 Minute—by—minute Pulse Wave Velocity acceleratory and deceleratory responses,

Part 2, Experiment 4.

Table 4.12

Systolic Blood Pressure (SBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup in each task during part 1 of the experimental session.[a] The concurrently measured Diastolic Blood Pressure (DBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) changes are shown in milliseconds and Arterial Pulse Wave Velocity (PWV) changes are in m/sec. The significance of deviation from pre-task baseline for each change is indicated.

(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory SBP	Concurrently Measured IBI	PWV	DBP
Easy	16	+5.63*	-58*	+0.12	+1.81
Hard	18	+5.67*	-68*	+0.59*	+3.50
Impos	17	+4.08*	-45	+0.57	+0.08
Pers	12	+2.41*	+ 3	+0.10	+0.50

Task	N	Deceleratory SBP	Concurrently Measured IBI	PWV	DBP
Easy	12	-4.25*	-26	-0.06	-1.92
Hard	8	-5.07*	-61*	+0.25	+2.14
Impos	12	-4.17*	-67*	+0.01	+1.35
Pers	16	-4.67*	+ 3	+0.04	-1.00

[a] all tasks produced at least one zero responder
* p<.05 or better

Table 4.13

Systolic Blood Pressure (SBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup in each task during part 2 of the experimental session.[a] The concurrently measured Diastolic Blood Pressure (DBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) changes are shown in milliseconds and the Arterial Pulse Wave Velocity (PWV) changes are in m/sec. The significance of deviation from pre-task baseline for each change is indicated.

(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory SBP	IBI	Concurrently Measured PWV	DBP
Easy	16	+4.83*	-72*	+0.19	+4.50*
Hard	16	+5.22*	-90*	+0.26	+8.11*
Impos	16	+3.00*	-41	-0.25	+2.71
Pers	17	+3.17*	-23	-0.11	+4.08*

Task	N	Deceleratory SBP	IBI	Concurrently Measured PWV	DBP
Easy	9	-3.69*	-34*	-0.17	+0.85
Hard	12	-3.46*	-39*	+0.30	-1.10
Impos	10	-4.70*	-67*	-0.44	-0.60
Pers	9	-4.07*	- 8	-0.20	-1.73

[a] all tasks produced at least one zero responder
* p<.05 or better

Table 4.14

Diastolic Blood Pressure (DBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup in each task during part 1 of the experimental session.[a] The concurrently measured Systolic Blood Pressure (SBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) changes are shown in milliseconds and the Arterial Pulse Wave Velocity (PWV) changes are in m/sec. The significance of deviation from pre-task baseline for each change is indicated.

(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory DBP	IBI	Concurrently Measured PWV	SBP
Easy	16	+5.13*	-55*	+0.20	+2.75*
Hard	12	+5.67*	-71*	+0.40*	-1.00
Impos	13	+3.71*	-43*	+0.17	-0.47
Pers	12	+3.42*	+ 4	+0.16	-0.83

Task	N	Deceleratory SBP	IBI	Concurrently Measured PWV	DBP
Easy	12	-4.92*	-32	+0.25	-0.83
Hard	14	-4.00*	-57*	+0.34	-0.38
Impos	17	-3.25*	-74*	+0.41	-0.75
Pers	14	-4.88*	- 6	+0.03	-2.06*

[a] all tasks except the impossible task produced at leastone zero responder
* p<.05 or better

Table 4.15

Diastolic Blood Pressure (DBP) Response Changes (in mmHg) from pre-task baseline levels for each subgroup in each task during part 1 of the experimental session.[a] The concurrently measured Systolic Blood Pressure (SBP) changes are also shown in mmHg. The concurrently measured Interbeat Interval (IBI) changes are shown in milliseconds and the Arterial Pulse Wave Velocity (PWV) changes are in m/sec. The significance of deviation from pre-task baseline for each change is indicated.

(Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory DBP	Concurrently Measured IBI	PWV	SBP
Easy	12	+7.50*	-57*	+0.33	+1.75
Hard	9	+6.31*	-73*	-0.16	-1.31
Impos	17	+5.38*	-49*	-0.39	-0.13
Pers	12	+4.35*	-13	-0.14	+0.94

Task	N	Deceleratory SBP	Concurrently Measured IBI	PWV	DBP
Easy	13	-3.33*	-53*	-0.17	-1.33
Hard	13	-4.33*	-28	-0.23	-1.75*
Impos	10	-5.10*	-52*	-0.33	+1.30
Pers	14	-5.89*	-14	-0.21	-4.22*

[a] all tasks except the impossible task produced at leastone zero responder
* p<.05 or better

DISCUSSION

The most important finding from the results of this experiment was that IBI was slower during the second part of the experimental session, and, as predicted, deceleratory PWV change was found during tasks in the second part of the experimental session. That finding provides some support for the hypothesized relationship between subject level of activation or behavioural coping and direction of PWV change.

Notably, the results from this experiment also replicated the major findings from Experiment 3. Accelerators and decelerators did not differ in their scores on any of the psychological scales used. Nor did they differ in any other physiological or behavioural measure tested.

The magnitude of IBI change continued to be determined by task difficulty, although the order of the magnitude changed: in Experiment 3 larger changes occurred during the easy task than during the impossible task, in the present experiment larger changes occurred during the impossible task than during the easy task. That finding can be interpreted to support the suggestion that easy and impossible tasks generate equivalent effects on IBI responses (Obrist, et al., 1978). However, to the extent that task demand or difficulty is seen as a primary determinant of IBI change, the finding emphasizes the need for more detailed specification of the nature of the relationship between environmental events and IBI acceleratory change.

Further, ingestion of caffeine was found to affect the resting levels of cardiovascular activity for every response except PWV. Both SBP and DBP were raised but, unexpectedly, IBI decelerated. The change in the response

constellation suggests that the dosage of caffeine ingested during the present experiment resulted in an increase in resting cardiac output through an increase in resting stroke volume. However, although caffeine ingestion enhanced both the IBI and DBP task-generated changes in highly specific interactions with task demands and subject variables, and was also associated with improved behavioural performance during the hard task, it did not alter the direction of the PWV change.

Thus although caffeine ingestion was associated with slower IBI, and with improved behavioural performance during the hard task in a manner consistent with previously reported findings and attributed to a change in subject activation or arousal (Revelle, et al., 1976; Revelle, et al., 1980), caffeine ingestion did not affect PWV changes. In addition, although task-generated responding during part 2 of the experiment session was dominated by a deceleratory PWV change which emerged from the analysis as the PWV response during that part, deceleratory changes were not uniform within tasks: invariably, both the acceleratory and the deceleratory PWV changes during all tasks were significant deviations from baseline levels. Thus, the results only partially supported the hypothesis. A further experiment was planned in an attempt to demonstrate uniform PWV change during task, in a direction predicted by resting HR level before task.

8 EXPERIMENT 5

Resting Cardiovascular Activity Over Five Sessions
and Response Change During a Novel and Demanding Task

INTRODUCTION

The experiment to be reported was designed to demonstrate a relationship between a change in HR during initial baselines over several sessions and task-generated change in PWV.

Subjects attended five experimental sessions. On the first session, half the subjects were advised that they would be required to complete a demanding problem solving task based on an intelligence test on the fifth day. It was predicted that, for these subjects, HR should increase over sessions in anticipation of the task. If that was the case, and HR during the initial baseline on session 5 was faster than the HR during the initial baseline on session 1, then PWV change during the task on session 5 should be acceleratory. But if the novelty of the experimental situation on the first session was a more powerful determinant of HR (Obrist, 1981) then multiple exposures to the laboratory should lead to a decrease in HR during the initial baseline on session 5 relative to session 1 and a deceleratory PWV change during task.

As in Experiment 4, IBI, PTT, SBP, DBP and RPI were recorded and PTT was converted to PWV (Avolio, et al., 1983; see Chapter 6). IBI was converted to HR and in addition, a calculated value of LVET was obtained from the recorded

values of RPI and PTT. The rationale and details of that calculation are presented in Appendix 2. As the LVET response formed part of the response constellation reported, the RPI data from this experiment is not included in Appendix 2.

METHOD

Subjects

Fourteen men and 22 women undergraduate psychology students volunteered to participate in the experiment. Data from one woman was excluded due to extreme values. They are presented as single subject data in the next chapter.

Apparatus and Physiological Recording

PTT, IBI, SBP and DBP were recorded as described in Experiment 4.

In addition RPI was recorded as described in Appendix 2, using the apparatus and recording elaborated in Experiment 1. The pulse detected at the auricular arterial site was fed into channel 1 of a Gould dual channel oscilloscope (OS 4000), with sweep speed set at 0.5cm/sec. Each sweep of the oscilloscope was triggered by the R-wave of the EKG (see Experiment 1). The time interval between the R-wave of the EKG and the pulse arrival at the auricular artery was digitized by the oscilloscope and output to the computer.

PTT was converted to PWV as described in Chapter 6.

IBI was converted to heart rate (HR) in beats-per-minute (bpm) on every collected beat according to the formula: 60000ms/IBIms=HR (bpm).

In addition, a measure of Left Ventricular Ejection Time was calculated as follows (also see Appendix 5). The distance between the manubrium sternum and the arterial site on the subject's right foot was divided by the calculated PWV to give an estimate of the actual time required for the pulse to travel the full distance between the aortic arch and the dorsalis pedis arterial site. That value was subtracted from the sum of PTT and the measured R-wave to pulse interval (RPI) to give an estimate of LVET which also included the isovolumetric contraction period of the cardiac pre-ejection period (PEP).

Otherwise the apparatus and physiological recording were as in Experiment 4.

PROCEDURE

Each subject attended 5 sessions spread over 14 days with each attendance at the same time of day (+/- 2 hours). Subjects each selected their own preferred times.

On every session, each subject signed an informed consent form upon arrival at the laboratory, then entered the sound attenuated, temperature controlled experimental room. As in the previous experiments all instructions and communications relating to the experiment were presented to the subject via the VDU.

Subjects were alternately assigned to either a Task or a No Task condition in order of arrival at the laboratory, ensuring that the numbers in each condition were balanced on the variable of sex. There were 7 men and 11 women in the Task condition; 7 men and 10 women in the No Task condition.

On first arrival at the laboratory, all subjects were verbally informed that the experiment was primarily concerned with the collection of resting levels of cardiovascular activity over successive occasions. Those subjects in the Task condition were also informed that on the fifth day they would be asked to complete a difficult problem solving task which was based on the Nufferno Intelligence Tests (Furneaux, 1956).

All five experimental sessions began with an instruction to "please sit quietly and relax" which was presented on the VDU screen while resting cardiovascular activity was recorded for 10 minutes. Those subjects in the No Task condition followed that procedure on all 5 days.

On the fifth day, following the rest period the subjects in the Task condition were presented with a 1-minute instruction period where instructions for the easy task from Experiments 3 and 4 were presented on the VDU screen. The instruction period was followed by a 4-minute task period and a 4-minute post-task recovery period. Except for their duration, these periods were identical in nature and content to the easy task and post-task periods in Experiments 3 and 4.

RESULTS

Beat-by-beat IBI, PTT and RPI were collected and converted to HR, PWV and LVET. Median values were obtained for each minute of each experimental session. Response change during task and pre-task instruction was calculated as described in Experiment 1 using the 10th minute of the rest period in the fifth session as the pre-task baseline. SBP and DBP measures were recorded as in the previous experiments on minutes 2, 5 and 9 of the rest periods and once

each during the instruction, task and post-task periods. Response change during task and pre-task instruction was calculated as described in Experiment 2 using the 9th minute of the rest period in the fifth session as the pre-task baseline.

Analyses of Absolute Levels

The absolute levels of HR, PWV, LVET, SBP and DBP collected from all subjects over all 5 10-minute rest periods were analysed for evidence of sex differences, tonic anticipation of task over sessions, comparative levels on different sessions and changes in levels within each rest period.

The absolute levels of HR, PWV, LVET, SBP and DBP collected from subjects in the task conditions during instruction, task and post-task periods were analysed for sex differences only.

Rest Periods

The absolute levels of HR, PWV and LVET from the 10-minute rest periods were analysed using an ANOVA with factors of Sex (2) x Task/No Task Condition (2) x Session (5) x Minute (10) with repeated measures on the last two factors. The absolute levels of each measure of SBP and DBP were analysed using a Sex (2) x Task/No Task Condition (2) x Session (5) ANOVA with repeated measures on the last factor only.

There was a Main Effect of Sex ($F(1/31)=4.14$, $p<.05$) for resting levels of HR. Women had faster HR than men (see Table 5.1).

There was a Main Effect of Session (F(4/124)=2.96, p<.02) for resting levels of HR. HR increased from session 1 to session 5 (72.23bpm, 71.96bpm, 73.37bpm, 75.99bpm, 75.33bpm).

There was a Main Effect of Minute for resting HR (F(9/279)=11.29, p<.0001) which reflected a general deceleratory response over each 10-minute rest period.

There was a Condition X Session X Minute Interaction found for resting HR (F(36/1116)=1.58, p<.02). HR increased over sessions for subjects in the Task condition, but not for those in the No Task condition. In addition, HR increased during the 10-minute rest period in session 5 for subjects in the Task condition, but not for subjects in the No Task condition. That interaction is shown in Figure 5.1.

There was a Main Effect of Sex (F(1/31)=9.52, p<.004) for resting levels of PWV. Women had slower PWV than men (see Table 5.1).

There was a Main Effect of Minute (F(9/279)=1.95, p<.05) for resting levels of PWV which reflected a general deceleratory response over each 10-minute rest period.

There was a Main Effect of Sex (F(1/31)=12.43, p<.001) for resting levels of LVET. Women had faster LVET than men (see Table 5.1).

There was a Sex x Condition x Minute Interaction (F(9/279)=2.22, p<.02) for resting levels of LVET. Men who were expecting a task on session 5 had faster LVET during the rest period than men who were not. There was no difference in resting levels of LVET for women who had task relative to women who did not,

and the women had faster LVET than the men in either condition. That interaction is shown in Figure 5.2.

There was a Main Effect of Sex found for all three measures of resting SBP ($F(1/31)$=25.61, 19.16, 29.55, all $p<.0001$). Women had lower SBP than men (see Table 5.1).

TABLE 5.1

Mean resting levels of Heart Rate (HR), Pulse Wave Velocity (PWV), Left Ventricular Ejection Time (LVET), averaged over five 10-minute rest periods, and the means of each of the three recordings of Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) averaged over the 5 sessions for men and women. A Main Effect of Sex was found for all comparisons except those involving DBP.

Response	Men (n=14)		Women (n=21)
HR (bpm)	68.73	*	77.14
PWV (m/s)	12.83	*	11.14
LVET (ms)	183.98	*	162.53
SBP1(mmHg)	119.89	*	105.04
SBP2(mmHg)	116.97	*	102.66
SBP3(mmHg)	118.70	*	103.29
DBP1(mmHg)	69.29		72.75
DBP2(mmHg)	67.69		72.70
DBP3(mmHg)	68.84		72.88

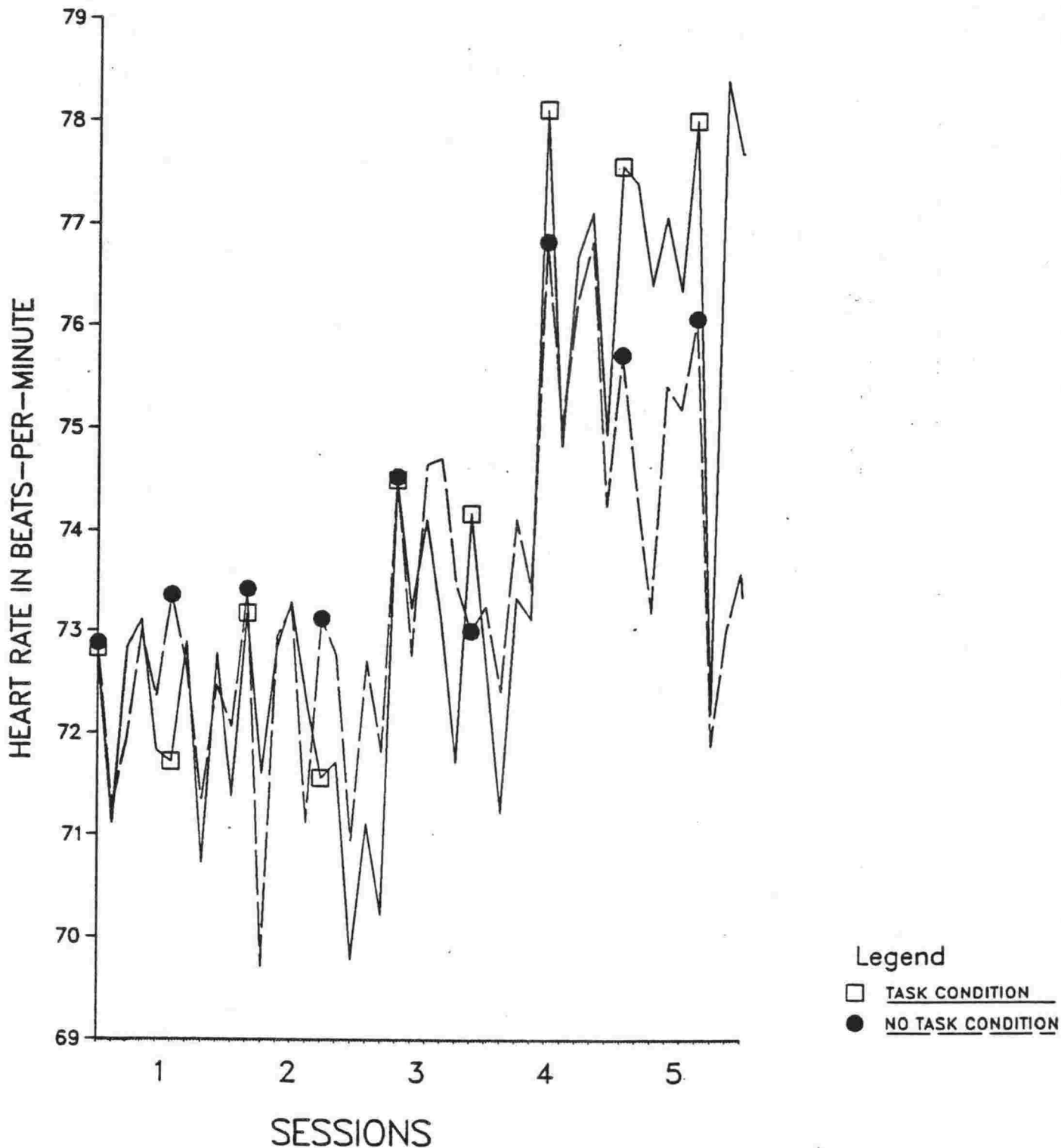* $p < .05$ or better

HEART RATE RESPONSE DURING REST

Figure 5.1 Anticipatory Heart Rate Response during 10-minute rest periods in 5 sessions, showing the Condition X Session X Minute Interaction reported in the text

Analyses of Response Change

Response change during instruction, task and post-task periods was calculated as change from the final minute of the rest period.

Instruction Period

Response change for all measures during instruction was analysed using an ANOVA with a factor of Sex (2). There were no significant effects found.

The magnitude of instruction-generated changes was compared to baseline levels using a t-test comparison for paired means. There were significant acceleratory changes HR, LVET and PWV (see Table 5.2).

Task Period

Task-generated changes in HR, PWV and LVET responses were analysed with a Sex (2) x Minute (3) ANOVA with repeated measures on the last factor. Task-generated changes in SBP and DBP were analysed using an ANOVA with a factor of Sex (2). In addition, the magnitudes of all response changes from baseline were evaluated using a t-test for paired mean comparisons.

There was a Main Effect of Minute found for HR ($F(2/32)=51.85$, $p<.0001$). The HR response peaked on minute 1, then stabilized.

There was a Main Effect of Minute found for PWV ($F(2/32)=4.73$, $p<.02$). The PWV response peaked on minute 2.

There was a Main Effect of Minute found for LVET ($F(2/32)=4.82$, $p<.01$) reflecting an acceleratory peak at minute 2.

All task-generated changes were signficant accelerations relative to baseline (see Table 5.2).

Post-task Period

Post-task recovery in HR, PWV and LVET responses were analysed with a Sex (2) x Minute (3) ANOVA with repeated measures on the last factor. Task-generated changes in SBP and DBP were analysed using an ANOVA with a factor of Sex (2). In addition, the magnitudes of all response changes from baseline were evaluated using a t-test for paired mean comparisons.

There were no significant effects. No post-task responses were significant deviations from baseline (see Table 5.2).

Table 5.2

Response change from pre-task baseline during instruction, task and post-task periods for all subjects. Response change was compared to baseline using t-tests for paired means comparisons. The significance of deviation is indicated. Responses are Heart Rate (HR), Pulse Wave Velocity (PWV), Left Ventricular Ejection Time (LVET), and Systolic (SBP) and Diastolic (DBP) Blood Pressures. (Inst.=instruction period; Task=task period; Post.=post-task period).

	HR	PWV	LVET	SBP	DBP
	All Subjects in Task Condition (N=18)				
Inst.	+ 5.78*	+0.33*	-4.17*	+1.17	+1.33
Task	+10.67*	+1.00*	-7.19*	-6.28*	+5.28*
Post.	- 1.00	+0.11	-1.17	+1.94	-0.89

* p<.05 or better

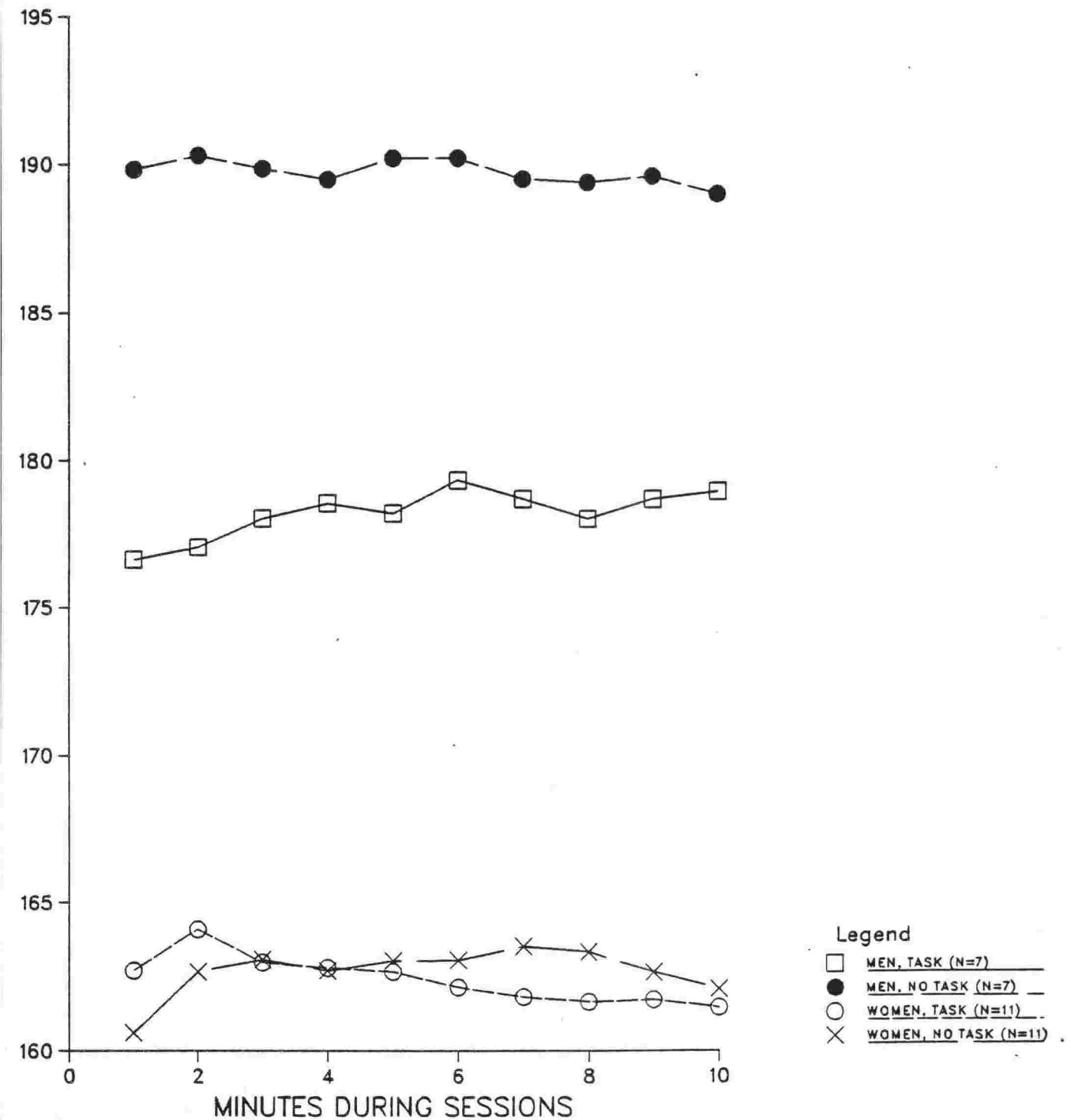EFT VENTRICULAR EJECTION TIME RESPONSE DURING REST

Figure 5.2 Left Ventricular Ejection Time Response during 10-minute rest periods over the 5 sessions, showing the Sex X Condition X Minute Interaction reported in the text

Behavioural Task Performance

Problem solving performance was scored as described in Experiment 1 and converted to percentage measures.

Relationship between Task Performance and Response Change

Pearson Product Moment correlation analyses were used to examine the relationship between task performance and associated changes in cardiovascular responses. There were no significant effects.

Task Performance and Sex

An ANOVA with a single factor of Sex (2) was performed on the behavioural task measures. There were no significant effects (see Table 5.3).

Response Direction

No deceleratory changes were significantly different from pre-task baseline level for any cardiovascular response. The frequencies of acceleratory and deceleratory changes are shown in Table 5.4.

TABLE 5.3

Behavioural task performance for men and women shown as percentage of total problems presented.

		Men (n= 7)	Women (n=11)
Easy Task	Correct	15.85	24.69
	Omitted	76.97	66.29
	Wrong	7.18	9.02

*   p<.05 or better

Table 5.4

The frequency of task-generated acceleratory and deceleratory changes for men and women, and for the total subject group. No deceleratory changes were significantly different from pre-task baseline levels, therefore the actual response changes are not presented.

	Acceleratory Response Frequency				
	HR	PWV	LVET	SBP	DBP
Women	10	10	8	11	8
Men	7	5	6	5	6
Total	17	15	14	16	14

	Deceleratory Response Frequency				
	HR	PWV	LVET	SBP	DBP
Women	1	1	3	0	3
Men	0	2	1	2	1
Total	1	3	4	2	4

DISCUSSION

The results confirmed the prediction that an increase in HR in anticipation of an impending task would be associated with acceleratory PWV change during task. That finding, considered in conjunction with the Experiment 4 results, supported the hypothesis that the internal physiological state, described as activation and reflected in changes in resting HR (Malmo, 1959), is a determinant of the directional variability in transient PWV changes.

Given the physiological significance of PWV, the results from Experiments 4 and 5 have some implications for stress-related cardiovascular dysfunction. They suggest that, given a lower activation level, acceleratory cardiac and blood pressure changes are apparently associated with a reduction in cardiac load during work, providing evidence of the beneficial effect of work on the cardiovascular system; but if there is a higher activation level, then acceleratory cardiac and blood pressure changes are associated with an increase in cardiac load, providing evidence of the detrimental effect of the same work on the cardiovascular system (O'Rourke, 1982; Avolio, et al., 1983).

The sensitivity of HR to anticipation of a task was again demonstrated in the results from the present experiment. That phenomenon supports the use of changes in HR as a measure of subject engagement or behavioural state and, as described in Chapter 6, the increase in anticipation of the task on session 5 of the present experiment is associated with an increase in the relative resting sympathetic tone on the cardiovascular system, in particular the myocardium. When novel and difficult tasks were presented to subjects in Experiment 1, each produced both acceleratory and deceleratory PTT changes in association with acceleratory IBI changes. When a novel and difficult task was

presented to subjects in the present experiment it generated uniformly acceleratory PWV changes in association with acceleratory IBI changes. The primary difference between the two experiments was in the manipulation of subject anticipation of the task. Overall the results support the hypothesis put forward in Chapter 6 that an anticipatory increase in resting HR prior to a task is likely to be associated with an increase in resting sympathetic tone and a pre-disposition to a rapid, predominantly neural response which results in an over-riding vasoconstriction and an acceleratory PWV change.

In addition, the inclusion of LVET in the measured responses provided information on changes both in sympathetic influences on the myocardium (Newlin, 1981; Pollack & Obrist, 1983) and in cardiac contractility (Berne & Levey, 1977; see also Appendix 2), with a reduction in LVET indicative of an increase in sympathetic influence and cardiac contractility. Thus the acceleratory LVET response suggests that an anticipated novel and difficult task will produce increased sympathetic activity on the myocardium, resulting in an increase both in HR and in cardiac contractility, and therefore an increase in cardiac output is also likely (Berne & Levey, 1977; Rushmer, 1976), again supporting the hypothesis presented in Chapter 6.

The uniformly acceleratory PWV changes recorded are indicative of increased vascular impedance and associated cardiac load. It is suggested that the changes reflect increased arterial rigidity through vasoconstriction of the vessel luminal diameter mediated by sympathetic neural activity and that an increase in cardiac output is also a contributing factor. It is further suggested that the uniformity of the direction of the changes is due to the increased activity in particular responses being superimposed on a relatively

high level of resting sympathetic tone, indicated by the increase in level of resting HR and consistent with a more active behavioural state, which was generated by the anticipation of a difficult task.

The second notable result from the current experiment was that there were sex differences in resting levels of every measure of cardiovascular activity except DBP. In particular, the men had faster PWV and higher SBP, but slower HR and LVET than the women. Faster PWV has been associated with higher blood pressure levels in epidemiological studies (Avolio, et al., 1983) and, to the extent that tonic levels of cardiovascular activity contribute to the predisposition of the male to cardiovascular dysfunction, it seems likely that arterial compliance is a more critical contributor than cardiac rate (Berne & Levey, 1977). Some additional support for that contention was provided during the adventitious recording of response constellations of cardiovascular activity from one subject before, during and after an emotional crisis. That case study is presented in the next chapter.

9   SINGLE SUBJECT DATA

'PWV Changes During A Case of Acute Emotional Distress'

INTRODUCTION

In the execution of Experiment 5, adventitious measurements were obtained from one subject immediately before, during and after an emotional crisis. The extraordinary values of PWV recorded resulted in the exclusion of her data from the report on Experiment 5. However, the nature of the attendant circumstances has led to the presentation of her data here.

METHOD

Subject

The subject of interest, EP, was a married woman with a school-age family who was undertaking part-time university studies in addition to holding down a week-end job as a night nurse to a Parkinson's patient. At the time that she attended the laboratory, she reported being physically fit, healthy and receiving no medication although suffering from pre-menstrual tension which was a regular occurrence for her.

Apparatus and Physiological Recording

As in Experiment 5.

PROCEDURE

As in Experiment 5.

EP was assigned to the task condition on her first attendance at the laboratory. She, like all other subjects in that condition, was told that she would be presented with a task on the fifth day. Because of the events reported here, EP was not actually presented with a task, but that omission was not noted by EP until approximately 4 months after recording was finished. All comparison data presented are those generated by the other subjects in the task condition, but only the resting levels of cardiovascular activity over 5 sessions are presented in the present report.

RESULTS

HR, PWV and LVET data were collected on a beat-by-beat basis and median values obtained for each minute of each experimental session. A mean value for each measure for each session was calculated. SBP and DBP were recorded as previously described during minutes 2, 5 and 9 of each rest period, and a single mean level for each session obtained.

The mean level of HR, PWV, SBP and DBP for subject EP on each of the 5 sessions is presented in Table 6.1. Equivalent data from the men and the women who participated in the task condition in Experiment 5 are also presented in that table for comparison.

Data Reliability.

Because of the extraordinary velocities recorded it is important to realize that the measurement procedure used enables the experimenters to make a direct check on the data by comparing the oscilloscope display with the PWV measure for that display. Even with HR in excess of 100bpm, the variance of measures of PWV is such that, when events give rise to large magnitude changes in PWV, the correspondence between the proximity of the two wave forms displayed on the oscilloscope and the values displayed on the computer can be verified for trend. In the present study, the data of critical interest were verified simultaneously by the experimenter and the supervisor and discussed critically so that every effort was made to ensure that, in terms of overall trend, what was seen visually on the oscilloscope conformed with the values displayed by the computer.

Behavioural Indices.

On session 3, when EP arrived at the laboratory she was in a state of extreme emotional distress in which she alternated periods of crying with attempts to bring herself under control. All attempts to persuade her to withdraw from the session met with vehement refusal and with insistence that the session be carried out as usual as she wanted to do whatever she could to help with the work of the experimenter. Her behavioural signs of anguish were those which might be expected to be associated with the news that one of the children had been run over.

Information was subsequently offered by a concerned neighbour of EP's who drove EP to the laboratory that, as she understood it, a severe emotional

crisis had developed in EP's life and had peaked on the evening preceding session 3.

Faced with EP's adamant insistence that she continue to participate in the experiment, professional clinical advice was sought and, following that, it was agreed that EP's participation could continue providing she would allow herself to be taken to the student counselling service immediately the session ended.

EP returned of her own volition to complete her remaining sessions. On both sessions 4 and 5, she continued to show agitation and pronounced physical tension, but not by any means of the order she showed in session 3. Notably, she was still receiving counselling 6 months after recording had been completed.

Physiological Indices.

At no time was there any difficulty in obtaining clean EKG or pressure pulse wave forms from EP, although there was a tendency for her to rub her feet together. Her feet were slightly cold, but no colder than experience has shown is the norm for women subjects who have been wearing nylon stockings and light shoes immediately prior to a laboratory session. The overall impression of her skin temperature was that it was normal. However, she was extremely tense physically.

It is important to note that there was no evidence that EP was in a state of shock. There were no signs of reduced tissue perfusion due to reduced cardiac output; the skin was not sweaty nor clammy, and no difficulty was experienced in detecting the peripheral pulses, indicating normal levels of vasoconstriction in the peripheral beds where pulse detection occurred.

TABLE 6.1

The mean response levels on each session for EP and for the men (M, n=7) and women (W, n=11) who participated in the task condition during Experiment 5.

	PWV (m/sec)			HR (bpm)			SBP (mmHg)			DBP (mmHg)			LVET (ms)		
Day	EP	W	M	EP	W	M	EP	W	M	EP	W	M	EP	W	M
1	13	11	12	83	77	65	120	105	116	74	73	71	158	161	175
2	14	11	12	92	74	68	123	101	114	68	70	68	153	169	181
3	36	11	11	111	76	68	137	106	122	77	73	69	164	156	169
4	16	11	12	102	81	69	133	103	123	74	71	65	131	166	178
5	19	11	13	87	81	73	137	104	124	84	73	67	161	160	189

DISCUSSION

The extreme value of PWV recorded during session 3 indicates a huge reduction in arterial compliance associated with a massive increase in cardiac load (Avolio, et al., 1983). As measured in the present study, PWV reflects overall changes in the systemic vasculature, and the vascular bed of major change is not specified.

The physiological indices showed no external manifestation of an excessive increase in peripheral resistance. There was no difficulty in registering the arterial pulses, their amplitude was not exceptional and EP's skin was not unusually cool or discoloured. Thus there appears to be no physiological evidence of increased constriction in peripheral vessels.

Of the other cardiovascular measures obtained, HR had a relatively large acceleratory change, but there were only moderate increases in BP and a deceleratory change in LVET, suggesting that cardiac output remained essentially constant, but with a diminished stroke volume. The interdependency of PWV and BP described in Chapter 1 further suggests that a much larger increase in BP than that actually seen would be required to achieve the remarkably large increase in EP's PWV which was observed during session 3. In addition, EP's mean pulse pressure, calculated for each day from the available SBP and DBP data (46, 55, 60, 59, 53), is suggestive of relatively rigid arterial walls from day 2 on (Berne & Levey, 1977; O'Rourke, 1982). Thus the evidence supports a reduction in arterial compliance, but not primarily in the peripheral vasculature. It seems most likely that the primary effect was on the walls of the large central and limb arteries, with increased neural and humoral sympathetic stimulation responsible for increased active muscle tension

on the smooth muscle cells in the walls of the large arteries, reducing their luminal diameter, and hence their compliance, resulting in the manifest acceleratory change in PWV.

An equally notable feature of the observed data is the pattern of cardiovascular activity obtained during the last two sessions that EP attended the laboratory. PWV returned to an apparently stable, but still high level; HR similarly returned to a level, not abnormally high, but still high enough to suggest quiescence had not been obtained; LVET accelerated then decelerated; SBP remained high and DBP showed signs of increasing. Thus in terms of the final two sessions, there are indications that there may be developing, in EP, a cardiovascular pattern suggestive of essential hypertension.

10 GENERAL DISCUSSION

The present research addressed the relationship between cardiovascular responding and behaviour, with particular emphasis on directional variability of stimulus-generated cardiovascular changes. The response of primary interest was PWV which was initially examined in the context of active/passive coping with IBI recorded as the reference response.

9.1 MAIN FINDINGS

Experiment 1 manipulated task difficulty as a function of task type. Two tasks hypothesized to generate active coping (mental arithmetic and problem solving), and two tasks hypothesized to generate passive coping (simple reaction time and personal tempo) were presented to men and women subjects, while IBI and PTT were recorded. Task difficulty had no effect on PTT changes which were influenced by experimental session and task order only. These effects involved comparisons of the direction, not magnitude, of PTT change. In addition, directional divergence was found to be the major characteristic of PTT changes during tasks in that significant acceleratory and deceleratory changes occurred during evey task. Notably, accelerators and decelerators did not differ on any other variable, including sex, task performance or baseline levels of PTT or other cardiovascular responses. However, in contrast to PTT, the magnitude of IBI acceleratory change was determined by task difficulty. But the IBI change was not distinguished into two distinct modes of response as predicted by the active/passive hypothesis. Instead, each task generated an

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IBI change of a particular magnitude. Consistent with the active/passive hypothesis, the largest acceleration occurred during the active task at the moderate difficulty level, and the smallest accelerations occurred during the two passive tasks. Notably, tasks categorized as active tasks generated a greater frequency of acceleratory IBI changes than did the two categorized as passive tasks, which also generated deceleratory IBI changes.

As noted above, both acceleratory and deceleratory PTT changes occurred on every task and therefore a proportion of subjects had acceleratory IBI changes indicative of active coping, but associated with a deceleratory PTT change, indicative of a reduction in cardiac load.

In Experiment 2, the difficulty level of problem solving tasks was manipulated and subjects completed problem solving tasks at two levels of difficulty. Directional variability was confirmed as the major characteristic of the PTT response during tasks: acceleratory changes were significant during both tasks, deceleratory changes were significant during the more difficult task. During the easier task, approximately half the subjects had a deceleratory change. The lack of statistical significance of that change appeared to be the result of between subject variability in magnitude of change, rather than in its size per se, or its frequency of occurrence. Once again accelerators and decelerators did not differ in measures of sex, task performance, or baseline activity, or accompanying response change in other measures. Nor was their direction of change during tasks in Experiment 2 predicted by their direction of change during problem solving in Experiment 1. In addition, the results showed that, consistent with Light & Obrist's (1983) report, problem solving tasks at two levels of difficulty (easy and hard)

generated equivalent changes in IBI, but the magnitude of SBP, and not DBP, changes was determined by task difficulty. In contrast to PTT, there were no deceleratory IBI changes, confirming the results from Experiment 1: firstly, that task type determined the frequency of IBI acceleratory changes, and secondly, that acceleratory IBI changes congruent with active coping can nonetheless be associated with a reduction in cardiac load.

Experiment 3 was an extension of Experiment 2. Its purpose was to begin an exploration into the relationship between psychological variables and the direction of PTT change during task. Men and women subjects completed the STAI, the EPI, the SACL and the JAS before participating in problem solving tasks at three levels of difficulty and a personal tempo task. As in the previous experiments, acceleratory and deceleratory PTT changes were significant during every task, and task difficulty did not affect PTT change. But an attempt to relate PTT directional variability to scores on the psychological measures used was unsuccessful. However, as in Experiment 1, each task generated acceleratory IBI responses which were specific to the task, and determined by task difficulty. But, as in Experiment 2, and consistent with Light & Obrist (1983) who reported equivalent changes in a variety of cardiac responses, IBI changes during the easy and hard problem solving tasks were more nearly equivalent than those during the easy and impossible tasks. That finding was considered to emphasize the need to examine the effect of task contingencies on cardiac changes. Consistent with Experiments 1 and 2, problem solving tasks generated only acceleratory IBI changes, but the personal tempo task generated both acceleratory and deceleratory IBI changes. As with PTT, all four tasks generated both acceleratory and deceleratory SBP and DBP changes. It was noted that the increased frequency and magnitude of

deceleratory BP changes could be adequately accounted for in terms of increased familiarity with both the experimental situation and the measurement procedures, but that the directional variability of the PTT response continued to be unexplained by any measured variable including scores on the psychological tests and previous performance. Elaboration of the determinants of PTT or PWV response directional variability continued to be the most important focus for investigation.

In Experiment 4, PTT was converted to PWV to reduce between-subject variability, and a further exploration into the determinants of PWV response directional variability was initiated. It was hypothesized that subject behavioural state prior to task, measured by relative HR during the initial baseline could be a contributing factor in the direction of task-generated PWV change. The results gave partial support to the hypothesis. They demonstrated that a reduction in HR during baseline in part 2 relative to part 1 of the experimental session was associated with deceleratory PWV change during tasks, but a caffeine induced reduction in HR during baseline in part 2 of the experimental session was not.

The hypothesis that the subject's behavioural state prior to task may help determine the direction of PWV change during task was further tested in Experiment 5. Specifically it was predicted that if the level of resting HR increased over successive baseline periods in anticipation of a task, then the PWV change during task should be acceleratory. If, however, the level of resting HR decreased over successive baseline periods due to familiarity with the experimental situation then the PWV change during task should be deceleratory. The results from Experiment 5 showed that an increase in HR

during baseline from session 1 to session 5 was associated with an acceleratory PWV change during the first exposure to a problem solving task in the fifth session. The acceleratory PWV change was part of a uniformly acceleratory change in a constellation of responses which included HR, LVET, SBP and DBP.

The uniform acceleratory changes are noted to be consistent with an account of increased sympathetic activity both on the vasculature and on the myocardium. Those results support the suggestion that the initial behavioural state of the subject interacts with the environmental demands to produce changes in cardiovascular responding during tasks.

It is considered that the laboratory manipulation of demanding tasks is a simulation of life events which may generate similar cardiovascular changes. Therefore the findings from the laboratory may be extrapolated from the laboratory to life events, and a link between life events and stress-related cardiovascular disease may be postulated. The adventitious recording of resting cardiovascular activity from a subject before, during and after she suffered a severe emotional crisis showed large magnitude PWV acceleration, indicating an enormous increase in cardiac load during the peak crisis time, and thus provides some support for the practice of extrapolating from the laboratory to the field, and for the postulated link between life events and stress-related cardiovascular disease.

But EP's data also highlight the disparity between the changes in cardiovascular responses obtained during research, and those which can occur in response to significant life events. In particular, during the present research, a change of 1m/sec was statistically signficant and found to be worthy of comment. In comparison, EP had a change of 23m/sec from day 1 to day

3. Clearly, ethical considerations limit the degree of aversive stimulation which can be applied in psychological research and it is unlikely that a change of that magnitude would be readily replicated in the laboratory. EP's data suggest that the limits on the data generated in the laboratory should be explicitly acknowledged. Initiatives to measure, or take into account, responding external to the laboratory already occur (e.g. Light & Obrist, 1980a) particularly in the biofeedback or clinical area when dealing with established dysfunction (e.g. Glasgow, Gaardner & Engel, 1981). It seems likely that, as technology advances, field studies on the relationship between significant life events and cardiovascular responses may become more prevalent.

In addition, EP's data identify PWV as a measure of physiological significance with particular relevance to stress-related changes in cardiovascular functioning. Except for LVET, the resting level accelerated in all of EP's response measures, with IBI and SBP having large increases during her crisis. Notably, DBP did not really begin to increase until the crisis peak was over, and LVET actually decelerated during the crisis suggesting a decrease in cardiac contractility. The magnitude of the change in PWV was huge, it occurred during the period of most acute crisis for EP, and it was followed by the more commonly reported increases in BP, supporting the possibility that increased arterial rigidity may be a critical contributing factor in the etiology of high blood pressure, or essential hypertension (Berne & Levey, 1977). As noted in Chapter 1, PWV has been found to be a measure which is useful in the early detection of arterial degeneration in various disease states, it may yet be found to be a measure which is also useful in the early detection of stress-related cardiovascular dysfunction.

Another finding of some importance was the prevalence of sex differences in resting levels of cardiovascular activity. The women were found to have consistently lower resting SBP than the men in Experiments 3, 4 and 5; and to have slower resting PWV than the men in Experiments 4 and 5. Further, in Experiment 5, the women had faster resting HR and LVET than the men, and thus men and women differed on every measure of resting cardiovascular activity except DBP,

In addition, in Experiment 4 PWV was unique in that it was unaffected by caffeine ingestion. IBI slowed, and both SBP and DBP were raised following caffeine consumption. The combined pattern of responses suggests that the increase in BP could be mediated by an increase in cardiac output through increased stroke volume.

9.2 MEASUREMENT ISSUES

The present research is unusual in the psychophysiological literature both in the use of continuous data sampling, and in the use of repeated measurements.

9.21 Continuous Data Sampling

Continuous sampling on a beat-by-beat basis over an extended period of time as utilized in the present research is a practice which has only recently been achieved through advances in technical facilities and computer-based data collection and analysis. Continuous sampling of cardiovascular activity as implemented for IBI and PTT in the present work, is recognized as the ideal (Ax, 1953; Black, 1974; Dutch & Redman, 1983; Greenfield & Sternbach, 1972;

Venables & Christie, 1981) in that the data obtained are an extremely sensitive
representation of response levels and changes. Discrete sampling ignores most
of the cardiovascular activity, and the accuracy of its representation is
dependent on the experimenter timing the collection point appropriately. The

9.3 PWV RESPONSES AND BEHAVIOURAL COPING

Systemic PWV is a noninvasive measure of physiological significance which is of interest in its own right (Avolio, et al., 1983). Changes in PWV reflect changes in arterial compliance and therefore indicate changes in the processes and mechanisms that control significant aspects of cardiac and vascular performance in the maintenance of an appropriate arterial pressure head. Aspects of performance influenced can include cardiac rate, contractility and force, and vasomotor reactivity (see Chapter 1). In addition, as measured in the present work, changes in systemic PWV provide immediate information on changes in cardiac load. Acceleratory changes indicate increased cardiac load, deceleratory changes indicate decreased cardiac load (Avolio, et al., 1983; O'Rourke, 1982).

The results from Experiments 1-4 showed that task-generated deceleratory PTT changes, indicating a reduction in cardiac load, could accompany the routinely obtained acceleratory IBI changes. The reduced cardiac load could be mediated by a reduction in cardiac output through reduced stroke volume, or by reduced vascular impedance through vasodilation of arterial vessels. Alternatively, acceleratory PTT changes, indicating an increase in cardiac load, could accompany acceleratory IBI changes. The increased cardiac load could be mediated by an increase in cardiac ouptut through increased stroke volume, or by an increase in vascular impedance through vasoconstriction of arterial vessels.

The alternative which was considered to be best supported by the combined cardiovascular measures, including those of RPI (see Appendix 2), and the behavioural performance data, was that of a sympathetically mediated change in

vascular impedance. It was proposed that acceleratory and deceleratory PWV changes may represent equivalent, sympathetically mediated changes, the first reflecting alpha-adrenergic vasoconstriction mediated by neural activity, the second reflecting beta-adrenergic evoked vasodilation predominantly mediated by humoral activity. That argument is elaborated in the following section (9.31)

However, as described in Chapter 1, PWV is determined by a complex interaction between transmural pressure, blood volume, vessel luminal diameter and vessel elasticity. To date there is no quantification of the degree to which each component contributes to any specific manifest change under particular conditions.

The results from Experiments 1, 2 and 3 showed that neither task difficulty, novelty nor type were determinants of the direction of change in PWV. It was noted that the Task Order Effect during tasks in Experiment 1, and the Sex Effect during instructions in Experiment 3 both reflected differences in direction of PWV change, not a difference in magnitude. These findings were further considered in the context of Experiment 1 as the most stressful laboratory experience (Obrist, 1981), and the superior behavioural performance demonstrated by women in Experiments 2 and 3 as suggesting an influence of occasion specific subject variables. It was hypothesized that the subject's internal physiological condition prior to task, described as the behavioural state associated with a particular level of engagement (Obrist, 1981), or less specifically as the level of activation (Malmo, 1959), and measured by the relative level of resting HR across baselines, could be a determinant in the subsequent direction of PWV change during task.

That hypothesis was supported in that, in Experiment 4, a reduction in IBI from the baseline in part 1 to the baseline in part 2 of the experimental session was associated with a greater prevalence of deceleratory PWV changes, and that, in Experiment 5, an increase in HR during the initial baselines over 5 sessions was associated with acceleratory PWV changes during task on the fifth session.

The demonstration that the direction of PWV change can be predicted shows that the characteristic directional variability is not a random phenomenon. Overall, the present research reports prevalent and lawful changes within physiological limits (Wilder, 1958, 1962) which provide evidence for the noted beneficial effects on cardiovascular functioning of work or stress, as well as the more commonly reported detrimental effects (Selye, 1976). The results show that, on any occasion for any individual, the particular task requirements interact with the internal physiological conditions (Malmo, 1959; Duffy, 1972) or behavioural state (Obrist, 1981) of the individual to determine whether task participation will be associated with an increase or a decrease in cardiac load. Specifically, they indicate an association between a more active state and increased cardiac load, and an association between a less active state and a decreased cardiac load.

The data from EP allowed the hypothesis that increased arterial rigidity may be a critical contributing factor in the etiology of stress-related high blood pressure. During her crisis peak, EP's reponse pattern shows an acceleration in HR accompanied by deceleration in LVET suggesting that any change in cardiac output would have been insufficient to produce a 17mmHg increase in SBP, and certainly could not have generated a 2.6-fold increase in

PWV. Thus the acceleratory changes in PWV and in SBP are most likely attributable to a huge increase in arterial rigidity due to a sympathetically mediated general reduction in vessel luminal diameter.

It is suggested that EP's emotional crisis triggered a relatively general activation of sympathetic influences which resulted in rapid and sustained vasoconstriction throughout the vasculature, but probably particularly in the highly reactive peripheral beds. Data recorded over the subsequent sessions as the crisis subsided are consistent with a gradual reduction in sympathetic influence, which nonetheless appears to have remained high relative to the group. The gradual increment in DBP suggests that the sympathetic tone was primarily reducing on the secondary vessels and was either being sustained or increasing on the peripheral vessels. Thus peripheral resistance may have been increasing even as the rigidity of the larger vessels was diminishing.

Notably, on day 5, EP's HR and LVET approximated those of the comparison group of other women, but her PWV, SBP and DBP remained higher, reflecting the influence of arterial compliance on BP and suggesting that increased arterial rigidity was being sustained. As detailed in Chapter 1, to the extent that such an increase in arterial rigidity affected mechanoreceptoractivity it can be expected that increased sympathetic influences on cardiovascular functioning would also be sustained. As recording was then terminated it is not known whether the apparent increase in arterial rigidity has eventually become a chronic condition, but there is no doubt that physiological adaptation to increased arterial rigidity, both in mechanoreceptor activity and in cardiovascular functioning, could occur relatively rapidly (Berne & Levey, 1977; Rushmer, 1976; Gunn, et al., 1972). It seems likely that if high-stress

life events should be sustained for long, they could be powerful determinants in generating such a chronic condition.

The LVET data were notable both in their lability and in the inverse relationship which was apparent between changes in PWV and LVET over the days. In EP's case, except from day 1 to day 2, when PWV accelerated between baselines, LVET decelerated, and when PWV decelerated between baselines, LVET accelerated. As detailed in Chapter 2, an inverse relationship has previously been recorded between PEP and PTT (Pollack & Obrist, 1983) and also between RPI and PTT (Lane, et al., 1983). As described, LVET was calculated from the recorded values of RPI and includes the isovolumetric contraction period of PEP. The inverse relationship seems to be a robust phenomenon which may be an indication of complementary changes between vascular and cardiac activity. The nature of the relationship would be a topic for future investigation which could provide valuable information on the integrated functioning within the cardiovascular system.

EP's data have allowed a direct link to be proposed between hyper-acceleratory PWV from baseline to baseline, indicating an enormous increase in cardiac load during rest, and severe emotional distress or tension, as well as the further proposal of a possible link between stress-related chronic cardiovascular dysfunction. Those proposals further raise the question of whether a reverse response, i.e. hyper-deceleratory PWV and the reduction in cardiac load, could be associated with the lethargy of severe depression or the more dramatic stress-related sudden death syndrome (O'Rourke, 1982; Miller & DiCara 1967).

Hyper-acceleratory changes are likely to be more readily demonstrated (Gunn, et al., 1972). The present research supports an association between resting HR and PWV changes during tasks. In particular, acceleratory PWV changes during task were predicted by an acceleratory HR change over baselines in Experiment 5. Further, PTT acceleratory changes during the easy and hard tasks in Experiment 3 appeared to be associated with larger magnitude acceleratory IBI changes than PTT deceleratory changes were. It may be that HR-reactive individuals (Light & Obrist, 1980) or those already identified as at risk from hypertension (Redman & Dutch, 1983) will be found to have acceleratory PWV changes, particularly during tasks which are associated with active coping. However, it should be noted that the PTT deceleratory change during mental arithmetic in Experiment 1, and those during the hard and impossible tasks in part 1 of Experiment 4, were associated with the largest IBI acceleratory changes, suggesting that task familiarity or even content may need to be controlled. In addition, the results from the present research show that the subject's relative behavioural state on the occasion should also be taken into account before the direction of PWV changes can be predicted. Therefore it is likely that any demonstrated relationship between HR reactivity and direction of PTT would be complex, but could result in the early detection of individuals at risk for stress-related cardiovascular disease and an increase in the understanding of the mechanisms which underlie some of those clinical conditions.

9.31 PWV Directional Variability: Possible Mechanisms

Over the first four experiments all acceleratory and deceleratory PTT changes, except the deceleratory change during the easy task in Experiment 2, were significant deviations from pre-task baseline. However, accelerators and decelerators did not differ in sex, baseline activity, behavioural performance, or psychological measures.

In the absence of invasive measures, consideration of physiological response mechanisms must be speculative, but nonetheless various alternatives should be considered (Obrist, 1976). As described in Chapter 1, transient changes in PTT reflect sympathetically mediated changes in arterial rigidity corresponding to changes in vessel luminal diameter relative to arterial blood volume and thus can reflect a change in cardiac output, a change in vessel calibre, or both. The observed PTT changes could involve both reflexive or passive changes as well as more direct, active ones. Firstly, the possible influence of the baroreceptor reflex on the deceleratory PTT change was considered. Changes in the stretch of the arterial wall in which the receptor is sited trigger the discharge of impulses which inhibit the vasoconstrictor centre in the medulla and excite the vagal centre, resulting in peripheral vasodilation and a decrease in both heart rate and contraction strength. However the IBI data are inconsistent with an interpretation which proposes a major contribution from the mechanoreceptor reflex, in that PTT accelerators and decelerators did not differ significantly in their IBI responses.

The smooth muscle reflex of the aorta underlies a direct relationship between changes in aortic arterial compliance and venous return (Nicolosi & Pieper, 1971). A greater venous return results in a greater aortic compliance

associated with a reduction in characteristic impedance and a deceleratory PWV change (Stone & Dujardin, 1981). The reflex, which can be prevented by alpha-adrenergic blockade, may be an important mechanism through which the aorta contributes to transient changes in PTT, as that vessel is less subject to other, more active, influences. In addition, the reflex may occur more generally throughout the vasculature in that peripheral vasodilation results in increased venous return and hence increased cardiac output (Guyton, 1981).

It seems likely that both the acceleratory and deceleratory PTT changes may involve active changes which are probably sympathetically mediated (O'Rourke, 1982; Guyton, 1981). Acceleratory PTT changes are likely to reflect sympathetic activation, in particular enhanced sympathetic neural influences on the alpha-adrenergic receptors throughout the vasculature, especially in the peripheral beds, decreasing vessel radius through vasoconstriction and increasing arterial rigidity and peripheral resistance. Deceleratory PTT changes may be a function of competing excitatory and inhibitory vasomotor neural impulses, or alternatively, they may be some combination of local vasodilatory influences in competition with centrally mediated vasoconstriction (Cox, 1979). But the relative characteristics of the deceleratory response appear to be indicative of a slower, longer lasting, humoral response, involving the release of epinephrine and associated with beta-adrenergic vasodilation and increased cardiac output. That interpretation appears to be the most consistent with the data, and attributes the directional variability to a difference in either the degree of the response, or its dominant components, rather than requiring a postulation that fundamentally distinct types of response are generated by similar conditions either within individuals on different occasions, or by different individuals on a single occasion.

9.4 IBI RESPONSES AND BEHAVIOURAL COPING

The combined results from Experiments 1, 2 and 3 suggest that if tasks are placed along a continuum of difficulty with a priori no demand at the lowest extreme and an impossible task at the highest extreme, then the magnitude of acceleratory change in IBI will show a curvilinear relationship to task difficulty. The peak (or the trough) of the curve should occur at about the midpoint of difficulty; the height and slope of the curve are determined by specific task parameters, specifically task type and novelty although it is likely that the task consequences or contingencies (experimental paradigm) may also be influential. Thus these data suggest that active and passive coping may represent the maximum and minimum responses on a curvilinear function which relates the magnitude of IBI changes to task demand along a continuum of behavioural coping rather than representing two dichotomous states.

Over the first four experiments it was notable that during problem solving or mental arithmetic tasks virtually no subjects made deceleratory IBI changes, and no deceleratory IBI changes were signficantly different from pre-task baseline. But during the reaction time task in Experiment 1 and particularly during the personal tempo task in Experiments 1-4, approximately half the subjects made deceleratory IBI changes, and the change was signficantly slower than pre-task baseline. Thus task type, not task difficulty, appears to be the determinant of the frequency of acceleratory IBI changes.

The basic distinction between the states of active and passive coping is described as the subject's level of engagement or involvement (Light, 1981; Obrist, et al., 1978; Obrist, 1981). A greater engagement is reflected in larger magnitude, acceleratory IBI change.

In Experiments 1 and 2, the magnitude of the instruction-generated changes in IBI was determined by the difficulty of the task to which the instructions applied. In Experiment 1, task-generated IBI changes could be viewed as simply an enhancement of those found during the instructions; in Experiment 2, task-generated IBI changes eliminated the difference in magnitude associated with difficulty shown during the instructions. The anticipatory responding found during the instructions is more consistent with an interpretation of differences in a preparatory behavioural state, or anticipatory engagement, than with either an interpretation of stimulus intake/rejection (Lacey, 1967; Lacey & Lacey, 1974), or one of differences in associated somatic activity (Obrist, et al., 1970; Obrist, 1976). Similarly, in Experiments 4 and 5, the change in levels of IBI or HR during consecutive baseline recordings is most readily accounted for in terms of a preparatory behavioural state, or anticipatory engagement. Hence, the use of changes in IBI as a reflection of changes in subject engagement, or state of behavioural coping, received some support from the present research.

Overall the results suggest three modifications of the active/passive hypothesis. Firstly, that the relationship between task difficulty and the magnitude of IBI acceleratory changes is most appropriately described by a curvilinear function along a continuum of behavioural coping, and not by two dichotomous behavioural states. Secondly, that the relationship between task type and IBI changes may be most appropriately described by the frequency of task-generated acceleratory and deceleratory changes. Tasks designated by type as active tasks are more likely to generate acceleratory IBI changes than those designated as passive tasks which are more likely to also generate deceleratory changes. Thirdly, that acceleratory IBI changes congruent with active coping

are generated by problem solving at various levels of difficulty at least under non-avoidance contingencies.

That third point is suggested to direct attention towards the contribution that task contingencies make in manifest IBI responses. In Experiment 3 reported above, IBI changes during the easy and hard problem solving tasks, but not during the impossible task, were congruent with those generated during active coping. But in Experiment 4, IBI changes during the impossible and hard tasks, but not during the easy task, were congruent with those generated during active coping. It is possible that the degree of task familiarity with several difficulty levels helps to make easy tasks more interesting or impossible tasks appear easier and therefore generates greater subject engagement during both those levels of difficulty on particular occasions. Alternatively, it may be that task contingencies are particularly influential in determining the relationship between subject engagement and task difficulty and thus in the manifest IBI response. As discussed in Experiment 3, the relative contributions by each of task contingency and task demands need to be clarified in order to resolve the question of whether problem solving tasks at various difficulty levels are likely to generate large magnitude, sustained accelerations in HR regardless of task contingencies (avoidance/non-avoidance), or whether the use of an avoidance paradigm is a necessary condition for the demonstration of a unique HR response to hard tasks. Evidence produced by Light & Obrist (1980a) showed that avoidance conditions produce active coping, yoked control conditions do not. However, the active/passive coping hypothesis relates tonic HR responding to successful task performance, not specifically to the task contingencies. The present data indicate that a more suitable proposal might relate tonic HR changes to an interaction between task

contingency and task difficulty. Hence the effect of task contingency on HR responding, particularly over multiple occasions, was identified as requiring further study in the future.

9.5 BP RESPONSES AND BEHAVIOURAL COPING

The SBP and DBP changes during tasks were, like those of IBI and PWV, very distinctive. The magnitude of SBP changes was, like IBI, determined by task difficulty, and both measures of BP were sensitive to the caffeine manipulation in Experiment 4.

In Experiments 2 and 5, only acceleratory changes in SBP and DBP were found during tasks; in Experiments 3 and 4, both acceleratory and deceleratory SBP and DBP changes occurred during all tasks. The incidence of acceleratory changes in Experiments 2 and 5 is therefore likely to be partly attributable to novelty, both of the measurement itself and of the experimental situation (Obrist, 1981; Redman & Dutch, 1983). The evidence suggests that more intensive study of the determinants of the direction of BP change is required.

9.6 SEX DIFFERENCES

It was notable that no systematic sex differences in task-generated responding were found. The specific interactions involving sex which were observed and reported were not replicated across experiments, and their reliability requires testing. The data suggest that sex differences in cardiovascular responding during tasks are either not invariant, or that they are not robust enough to dominate response patterns.

A consistent sex difference was found in the resting levels of cardiovascular activity, particularly SBP and PWV. That difference is considered to be most clearly demonstrated in Experiment 5, suggesting that repeated measurements are necessary for its manifestation. It seems likely that the difference in resting level of cardiovascular activity may persist from day to day under routine conditions, and may be particularly relevant to identifying the mechanisms which predispose the male to cardiovascular disease. In that regard, the difference between men and women in PWV suggests that the men have less compliant arterial walls than the women, with implications for all of the processes mediated by arterial compliance, including mechanoreceptor activity, BP control, cardiac function and autonomic balance in the cardiovascular system.

A second notable sex difference was in the behavioural performance on the tasks which was also evident only after repeated measurement. Although the magnitude of difference in problem solving performance evident in Experiments 2, 3, and 4 could have been partly an artefact of subject selection, a similar, but smaller, difference was also evident in Experiments 1 and 5. In addition, women and men differed in personal tempo button pressing during Experiments 3 and 4, but not in Experiment 1. The generality of the difference, and the possibility that its direction would change if task content was changed, for example if mental arithmetic was the task of choice (Dembroski, et al., 1978; MacDougall, et al., 1981) are questions which remain to be resolved.

11 CONCLUSION

The relationship between task difficulty and changes in cardiovascular responding during task was investigated in the context of active/passive coping. Focus was placed on arterial pulse transit time (PTT) and interbeat interval (IBI) was measured as the reference response. Specifically, that hypothesis postulates that the magnitude of IBI change during task is directly related to the subject's state of behavioural coping, or level of engagement, in the task. That is, the magnitude of change and the behavioural state of coping are determined by environmental contingencies, in particular the task difficulty. In addition, acceleratory and deceleratory cardiovascular response changes were examined.

In Experiment 1, men and women completed mental arithmetic, problem solving, reaction time, and voluntary button pressing (personal tempo) tasks. The magnitude of PTT change during task and pre-task instruction periods was not determined by task difficulty, but that of IBI was. Both acceleratory and deceleratory PTT changes occurred during all tasks. Acceleratory IBI changes occurred during all tasks, but deceleratory IBI changes were significant only during reaction time and personal tempo. Accelerators and decelerators were compared for differences in sex, in baseline levels of cardiovascular activity and in behavioural task performance. No differences were found.

In Experiment 2, men and women completed problem solving tasks at two levels of difficulty. Acceleratory PTT and IBI changes were significant during

both tasks; in addition, deceleratory PTT changes were significant during the harder task. The magnitude of change in IBI was not determined by task difficulty during task, but was determined by task difficulty during pre-task instruction. Systolic (SBP) and Diastolic (DBP) Blood Pressures were also recorded, and larger increases in SBP were found during the harder task. Only acceleratory SBP and DBP changes were significant during either task.

At the beginning of Experiment 3, subjects completed the State Trait Anxiety Inventory, the Stress Arousal Checklist, the Jenkins Activity Survey and the Eysenck Personality Inventory before completing problem solving tasks at three levels of difficulty and personal tempo. The magnitude of IBI change was determined by task difficulty in the following descending order: hard, easy, impossible and personal tempo. Both acceleratory and deceleratory PTT, SBP and DBP changes were significant during all tasks, but deceleratory IBI changes were significant only during personal tempo. No systematic effects were found in an exploratory attempt to relate the directional variability in PTT change to scores on any of the psychological measures administered at the beginning of the experiment.

In summary, the magnitude of task-generated change in IBI was determined by task difficulty in Experiments 1 and 3, the magnitude of instruction generated changes in IBI was determined by task difficulty in Experiments 1 and 2. Directional variability characterized the PTT response during all tasks in all experiments. Acceleratory IBI changes only were significant during mental arithmetic and problem solving tasks in all experiments, but acceleratory and deceleratory IBI changes were significant during reaction time and personal tempo. The magnitude of SBP change was determined by task difficulty in

Experiment 2, but not in Experiment 3 when directional variability was also evident in both the SBP and DBP changes during task.

Two extensions of the active/passive coping hypothesis were proposed: 1) that IBI change is more appropriately related to task demands along a continuum of behavioural coping rather than by two dichotomous behavioural states; 2) that the frequency of acceleratory IBI change is determined by task type as distinct from task difficulty. It was also noted that the effects of task novelty and task contingency were not explicit in the active/passive coping hypothesis. But the present results suggest that task novelty combines with task difficulty to determine the magnitude of IBI change. In addition, the need to quantify the effect of task contingency on IBI responding was identified as an area requiring future study.

It was also concluded that the observed changes in SBP and DBP could be accounted for by a combination of task difficulty and novelty. But the extraordinary directional variability of PTT changes remained unexplained by task difficulty or by differences in sex, baseline activity, task performance or psychological measures.

Thus an exploration into the directional variability of PTT was begun. It was hypothesized that the subject's behavioural state as measured by relative HR prior to task could be a determinant of the direction of PTT change during task. PTT was converted to pulse wave velocity (PWV) in order to control for differences in subject height.

In Experiment 4, men and women completed four tasks twice, once in each part of an extended experimental session. In the interval between tasks, half

the subjects each consumed a caffeine or a placebo drink, and also completed the psychological measures used in Experiment 3. IBI was slower, suggesting lower activation, during the baseline in the second part of the experimental session. PWV accelerated during the tasks in the first part of the experimental session and decelerated during tasks in the second part. However, IBI also decelerated following caffeine ingestion, but caffeine had no effect on PWV changes although it enhanced the acceleratory IBI and DBP changes during tasks as well as increasing the levels of both SBP and DBP. In addition, both acceleratory and deceleratory PWV, and SBP and DBP changes during tasks continued to be significant. Thus the hypothesized relationship between behavioural state as measured by relative HR during baseline and the direction of the subsequent task-generated PWV change received only partial support.

That hypothesis was further investigated in Experiment 5. IBI was converted to heart rate (HR), and a calculated estimate of left ventricular ejection time (LVET) was recorded during five separate rest periods each of 10 minutes duration, and each separated by a minimum of 24 hours. At the completion of the rest period on session 5, men and women completed a single problem solving task. HR accelerated over the 5 baseline periods in anticipation of the task, suggesting an increase in activation. All acceleratory changes, but no deceleratory changes, during task were significant, thus supporting activation level as a mediating variable of the direction of PWV change. Further support for that hypothesis was derived from the adventitious recording of 2.6-fold acceleratory changes in PWV, accompanied by an increase in resting HR, from a subject suffering under a severe emotional crisis. Further study of the directional variability in PWV change during stress and the contribution to that change by initial individual behavioural

state is indicated and individual differences in HR responsivity were suggested as one context for future research into PWV response to stress.

In the course of the research, relatively detailed comments were made on the likely mechanisms of the changes observed and their possible physiological significance. While it should be noted that because of the absence of invasive tests such comments must be recognized as being speculative, it is considered that such speculation is necessary in order that the research and the researchers maintain an orientation toward biological significance and physiological reality.

Throughout the research it was found that acceleratory HR changes could be accompanied either by acceleratory PWV change, indicating an increase in cardiac load, or by deceleratory PWV change, indicating a decrease in cardiac load. The results of Experiments 4 and 5 suggest that a more active behavioural state prior to task, indicated by a relatively faster resting HR, is likely to be associated with an increase in cardiac load during task, and that a less active behavioural state prior to task, indicated by a relatively slower resting HR, is likely to be associated with a decrease in cardiac load during task.

APPENDIX 1. TASK RESPONSES FOR MEN AND WOMEN

This appendix presents the cardiovascular changes during all the experimental conditions for men and women. It should be noted that none of the comparisons presented here reached statistical significance.

It comprises 8 Figures and 1 Table. The figures present the response changes for men and women during each task on each response for Experiments 1-4, with the exception of the response pattern for DBP in Experiment 4. That interaction (Sex X Part X Condition X Task) was statistically significant and is presented in the text of Experiment 4. The table presents the change during tasks of all the measured responses for men and women in Experiment 5.
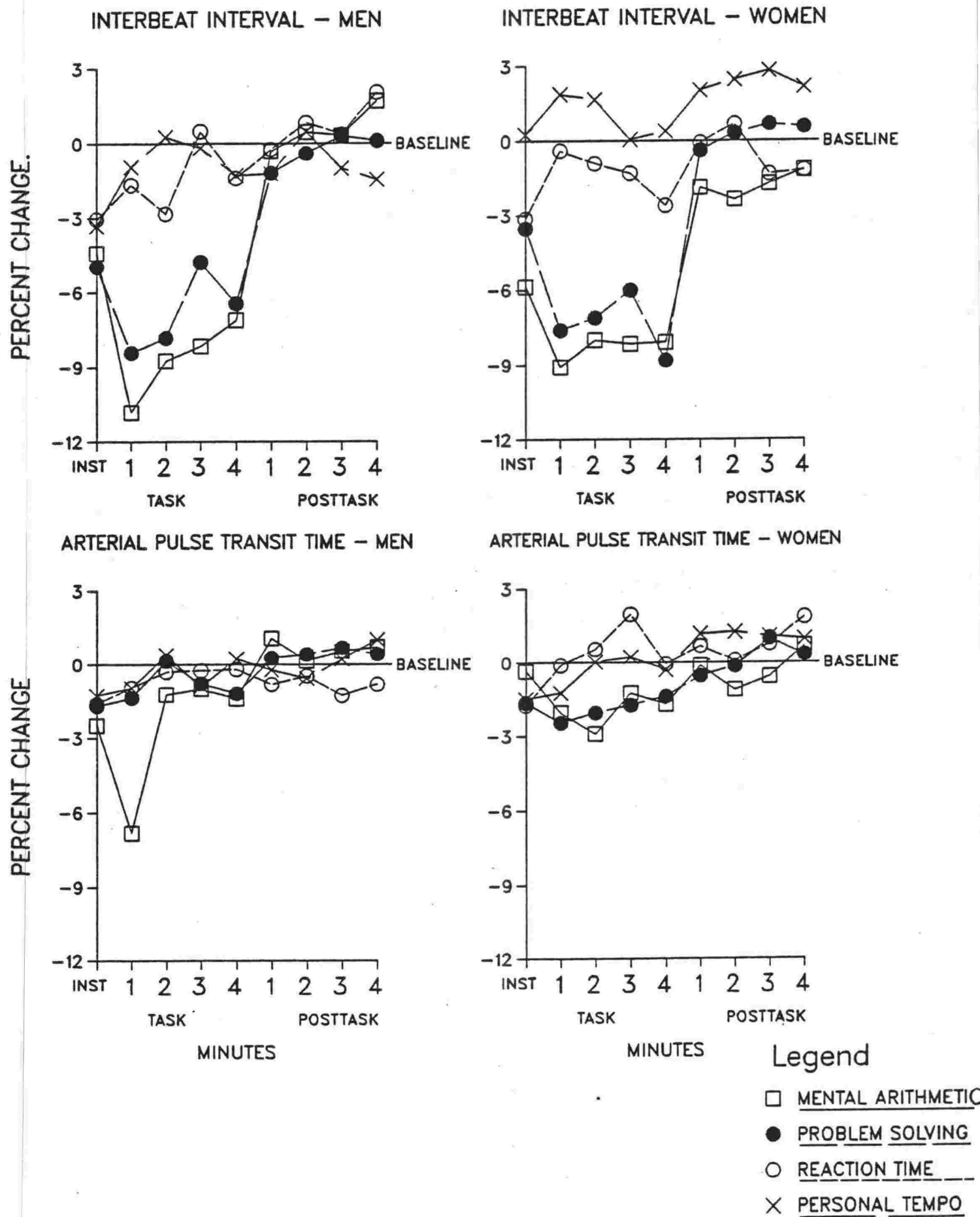
Figure A. Percentage Change During All Conditions for
men (left side) and women (right side) in Experiment 1
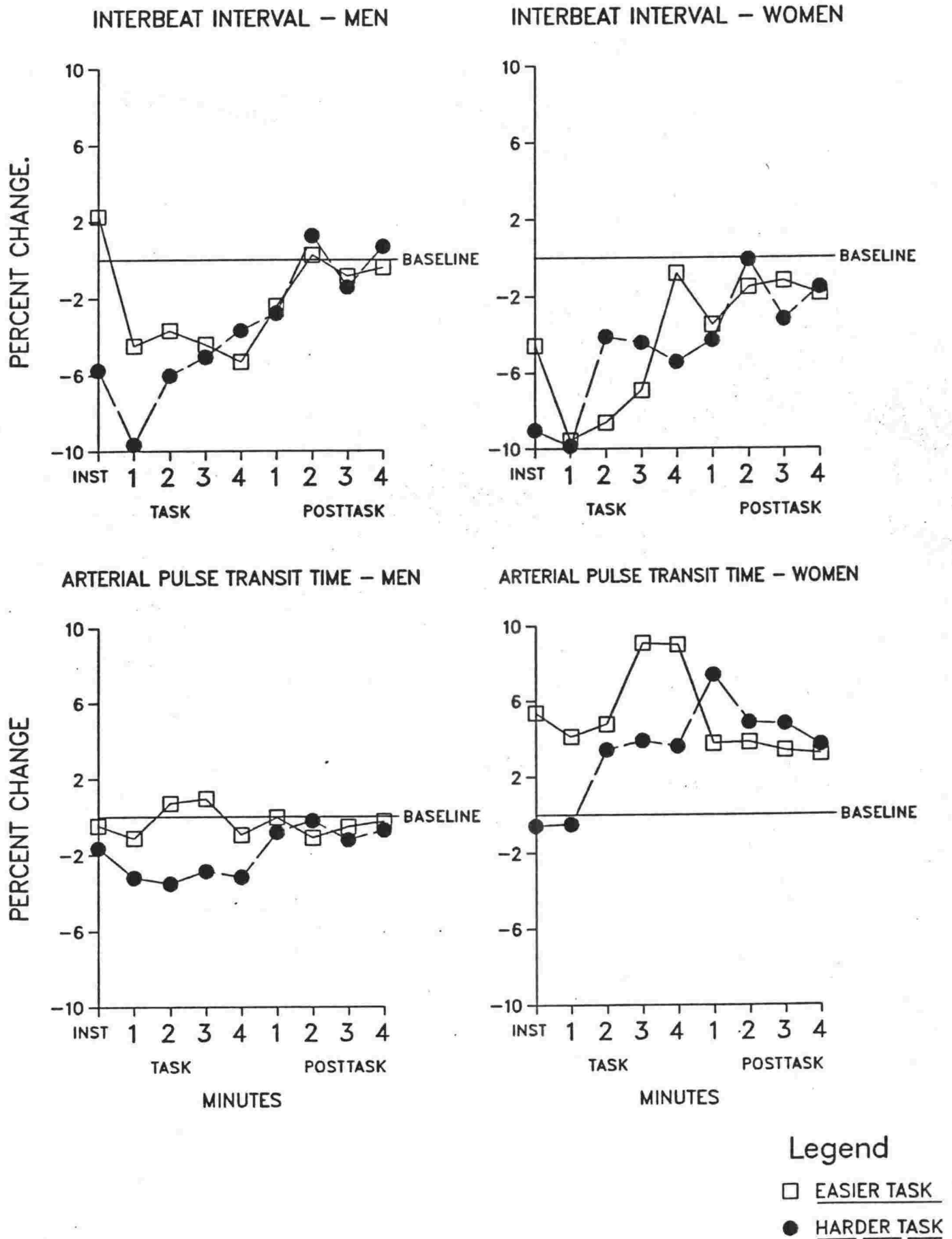
Figure B. Percentage Change During All Conditions for men (left side) and women (right side) in Experiment 2
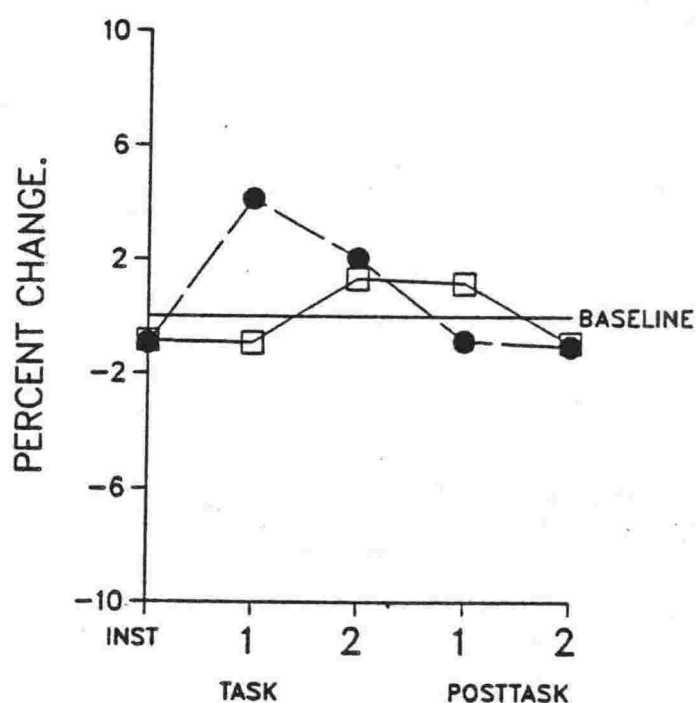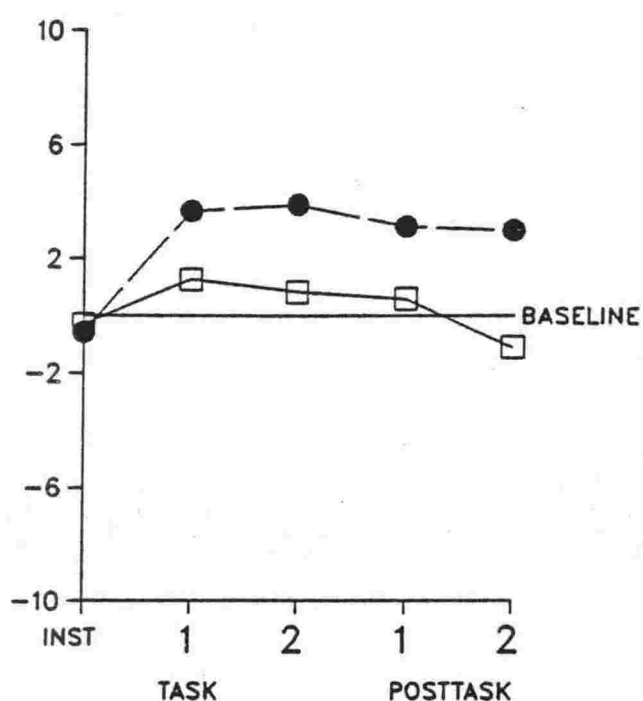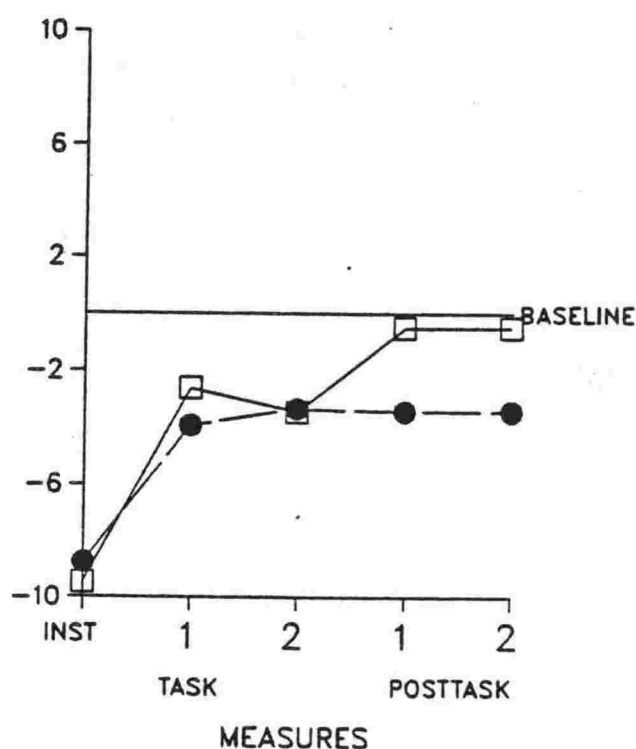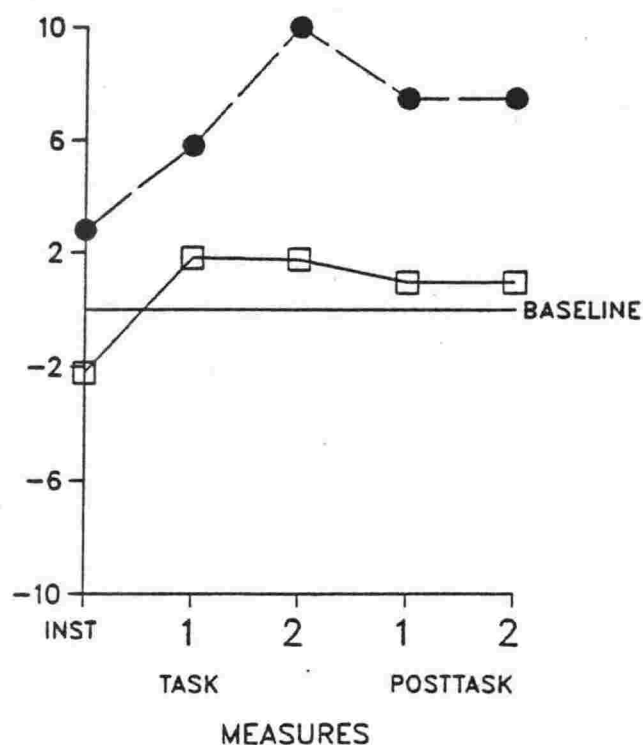
Figure C. Percentage Change During All Conditions for men (left side) and women (right side) in Experiment 2
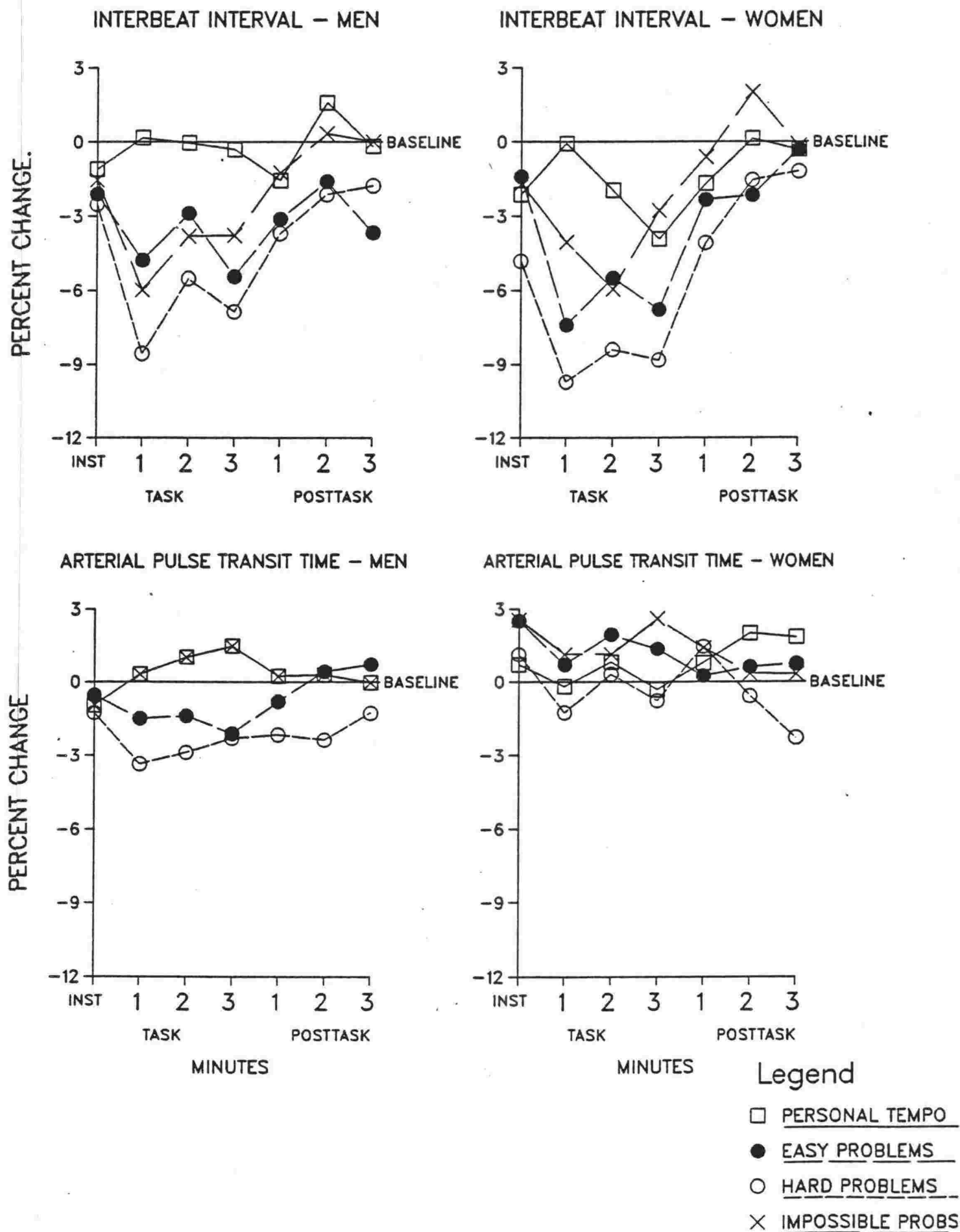
Figure D. Percentage Change During All Conditions for men (left side) and women (right side) in Experiment 3
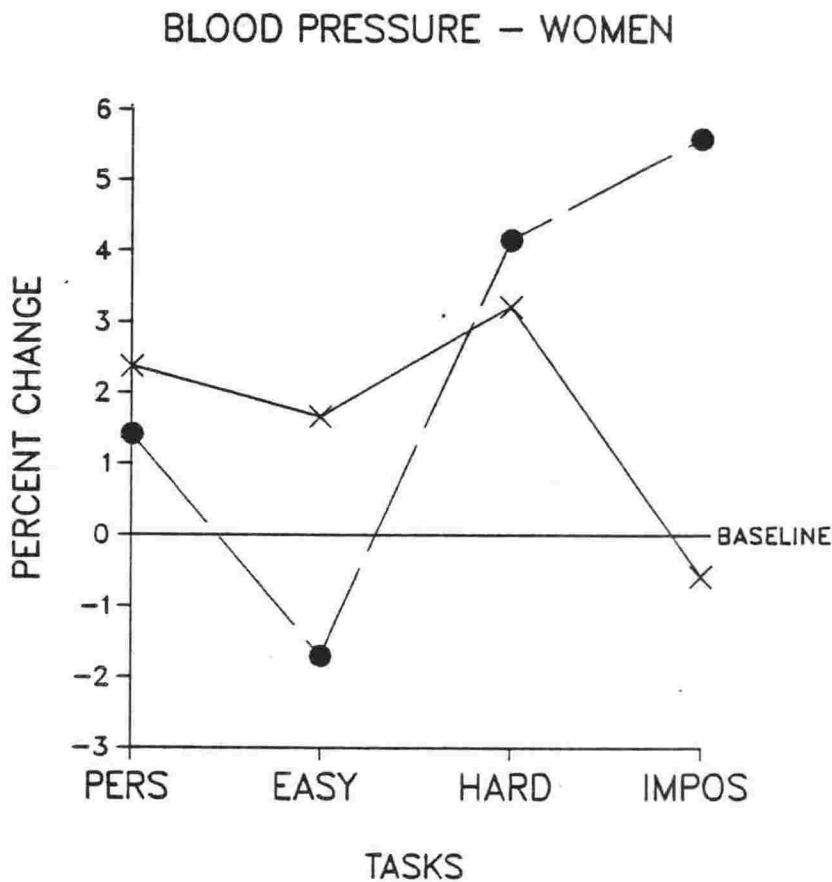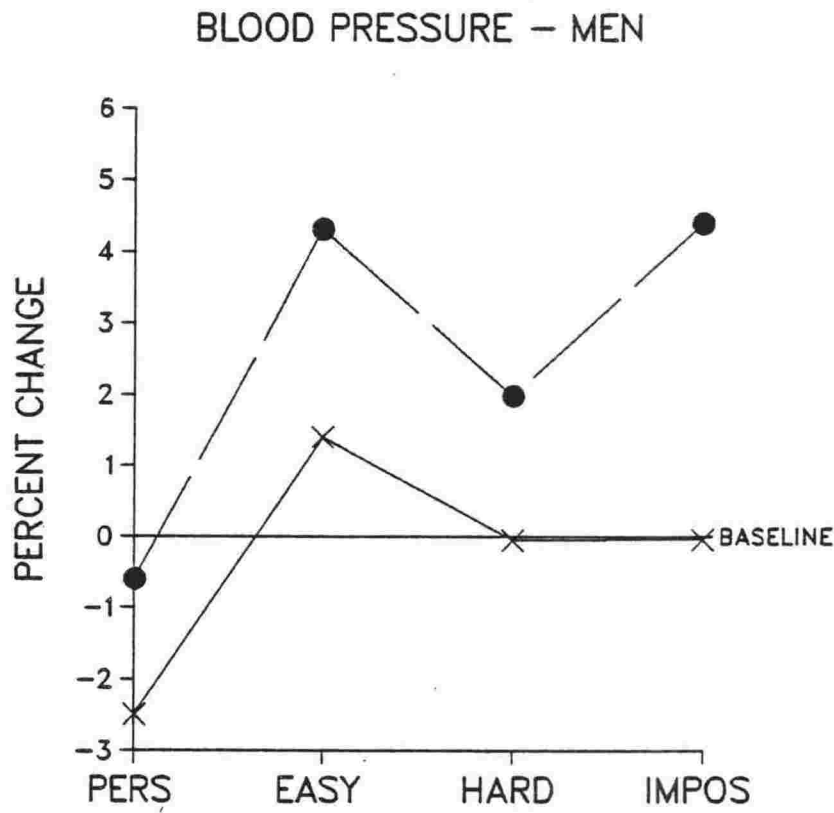
Figure E. Systolic and Diastolic Blood Pressure
Responses for men and women in Experiment 3.
(EASY=easy problems; HARD= hard problems;
IMPOS=impossible problems; PERS=personal tempo

INTERBEAT INTERVAL — MEN

INTERBEAT INTERVAL — WOMEN
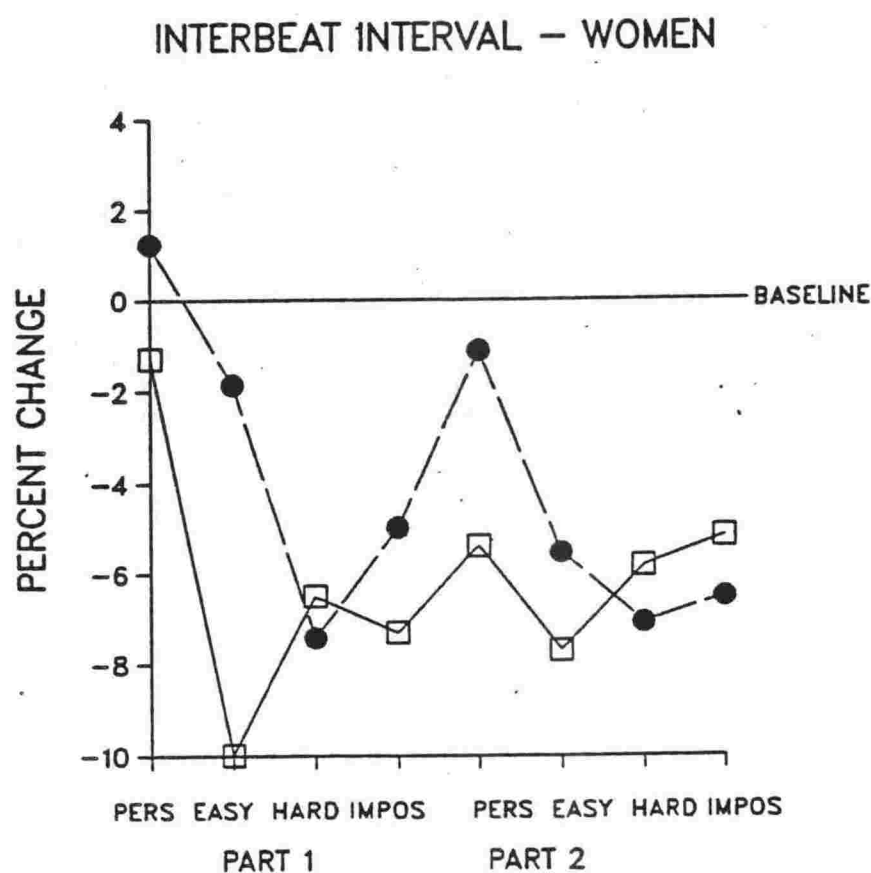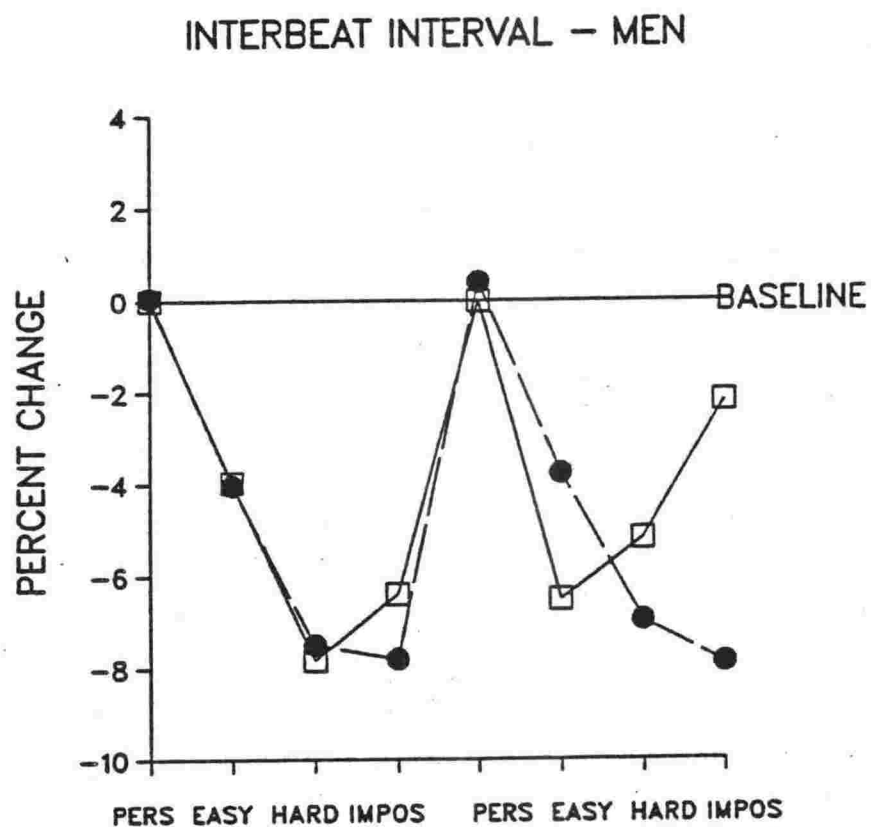
Legend

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Figure F. Interbeat Interval Response during task
in both session parts for men and women in Experiment 4.

(PERS=personal tempo; EASY=easy problems;
HARD=hard problems; IMPOS=impossible problems.
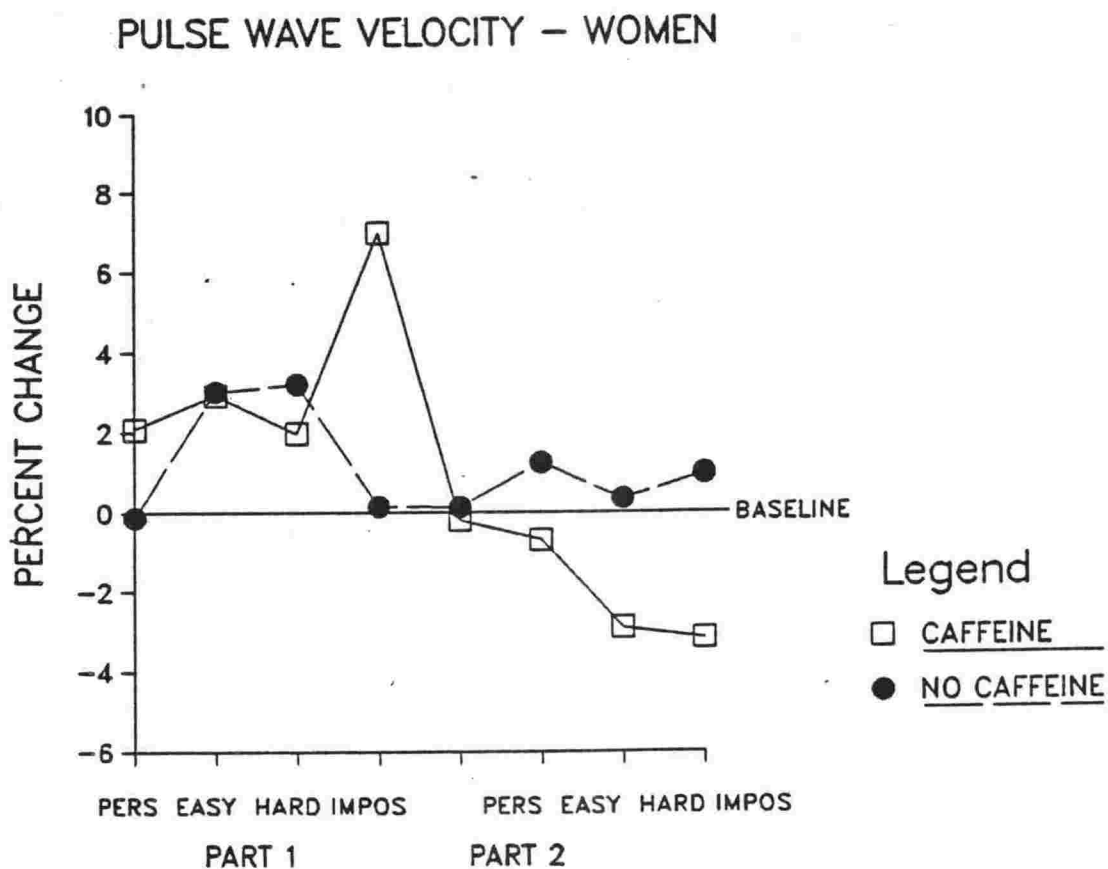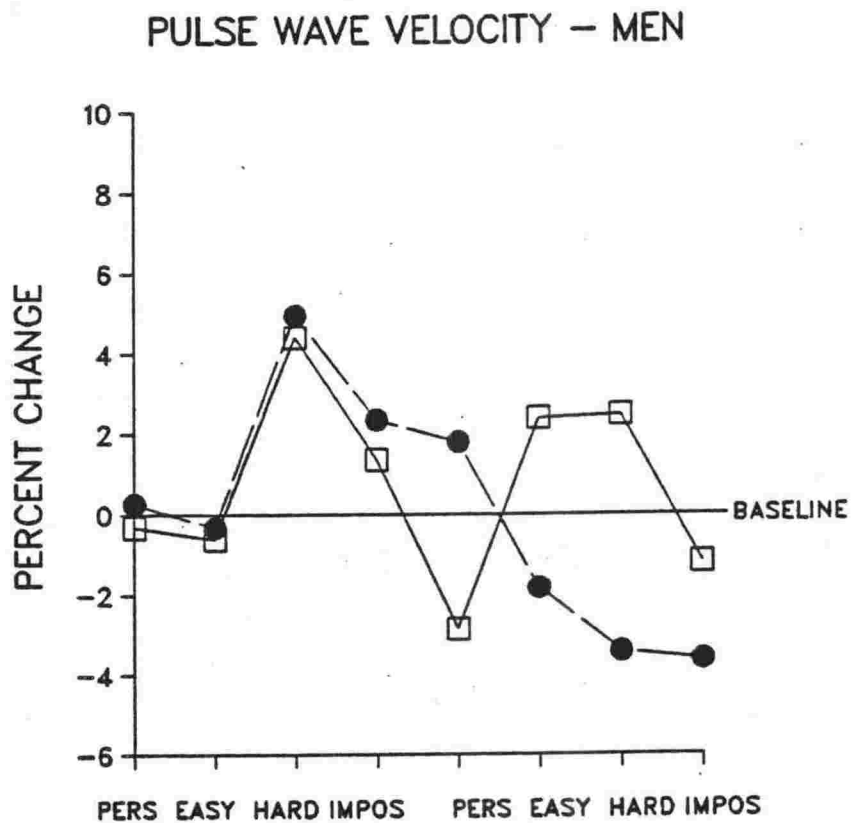
Figure G. Pulse Wave Velocity Response during task
in both session parts for men and women in Experiment 4.

(PERS=personal tempo; EASY=easy problems;
HARD=hard problems; IMPOS=impossible problems.
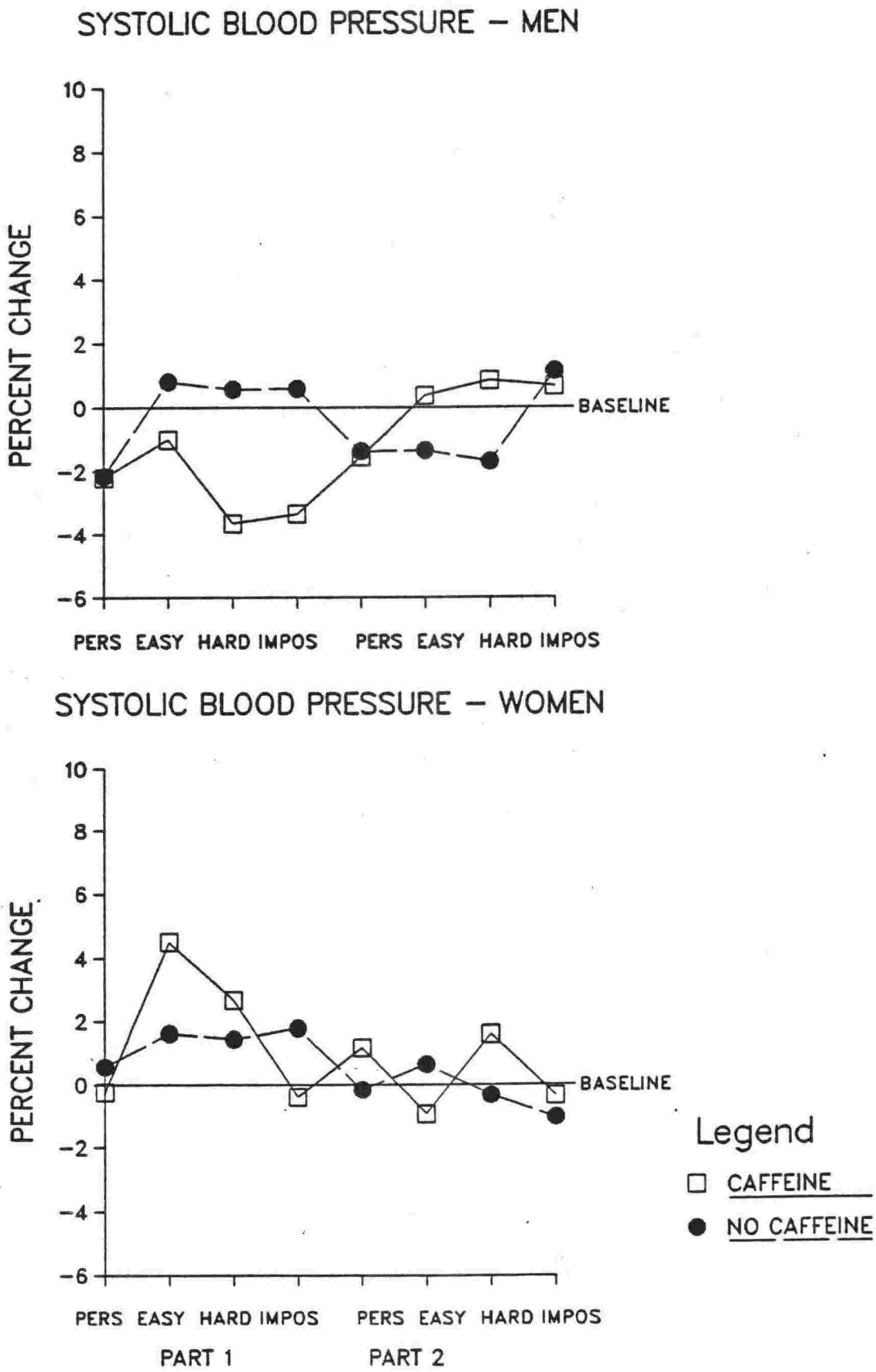
Figure H. Systolic Blood Pressure Response during task
in both session parts for men and women in Experiment 4.

(PERS=personal tempo; EASY=easy problems;
HARD=hard problems; IMPOS=impossible problems.

Table A

Response change from pre-task baseline during instruction, task and post-task periods for men and women in Experiment 5. Response change was compared to baseline using t-tests for paired means comparisons. The significance of deviation is indicated. Responses are Heart Rate (HR), Pulse Wave Velocity (PWV), Left Ventricular Ejection Time (LVET), and Systolic (SBP) and Diastolic (DBP) Blood Pressures. (Inst.=instruction period; Task=task period; Post.=post-task period).

	HR	PWV	LVET	SBP	DBP
		Men Subjects (n=7)			
Inst.	+ 5.14*	+0.43	-4.85	+0.43	+1.00
Task	+10.95*	+1.43	-6.05	+4.29	+7.29
Post.	- 0.57	+0.10	-1.90	-0.57	-0.29
		Women Subjects (n=11)			
Inst.	+ 6.58*	+0.27	-3.73*	+1.64	+1.55
Task	+10.48*	+0.73*	-7.91*	+7.55*	+4.00
Post.	- 0.27	+0.12	-0.70*	+3.54	-1.27

* p<.05 or better

APPENDIX 2. R-WAVE TO PULSE INTERVAL DATA

R-wave to pulse interval (RPI) was recorded as a noninvasive cardiac measure of interest (Pollack & Obrist, 1983). Specifically, changes in RPI have been argued to reflect changes in the beta-adrenergic influences on the myocardium (Newlin & Levenson, 1979; Obrist et al., 1979) and, because RPI is readily measured noninvasively and is relatively artefact free (Obrist, et al., 1979) it is a response which is likely to be recorded with increasing frequency. Given these considerations, and also given the fact that, in the system under use in the present research, RPI was incidentally recorded in the measurement of PTT, it was decided to also present the RPI data from Experiments 1-4 as being of interest and of value for future reference. The RPI measures from Experiment 5 were used to calculate LVET. The detail of that calculation is presented in the final section of this appendix. As the LVET response was presented in the text of Experiment 5, the associated RPI data from Experiment 5 are not presented.

GENERAL METHOD

As RPI was recorded incidentally with PTT, the experimental method, subjects and procedures are those already reported in Experiments 1-4.

Apparatus and Physiological Recording

The apparatus was that described in Experiments 1-4.

As previously described, the R-wave of the EKG was used to trigger each sweep of the oscilloscope; and the pulse arrival at the auricular artery on the right ear was detected by a photocell and fed into an oscilloscope (see Experiment 1). RPI was measured as the interval between the R-wave of the EKG and the foot of the ensuing auricular pulse. The oscilloscope digitized the information and output it to the controlling computer.

RESULTS

The presentation of the results from each experiment parallels that of IBI and PTT. Analyses of absolute levels during all experimental conditions are presented first, then response changes, and finally the frequency and magnitude of acceleratory and deceleratory RPI changes are reported.

ANALYSES OF ABSOLUTE LEVELS

Absolute levels of RPI during the initial baseline in each experiment were analysed for evidence both of sex differences and of deceleratory changes (Sex X Minutes ANOVA with repeated measures).

Absolute levels of RPI during the pre-task baselines in each experiment were analysed for evidence both of sex differences and of task-related differences which could confound subsequent task-generated responding (Sex X Task ANOVA with repeated measures).

Absolute levels of RPI during the instruction, task and post-task periods were analysed for evidence of sex differences (Sex ANOVA).

EXPERIMENT 1

Initial Baseline

A Main Effect of Minute ($F(9/432)=3.11$, $p<.001$) was found for absolute levels of RPI during the initial baseline period in Experiment 1, reflecting an overall deceleratory change during that period.

Pre⁻task Baseline

No significant effects were found.

Instruction Periods

No significant effects were found.

Task Periods

No significant effects were found.

Post⁻task Periods

No significant effects were found.

EXPERIMENT 2

No significant effects were found on any analysis of any measure of absolute RPI activity in Experiment 2.

EXPERIMENT 3

No significant effects were found on any analysis of any measure of absolute RPI activity in Experiment 3.

EXPERIMENT 4

Initial Baseline

There was a Main Effect of Minute ($F(4/104)=3.49$, $p<.01$) found for RPI over the initial baseline period in Experiment 4 which reflected a deceleratory change during that period.

Pre‾task Baseline

No significant effects were found.

Task Periods

No significant effects were found.

Post‾Task Periods

No significant effects were found.

ANALYSES OF RESPONSE CHANGES

Response change from pre-task baseline was calculated for RPI during each pre-task instruction period, and for each minute of all task and post-task periods. Analyses were performed to ascertain the magnitude and variability of the changes which occurred during each of the experimental conditions (Sex X Task X Minute ANOVA with repeated measures on the last two factors). Response changes during tasks in all four experiments are shown in Figure I.

EXPERIMENT 1

Instruction Periods

No significant effects were found.

Task Periods

There was a Main Effect of Task ($F(3/144)=6.01$, $p<.0008$) found for task-generated accelerations in RPI. Follow-up REGWF tests on the means confirmed a significant difference between task-generated acceleratory responses for RPI in the following order of decreasing magnitude across tasks: problem solving (-7.69ms), mental arithmetic (-6.60ms), reaction time (-3.94ms) and personal tempo (-1.07ms). That effect is shown in Figure I (top left graph).

A Main Effect of Minute was found for task-generated changes in RPI ($F(3/144)=6.23$, $p<.0006$). Acceleratory changes peaked on minute 2.

There was a Sex X Task X Minute Interaction found for task-generated changes in RPI ($F(9/432)=2.02$, $p<.04$). Men had larger, but less sustained

changes in RPI than the women, particularly during the reaction time task. In addition, while women had a larger acceleratory change during mental arithmetic than during problem solving, men had a larger acceleratory change during problem solving than during mental arithmetic (Sex X Task X Minute Interaction, $F(9/432)=2.02$, p<.04). That interaction can be seen in Figure J.

Post-task Periods

There was a Main Effect of Minute found for RPI recovery during post-task ($F(3/144)=15.82$, p<.0001), reflecting a return of the response to pre-task baseline level.

There was a Task X Minute Interaction ($F(9/432)=1.98$, p<.04). The R-wave to pulse interval response returned to pre-task baseline level over successive minutes of post-task recovery following mental arithmetic, problem solving and personal tempo, but over-recovered following the reaction time task prior to returning to pre-task baseline level.

EXPERIMENT 2

Instruction Periods

There was a Main Effect of Sex found for the RPI response during the instruction period ($F(1/17)=5.38$, p<.03). The women had a larger acceleratory change than the men (-2.1% vs -0.57%).

Task Periods

A Main Effect of Task was found for task-generated changes in RPI ($F(1/17)=12.33$, $p<.003$). There were accelerations of greater magnitude during the more difficult task (-3.29ms vs -6.75ms). That effect is shown in the top right graph in Figure I. The RPI change during all experimental conditions for men and women in Experiment 2 is shown in Figure K.

Post⁻task Periods

There was a Main Effect of Minute ($F(3/51)=3.48$, $p<.03$) found during the post-task period reflecting the return of the RPI response to the pre-task baseline level.

A Sex X Task Interaction, ($F(1/17)=4.5$, $p<.05$) was found for the RPI recovery response during the post-task periods. For the men, RPI decelerated following Task E but accelerated following Task D. For the women, RPI accelerated following both tasks.

EXPERIMENT 3

Instruction Periods

No significant effects were found.

Task Periods

There was a Main Effect of Task found for task-generated changes in RPI ($F(3/84)=3.17$, $p<.03$). Follow-up REGWF means tests showed that the magnitude of change was determined by task difficulty in the following descending order:

easy, hard, impossible, and personal tempo. That effect is shown in bottom left graph in Figure I.

There was a Main Effect of Minutes ($F(2/56)=3.59$, $p<.03$) for task-generated changes in RPI. Follow-up means tests showed that, as in Experiment 1, the acceleratory response peaked on minute 2 of the task periods. The RPI change during all experimental conditions for men and women in Experiment 3 is shown in Figure L.

Post-task Periods.

There was a Main Effect of Minute ($F(2/56)=10.64$, $p<.0001$) for the recovery of RPI during post-task. The follow-up means tests showed a significant deceleration from minute 1 to minute 2 of the post-task, and a return to pre-task baseline levels by minute 3.

EXPERIMENT 4
Task Periods

There was a Main Effect of Tasks found for task-generated changes in RPI ($F(3/78)=3.35$, $p<.02$). Follow-up REGWF means tests showed that the magnitude of change was determined by task difficulty in the following descending order: easy, hard, impossible, and personal tempo. That effect is shown in the bottom right graph in Figure I. The RPI change during all experimental conditions for men and women in Experiment 4 is shown in Figure M.

Post-task Periods

There was a Sex X Part Interaction found for RPI recovery during post-task ($F(a/26)=5.80$, $p<.02$). Women had a deceleratory recovery response in both parts which was larger in part 2 than in part 1; the men had a deceleratory recovery response in part 1 and an acceleratory recovery response in part 2.

There was also a Part X Task Interaction found for RPI recovery ($F(3/78)=2.73$, $p<.05$). RPI recovery was deceleratory following the hard and impossible tasks in both parts of the experimental session with larger magnitude changes in part 2; RPI recovery was deceleratory following the easy task in part 1 and acceleratory following the easy task in part 2; it was acceleratory following the personal tempo task in part 1 and deceleratory following the personal tempo task in part 2.
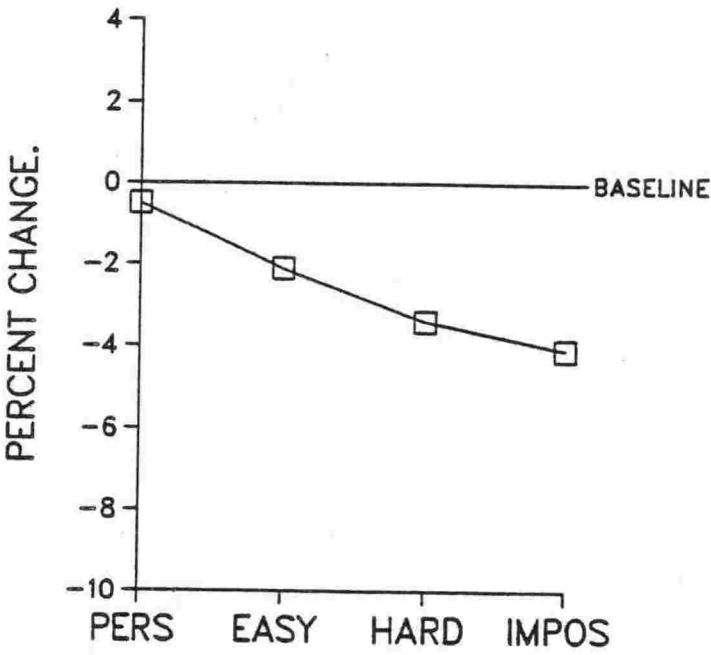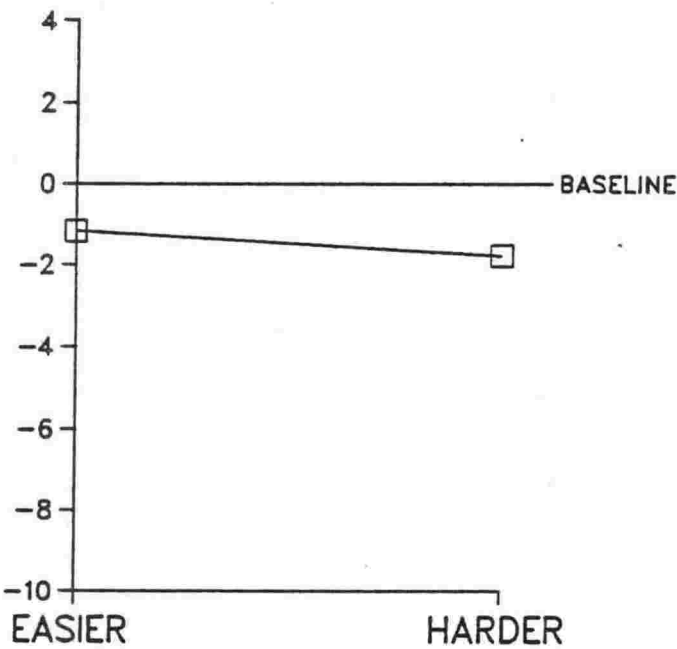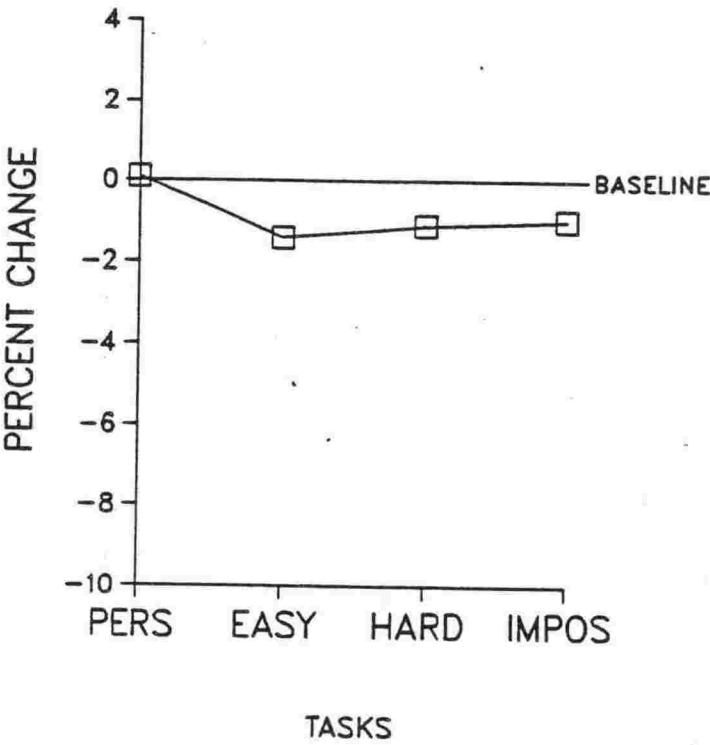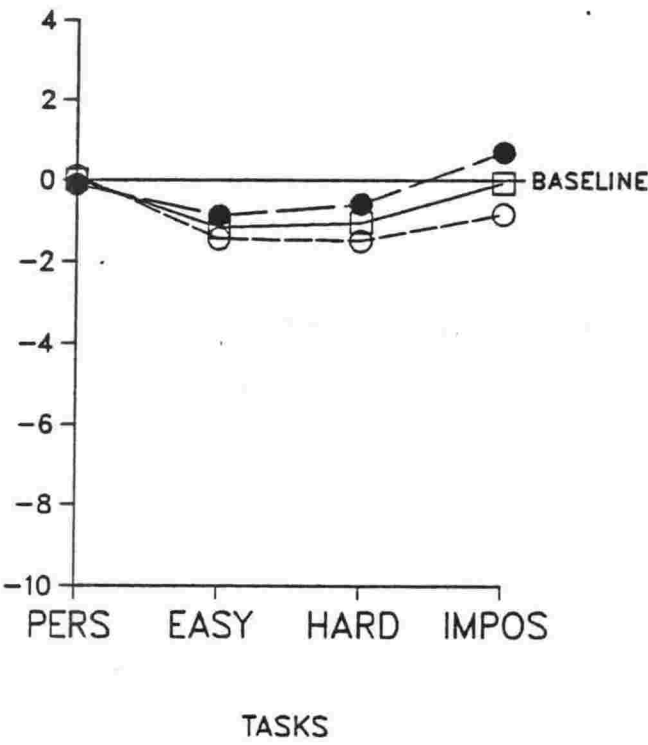
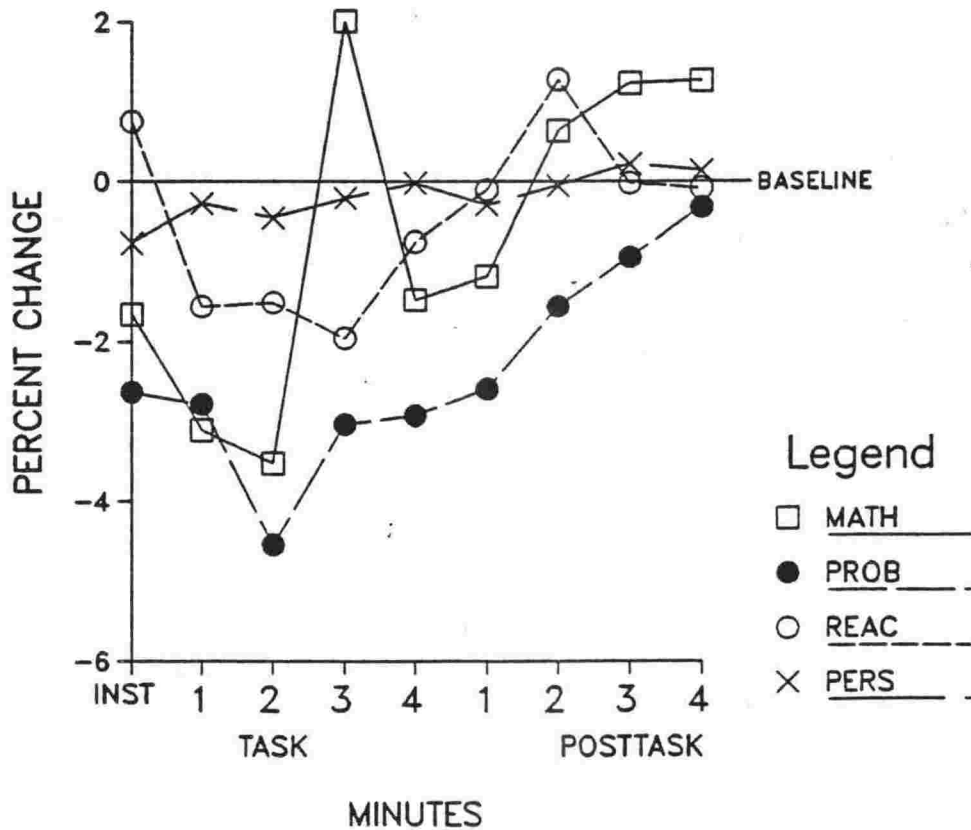Figure I. Percentage Change in R—wave to pulse interval showing the Task Effect found in all four experiments

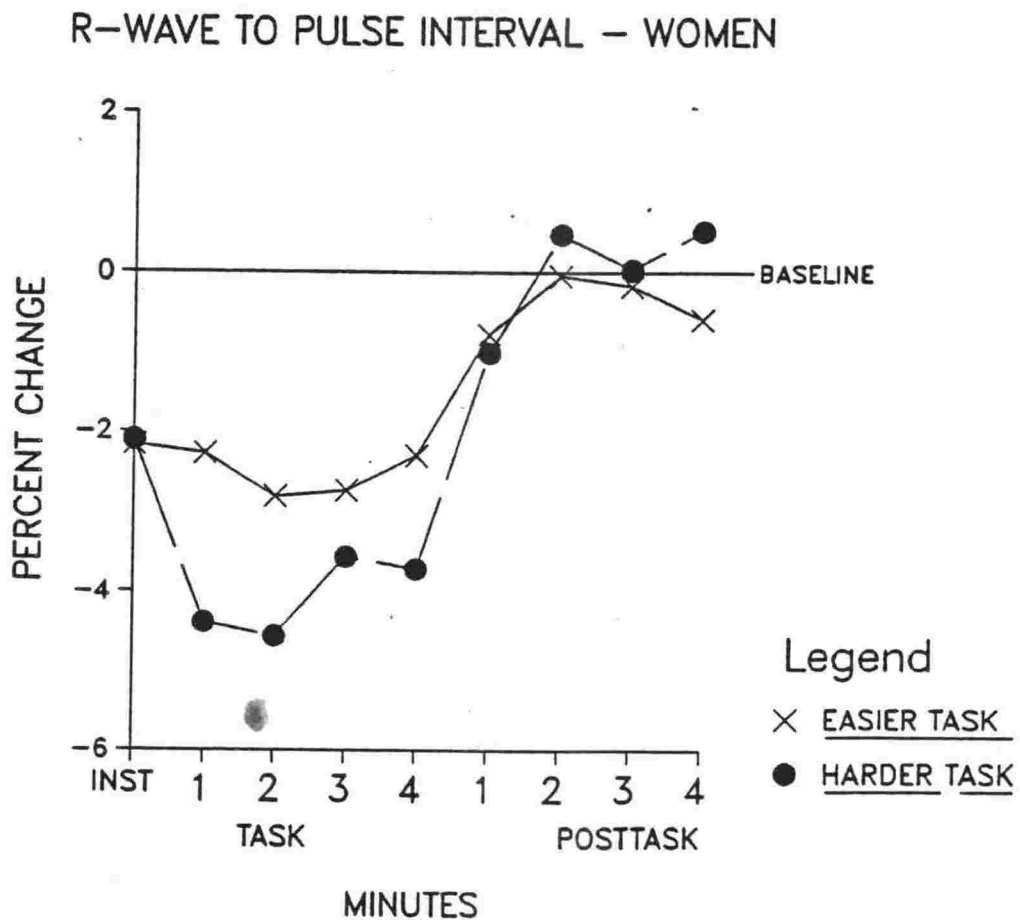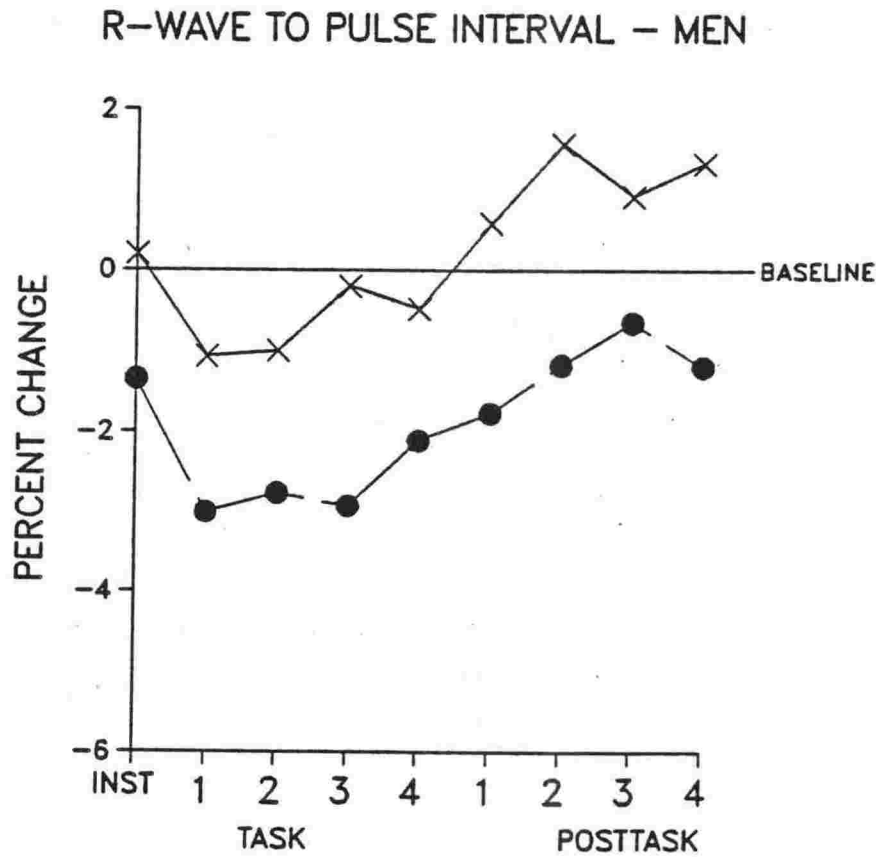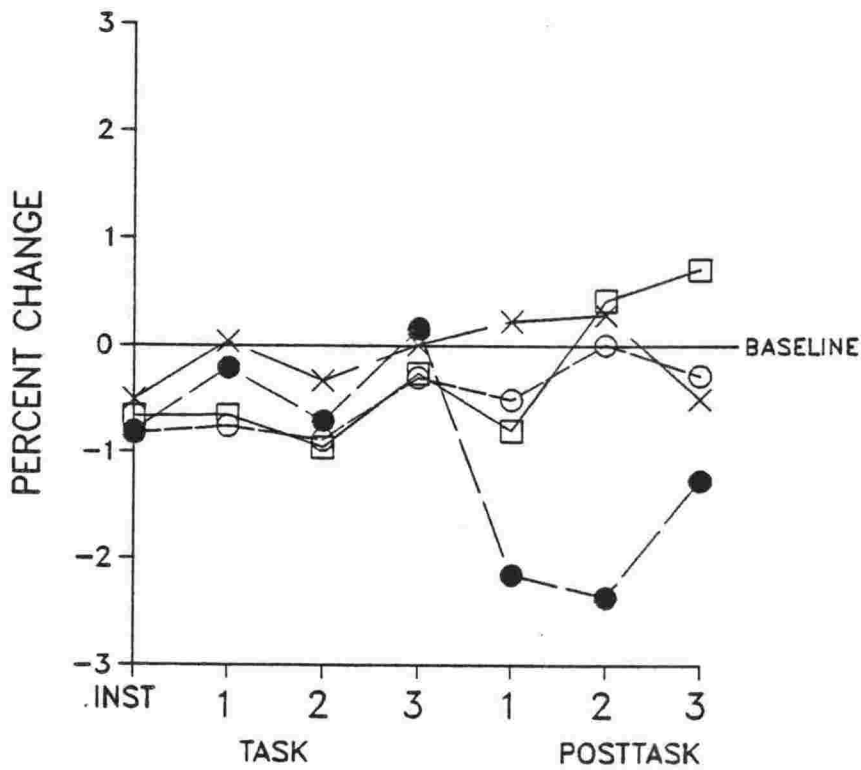Figure J. Percent change in R–wave to pulse Interval during Experiment 1 for men (top) and women (bottom)

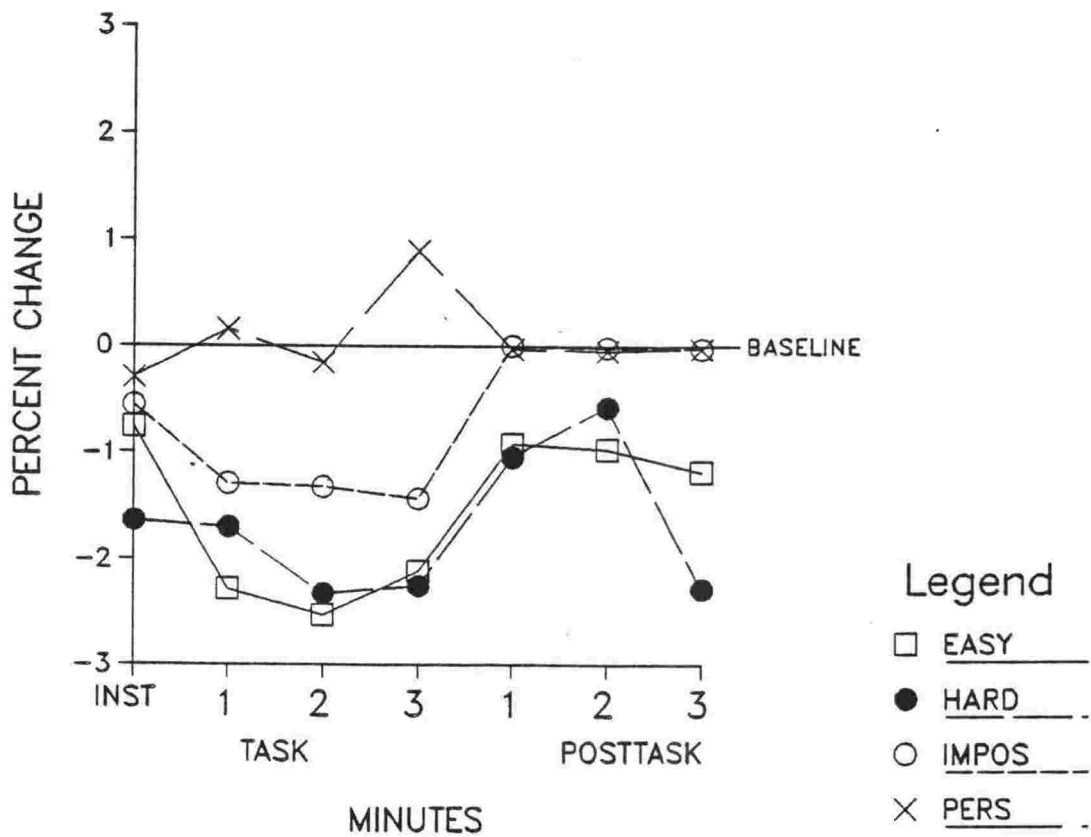Figure K. Percent change in R—wave to pulse Interval during Experiment 2 for men (top) and women (bottom)

Figure L. Percent change in R–Wave to Pulse Interval during Experiment 3 for men (top) and women (bottom) PERS=personal tempo; EASY=easy problems; HARD= hard problems; IMPOS=impossible problems).

R−WAVE TO PULSE INTERVAL − MEN

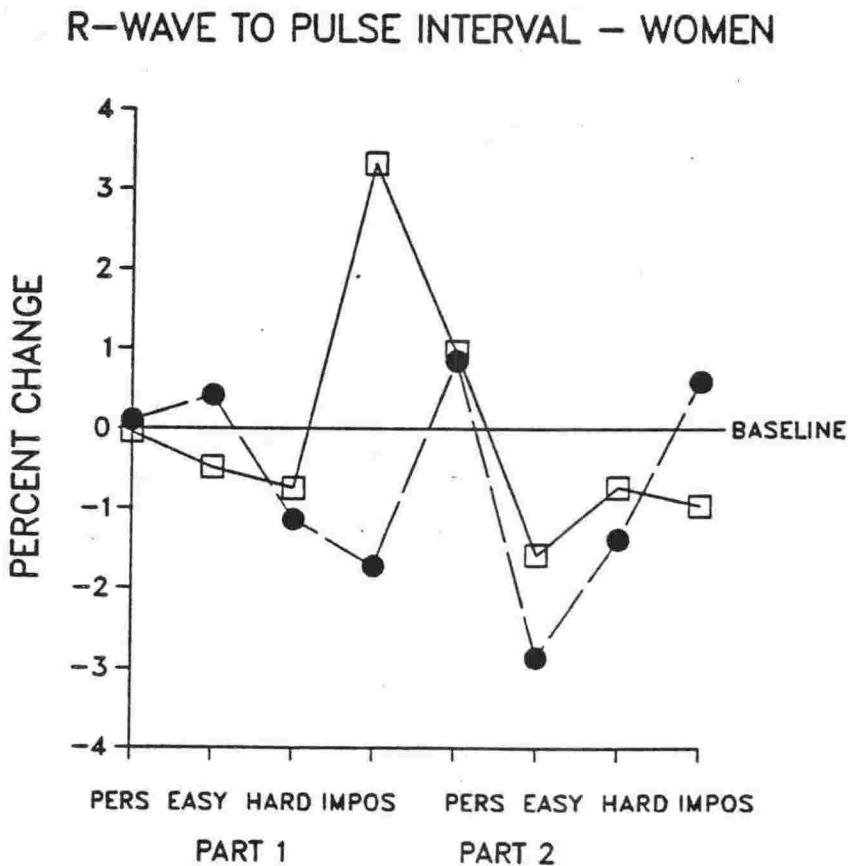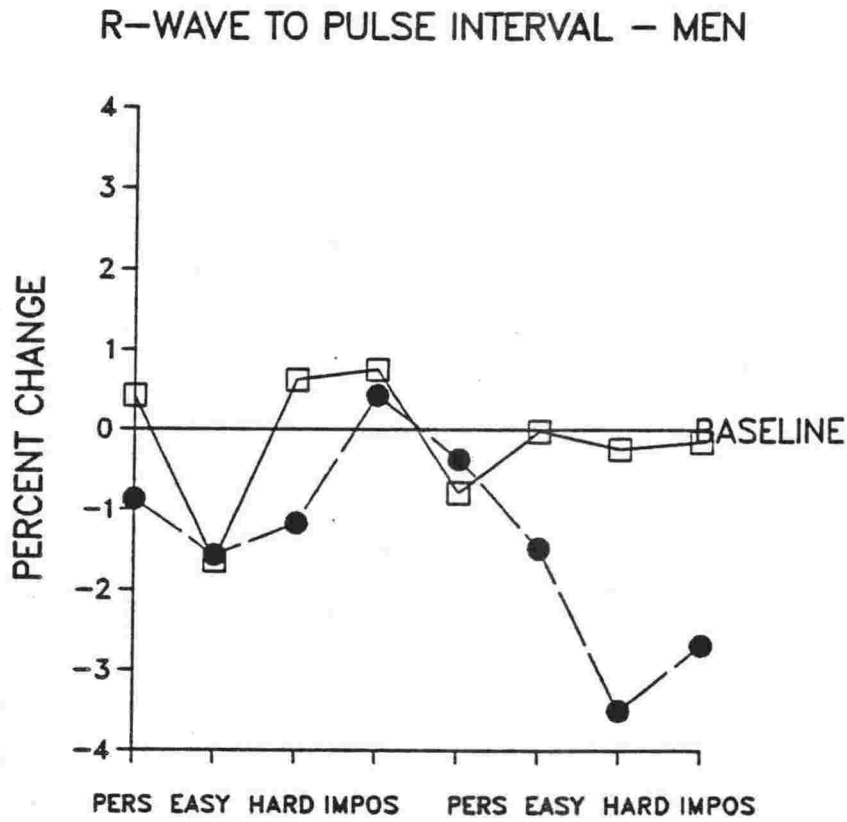

R−WAVE TO PULSE INTERVAL − WOMEN

Figure M. Percent change in R−Wave to Pulse Interval during task in both session parts for men and women in Experiment 4.

(PERS=personal tempo EASY=easy problems;
HARD=hard problems IMPOS=impossible problems;).

DIRECTION OF RESPONSE CHANGE

As with the other responses measured, the frequency and the significance of acceleratory and deceleratory RPI changes for each task during each experiment were tabulated. The method used to classify each subject as an accelerator or decelerator on each task was first described in Experiment 1. First, the mean of the task-generated RPI response was found; then, if the response was less than pre-task baseline, the subject was classified as an accelerator, but if the mean was greater than pre-task baseline, the subject was classified as a decelerator. As with the other groupings, the RPI groupings were tested for differences between accelerators and decelerators in sex, pre-task baseline levels, behavioural performance, and in the data from Experiments 3 and 4, for differences in scores on the psychological measures used. No differences were found. The tables also give the values of the other concurrently measured responses.

EXPERIMENT 1

The frequency and the significance of the acceleratory and deceleratory RPI changes during task are shown in Table B. The minute-by-minute response changes are shown in Figure N.

EXPERIMENT 2

The frequency and the significance of the acceleratory and deceleratory RPI changes during task are shown in Table C. The minute-by-minute response changes are shown in Figure O.

EXPERIMENT 3

The frequency and the significance of the acceleratory and deceleratory RPI changes during task are shown in Table D. The minute-by-minute response changes are shown in Figure P.

EXPERIMENT 4

The frequency and the significance of the acceleratory and deceleratory RPI changes during task in part 1 are shown in Table E, and those in part 2 are shown in Table F. The minute-by-minute response changes for parts 1 and 2 are shown in Figures Q and R respectively.

Table B

R-wave to Pulse Interval (RPI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup in Experiment 1. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds. The significance of deviation from pretask baseline for each change is indicated.

(Math=mental arithmetic, Prob=problem solving, Reac=reaction time, Pers=personal tempo).

		Acceleratory	Concurrently Measured	
Task	N	RPI	IBI	PTT
Math	38	-11.0*	-73*	+0.35
Prob	39	-10.3*	-61*	-0.09
Reac	36	- 7.2*	-17*	+0.87
Pers	24	- 6.1*	- 1	+1.19

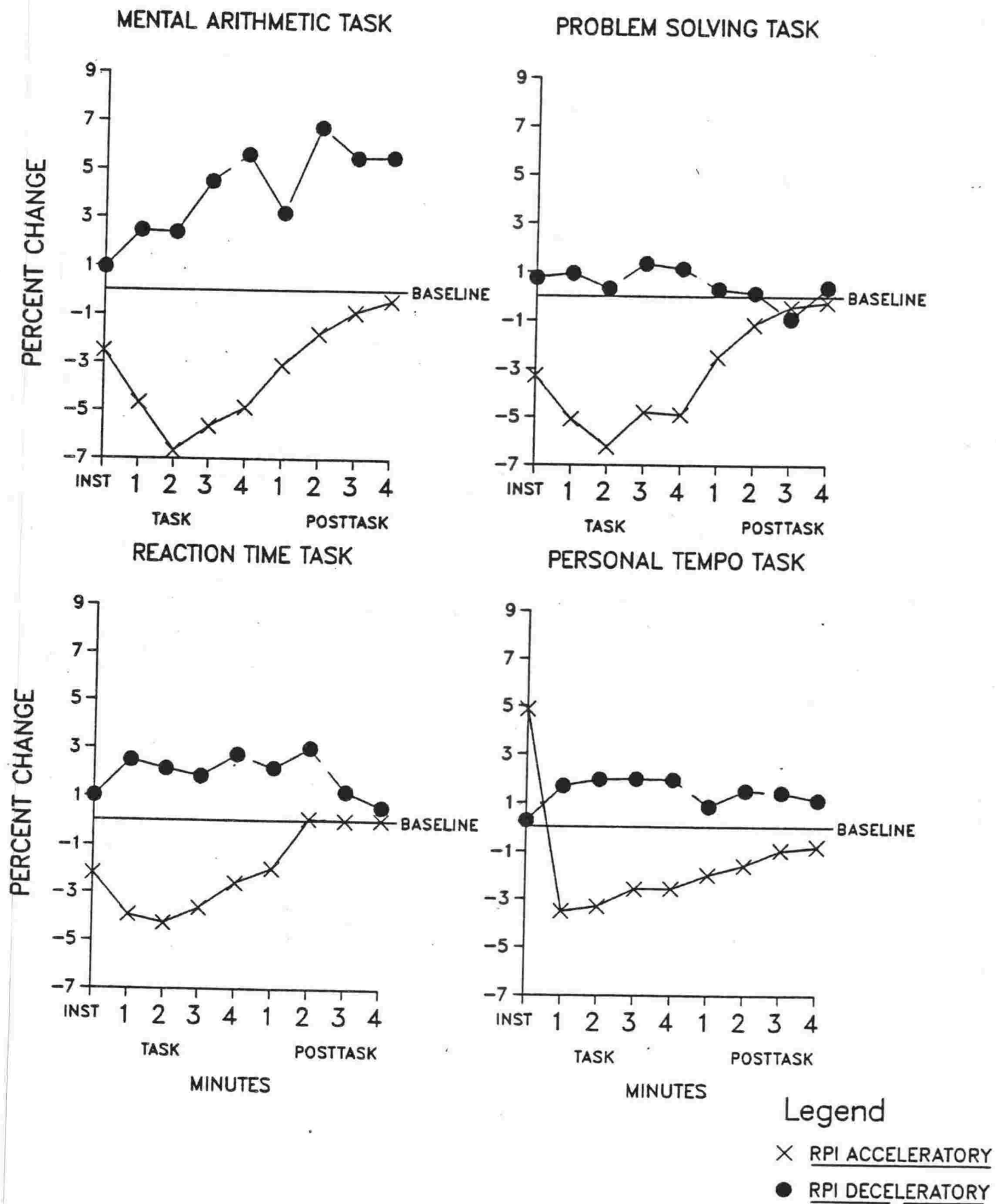|		Deceleratory	Concurrently Measured	
Task	N	RPI	IBI	PTT
Math	12	+7.3*	-69*	-12.6*
Prob	11	+1.5	-57*	- 6.2*
Reac	14	+4.3*	+ 1	- 0.9
Pers	26	+3.6*	+ 2	- 1.5

*   p<.05 or better

Figure N. Minute—by—minute R-wave to pulse interval acceleratory and deceleratory responses, Experiment 1.

Table C

R-wave to Pulse Interval (RPI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup in Experiment 2. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds. Systolic (SBP) and Diastolic (DBP) changes are shown in mmHg. The significance of deviation from pretask baseline for each change is indicated. (Easy=easier problems, Hard= harder problems).

		Acceleratory	Concurrently Measured			
Task	N	RPI	IBI	PTT	SBP	DBP
Easy	15	-4.7*	-50*	+6.5*	+0.6	+0.2
Hard	16	-8.3*	-59*	+0.5	-15.2	-7.5

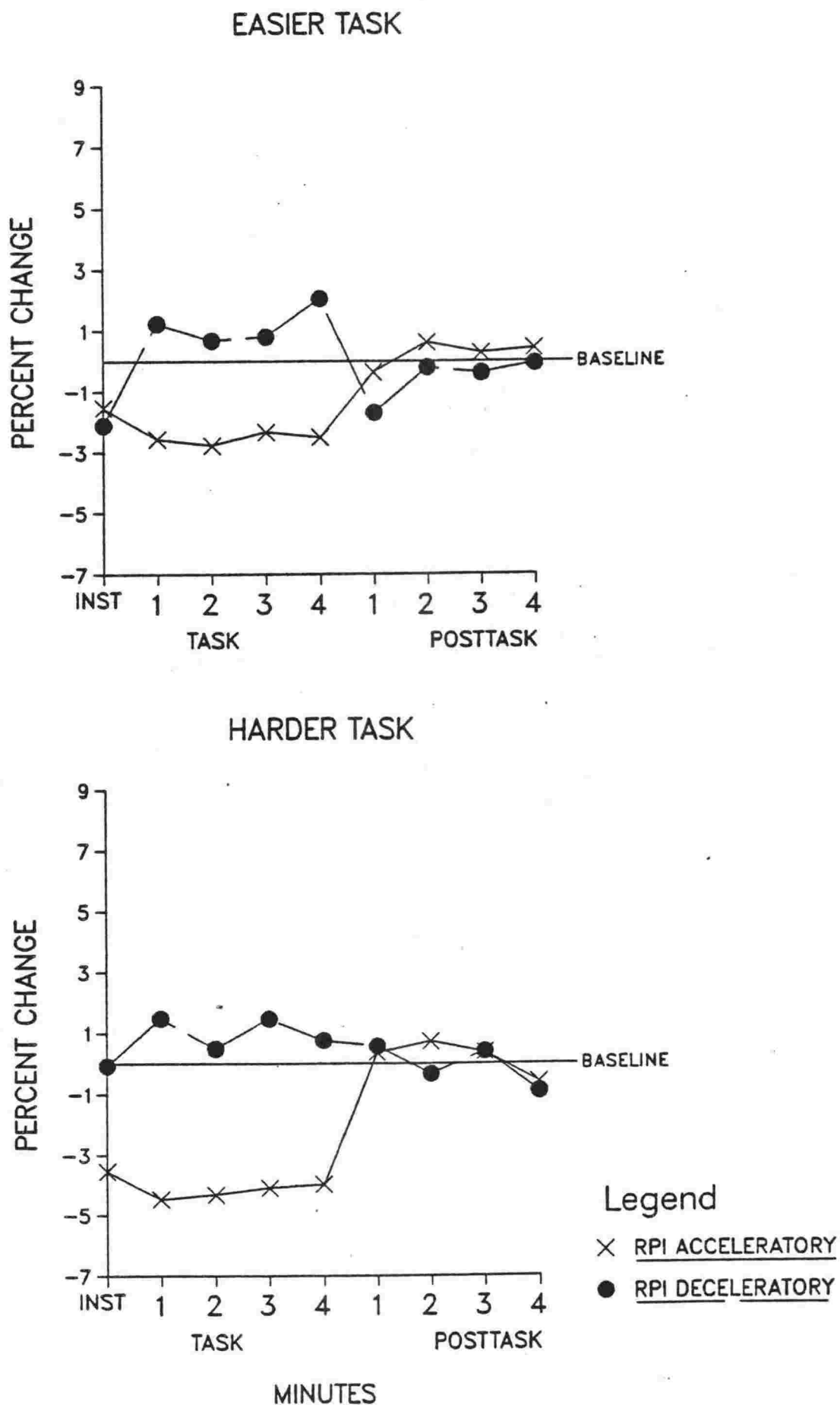|		Deceleratory	Concurrently Measured			
Task	N	RPI	IBI	PTT	SBP	DBP
Easy	4	+2.2*	-31	-4.03	-17.1	-11.8
Hard	3	+1.81	-12	-4.1	-1.0	-0.3

* p<.05 or better

Figure O. Minute—by—minute R—Wave to Pulse Interval acceleratory and deceleratory responses, Experiment 2.

Table D

R-wave to Pulse Interval (RPI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup in Experiment 3. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds. Systolic (SBP) and Diastolic (DBP) changes are shown in mmHg. The significance of deviation from pretask baseline for each change is indicated. (Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

Task	N	Acceleratory RPI	Concurrently Measured IBI	PTT	SBP	DBP
Easy	22	-4.5*	-46*	-0.6	+0.9	+1.0
Hard	20	-6.0*	-65*	+0.5	+2.6*	+1.9
Impos	18	-4.2*	-57*	+1.3	+0.3	+3.83*
Pers	14	-1.8*	-13	+0.1	+0.7	+0.1

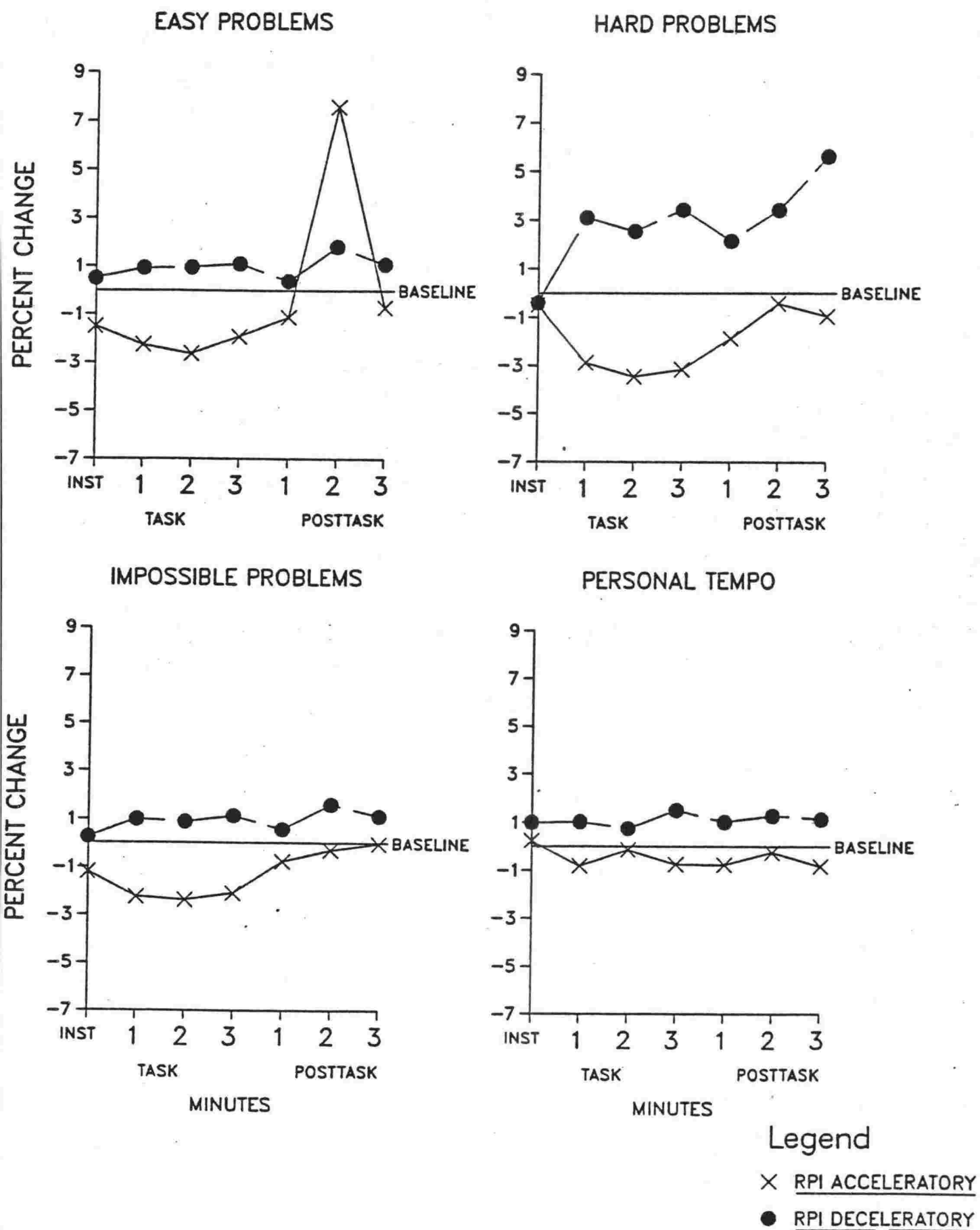| Task	N	Deceleratory RPI	Concurrently Measured IBI	PTT	SBP	DBP
Easy	8	+1.8*	-62	-0.75	+ 4.1	+ 2.8
Hard	10	+5.2*	-80*	-5.4*	-0.5	+3.0
Impos	11	+2.0*	-10	+0.61	- 1.8	+ 2.2
Pers	15	+2.2*	- 7	+1.3	-1.6	+0.6

* p<.05 or better

Figure P. Minute—by—minute R—Wave to Pulse Interval accleratory and deceleratory responses, Experiment 3.

Table E

R-wave to Pulse Interval (RPI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup in part 1 during Experiment 4. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds. Systolic (SBP) and Diastolic (DBP) changes are shown in mmHg. The significance of deviation from pretask baseline for each change is indicated. (Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

		Acceleratory		Concurrently Measured		
Task	N	RPI	IBI	PTT	SBP	DBP
Easy	19	-4.6*	-49*	+0.1	+3.3*	+0.8
Hard	17	-5.0*	-73*	+0.3	+1.8	+2.6
Impos	9	-5.4*	-64*	-0.1	+1.9	+0.6
Pers	15	-2.4*	-14	-0.1	-0.9	+0.7

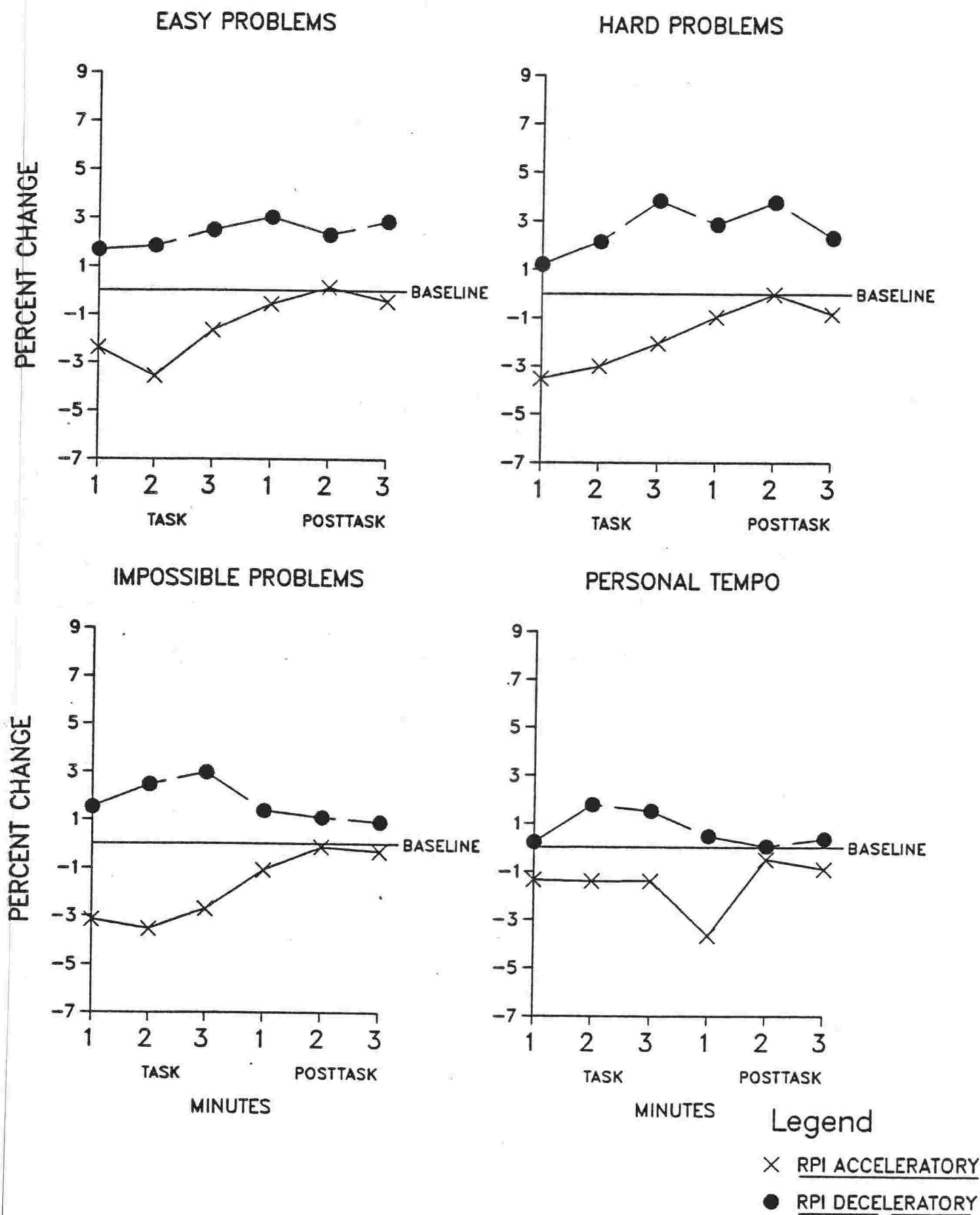|		Deceleratory		Concurrently Measured		
Task	N	RPI	IBI	PTT	SBP	DBP
Easy	11	+3.2*	-33	+0.3	- 2.2	+ 0.6
Hard	13	+4.0*	-50*	+0.5	-2.5	+2.0
Impos	21	+4.1*	-55*	+0.4	- 1.7	+0.9
Pers	15	+2.2*	- 8	+0.1	-1.9	-1.5

* p<.05 or better

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Figure Q. Minute-by-minute R-Wave to Pulse Interval
acceleratory and deceleratory responses,

Part 1, Experiment 4.

Table F

R-wave to Pulse Interval (RPI) Response Changes (in milliseconds) from pre-task baseline levels for each subgroup in part 2 during Experiment 4. The concurrently measured Interbeat Interval (IBI) and Arterial Pulse Transit Time (PTT) changes are also shown in milliseconds. Systolic (SBP) and Diastolic (DBP) changes are shown in mmHg. The significance of deviation from pretask baseline for each change is indicated. (Easy=easy problems, Hard=hard problems, Impos=impossible problems, Pers=personal tempo).

		Acceleratory		Concurrently Measured		
Task	N	RPI	IBI	PTT	SBP	DBP
Easy	19	-5.1*	-50*	-0.2	+0.1	+3.0*
Hard	22	-4.6*	-63*	-0.4	+0.6	+2.4
Impos	19	-4.8*	-45*	-0.6	+0.9	+1.3
Pers	13	-2.9*	-17	-0.5*	-2.8*	-0.9

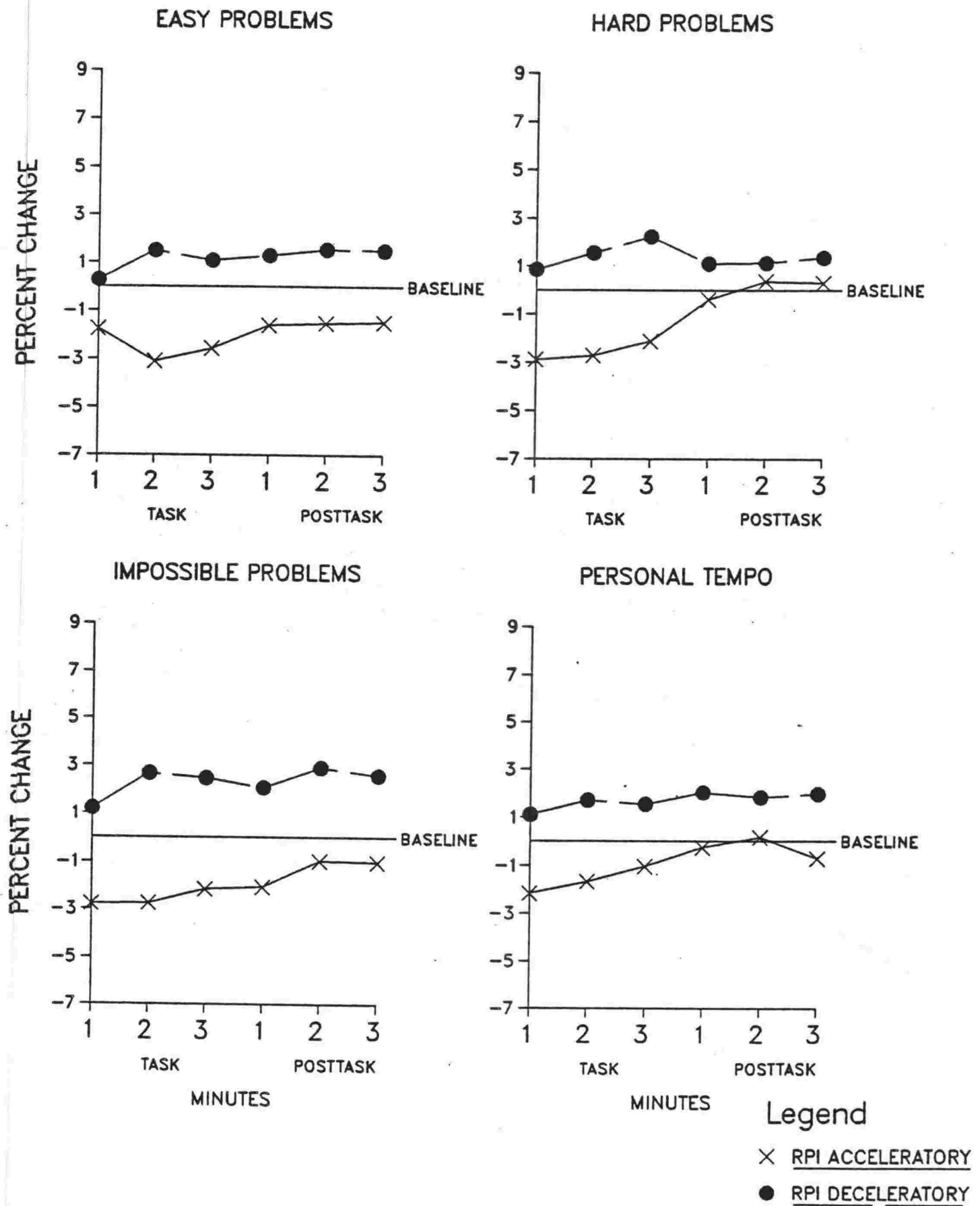|		Deceleratory		Concurrently Measured		
Task	N	RPI	IBI	PTT	SBP	DBP
Easy	11	+1.6*	-54*	+0.4	+ 0.8	+ 3.0
Hard	13	+2.7*	+25	+0.4	-1.4	-0.5
Impos	21	+3.6*	-59	+0.3	-1.2	+1.0
Pers	15	+2.5*	-11	+0.1	+0.8	+1.9

* p<.05 or better

Figure R. Minute—by—minute R—Wave to Pulse Interval acceleratory and deceleratory responses, Part 2, Experiment 4.

DISCUSSION

The magnitude of changes in RPI appears to be sensitive to differences in task difficulty as an effect of task was found in all four experiments. However, when the figures are compared, it is found that the magnitude of change decreased, and in addition the relationship between the magnitude of RPI change and task difficulty reversed over multiple recordings: in Experiments 1 and 2 the more difficult tasks generated larger acceleratory change, suggesting a linear relationship between response magnitude and difficulty; but in Experiments 3 and 4, the easy task generated larger acceleratory RPI changes suggesting a negative linear relationship between response magnitude and task difficulty.

It may be that task novelty interacts with task difficulty to determine the magnitude of RPI change (Light & Obrist, 1980a; Obrist, 1982). The present data suggest that increased familiarity with task events may not only reduce the magnitude of the acceleratory change (Obrist, 1982), but may also change the relative response to tasks as a function of task difficulty. To the extent that changes in RPI reflect changes in sympathetic influence on the myocardium, and that behaviourally-induced increases in that influence contribute to stress-related cardiovascular dysfunction, there would appear to be a need to clarify the effect of familiarity as a determinant of cardiac responding.

2.1 CALCULATION OF LEFT VENTRICULAR EJECTION TIME

As described, RPI is the time interval which begins at the R-wave of the EKG and terminates with the detection of the pulse at an arterial site and is comprised of intra-cardiac components in addition to a pulse propagation component (Newlin & Levenson, 1979; Pollack & Obrist, 1983; also see Chapter 2). In particular, RPI includes both the isovolumetric contraction period, the onset of which coincides with the R-wave of the EKG, and the LVET which begins at the end of the isovolumetric contraction period when the semi-lunar valves open and the pressure pulse in the left ventricle is ejected into the aorta, and that in the right ventricle is ejected into the pulmonary artery (coinciding with the S-wave of the EKG). LVET terminates with the closure of the aortic valve which generates the second heart sound.

The particular value of RPI in psychophysiological research has been in its relationship with the cardiac pre-ejection period (PEP), the onset of which coincides with the Q-wave of the EKG and which includes the isovolumetric contraction period, ending when the semi-lunar valves open. PEP is understood to be the only myocardial event which is primarily governed by sympathetic activity (Randall, 1976) and both RPI and QPI (which includes the entire PEP) have been found to change in parallel with PEP, and to be relatively uninfluenced by the pulse propagation rate (Newlin, 1981; Newlin & Levenson, 1979). Thus RPI has been considered as a stable non-invasive measure which reflects sympathetic influences on the myocardium (Obrist, et al., 1979; Newlin, 1981), and which can be also used to isolate and study cardiac events

which are difficult to measure noninvasively, such as PEP and LVET (Pollack & Obrist, 1983; Newlin, 1981) but which are of particular biological significance in that the relative duration of both PEP and LVET provides direct information on the contractility of the myocardium.

The facilities available and the techniques used in the present research enabled the calculation of the intracardiac components of RPI, specifically the combined duration of LVET and the isovolumetric contraction period, and that calculation was performed in Experiment 5.

In order to avoid the repeated use of the assumption that pulse propagation rate is equal when travelling toward the auricular artery and the dorsalis pedis artery (see Chapter 6), the duration of the R-wave to the dorsalis pedis arterial site was employed for the calculation by summing the recorded values of RPI to the auricular detection site and PTT to the dorsalis pedis detection site.

The measured distance between manubrium sternum and the dorsalis pedis arterial detection site was divided by the calculated value of PWV (see Chapter 6) to give an estimate of the actual time required for the pulse to travel the full distance between the aortic arch and the dorsalis pedis arterial site. That value was then subtracted from the sum of the recorded PTT and RPI (R-wave to dorsalis pedis arterial site) to give an estimate of the LVET which also included the isovolumetric contraction period of the PEP.

APPENDIX 3- ANALYSES OF ABSOLUTE LEVELS

Absolute levels of IBI, PTT, and except in Experiment 1, SBP and DBP during the instruction, task and post-task periods were analysed for evidence of sex differences only over Experiments 1, 2, and 3 (Sex ANOVA).

The absolute levels of IBI, PWV, SBP and DBP during Experiment 4 task and post-task periods were analysed using a Sex (2) X Condition (2) X Part (2) ANOVA with repeated measures on the last factor. The results of these analyses are presented below.

EXPERIMENT 1

Instruction, Task and Post-task Periods:. There was no Main Effect of Sex found on the absolute levels of cardiovascular responding during any of the periods in Experiment 1.

EXPERIMENT 2

Instruction, Task and Post-task Periods:. There was no Main Effect of Sex found on the absolute levels of cardiovascular responding during any of the periods in Experiment 2.

EXPERIMENT 3

Instruction Periods:. A Main Effect of Sex was found for SBP ($F(1/28)=14.03$, $p<.0006$). Women had lower SBP than men.

Task Periods:. A Main Effect of Sex was found for SBP ($F(1/28)=6.93$, $p<.01$). Women had lower SBP than men.

Post⁻task Periods:. A Main Effect of Sex was found for SBP ($F(1/28)=9.70$, $p<.004$). Women had lower SBP than men.

The sex differences found in SBP during each experimental condition are shown in Table G.

EXPERIMENT 4

Task Periods:. A Main Effect of Part ($F(1/26)=14.0$, $p<.0009$) was found for IBI. Levels of IBI during tasks in part 1 of the experimental session were faster than those in part 2 (796ms vs 827ms).

A Main Effect of Sex ($F(1/26)=5.73$, $p<.02$) was found for PWV during tasks. Women had slower PWV than men.

A Main Effect of Sex ($F(1/26)=8.26$, $p<.008$) was found for SBP during tasks. Women had lower SBP than men.

A Main Effect of Part ($F(1/26)=9.22$, $p<.0006$) was found for SBP. The level of SBP during tasks was lower in part 1 of the experimental session than in part 2 (114.19mmHg vs 117.16mmHg).

There was a Condition x Part Interaction (F(1/26)=8.21, p<.008) for SBP during tasks. Subjects who had consumed caffeine had an increase in level of SBP, those who had not consumed caffeine did not.

A Main Effect of Part (F(1/26)=16.67, p<.0004) was found for DBP. Levels of DBP were lower during tasks in part 1 of the experimental session than during those in part 2 (67.87mmHg vs 72.23mmHg).

There was a Condition x Part Interaction (F(1/26)=7.78, p<.01) for DBP. Subjects who had consumed caffeine had an increase in level of DBP, but those who had not consumed caffeine did not.

The sex differences in PWV are shown in Table H, those in SBP are shown in Table I.

The Condition X Part Interaction for SBP is shown in Table J, that for DBP is shown in Table K.

Post-task Periods. There was a Main Effect of Part (F(1/26)=10.85, p<.003) on IBI. Levels of IBI were faster during the post-task periods in part 1 of the experimental session than during those in part 2 (827ms vs 852ms).

There was a Main Effect of Sex (F(1/26)=5.84, p<.02) for PWV during post-task. Women had slower PWV than men.

There was a Main Effect of Sex (F(1/26)=9.91, p<.004) for SBP during post-task. Women had lower SBP than men.

There was a Main Effect of Part (F(1/26)=12.06, p<.002) for SBP. Levels of SBP during the post-task periods in part 1 of the experimental session were lower than during those in part 2 (114.12mmHg vs 117.39mmHg).

There was a Condition x Part Interaction (F(1/26)=6.79, p<.01) for SBP during post-task. Subjects who had consumed caffeine had an increase in level of SBP, those who had not consumed caffeine did not.

There was a Main Effect of Part (F(1/26)=18.86, p<.0002) for DBP. Levels of DBP during post-task periods in part 1 of the experimental session were lower than during those in part 2 (68.18mmHg vs 72.58mmHg). .

There was a Condition x Part (F(1/26)=12.97, p<.001) Interaction for DBP during the post-task periods. Subjects who had consumed caffeine had an increase in level of DBP, those who had not consumed caffeine did not.

The sex differences in PWV are shown in Table H, those in SBP are shown in Table I.

The Condition X Part Interaction for SBP is shown in Table J, that for DBP is shown in Table K.

TABLE G

Systolic (SBP) and Diastolic (DBP) Blood Pressure levels in mmHg for men and women during the instruction, task and post-task periods in Experiment 3. A Main Effect of Sex was found for all SBP comparisons.

		Men (n=16)		Women (n=14)
Instruction Periods	SBP	119.39	*	107.98
	DBP	66.31		70.34
Task Periods	SBP	119.80	*	110.30
	DBP	69.45		71.80
Post-task Periods	SBP	120.47	*	110.02
	DBP	68.33		71.11

* p<.05 or better

TABLE H

Pulse Wave Velocity levels in m/sec for men and women during the task and post-task periods in Experiment 4. A Main Effect of Sex was found for all comparisons.

		Men (n=16)		Women (n=14)
Task Periods	PWV	12.55	*	10.68
Post-task Periods	PWV	12.50	*	10.64

* p<.05 or better

TABLE I

Systolic (SBP) and Diastolic (DBP) Blood Pressure levels in mmHg for men and women during the task and post-task periods in Experiment 4. A Main Effect of Sex was found for all SBP comparisons.

		Men (n=16)		Women (n=14)
Task Periods	SBP	120.85	*	109.76
	DBP	68.97		71.29
Post-task Periods	SBP	121.24	*	109.48
	DBP	69.47	.	71.41

*   p<.05 or better

TABLE J

Systolic Blood Pressure (SBP) levels in mmHg recorded in the task and post-task periods during each part of Experiment 4 for subjects who ingested caffeine and those who did not. The tabled data show the Condition (Caffeine/No Caffeine) X Part (Part 1/Part 2) Interaction reported in the text.

		Caffeine	No Caffeine
		Task Periods	
Part 1	SBP	114.22	114.17
Part 2	SBP	119.98	114.33
		Post-task Periods	
Part 1	SBP	115.32	112.92
Part 2	SBP	121.05	113.73

* $p < .05$ or better

TABLE K

Diastolic Blood Pressure (DBP) levels in mmHg recorded in the task and post-task periods during each part of Experiment 4 for subjects who ingested caffeine and those who did not. The tabled data show the Condition (Caffeine/No Caffeine)

X Part (Part 1/Part 2) Interaction reported in the text.

		Caffeine	No Caffeine
		Task Periods	
Part 1	DBP	66.18	69.55
Part 2	DBP	73.53	70.93
		Post-task Periods	
Part 1	DBP	66.68	69.68
Part 2	DBP	74.72	70.43

* p<.05 or better

APPENDIX 4- RECOVERY RESPONSES IN POST-TASK PERIODS

The change scores during the post-task periods were analysed using identical analyses performed on the corresponding task data. Thus in Experiment 1 post-task recovery data were analysed using a Sex X Task X Minute X Session ANOVA with repeated measures on the last three factors; in Experiments 2 and 3 post-task recovery data were analysed using Sex X Task X Minute ANOVA with repeated measures on the last two factors; and in Experiment 4 post-task recovery data were analysed using a Sex X Condition X Part X Task ANOVA with repeated measures on the last two factors. Secondary analyses which substituted Task Order for Task were also performed on each set of data.

It should be noted that in all cases response levels returned to approximately pre-task baseline levels during the post-task recovery periods.

Absolute levels of IBI, PTT, SBP and DBP during post-task are presented in Appendix 3; those for RPI are presented in Appendix 2.

EXPERIMENT 1. A Sex X Task Interaction ($F(3/144)=5.11$, $p<.001$) was found for IBI post-task recovery. The women had acceleratory responses in the post-task periods following the mental arithmetic and reaction time tasks but the men had deceleratory responses in the post-task periods following those two tasks; further the women had deceleratory responses in the post-task periods following the problem solving and personal tempo tasks but the men had acceleratory responses in the post-task periods following those two tasks.

An Order Effect (F(3/144)=4.80, p<.003) was found for IBI post-task recovery. During the post-task period following the first task in each session, there was a deceleratory IBI response, but during the post-task period following the second task in each session there was an acceleratory IBI response.

An Order Effect was found for PTT recovery during post-task (F(3/144)=3.84, p<.01) and that effect is shown in Figure 1.2 in Chapter 3. Relative PTT deceleration followed tasks 1 and 4 and relative acceleration following tasks 2 and 3.

EXPERIMENT 2. No significant effects or interactions were found.

EXPERIMENT 3. There was a Main Effect of Tasks (F(3/84)=3.0, p<.03) for the recovery of IBI during the post-task periods. Follow-up means tests showed that post-task recovery was more marked following the personal tempo and impossible tasks than during the hard or easy tasks.

No other effects or interactions were significant.

EXPERIMENT 4.

There was a Part x Task Interaction (F(3/78)=2.9, p<.04) found for post-task recovery in IBI. The recovery responses following the hard and impossible tasks were acceleratory in both parts of the experimental session with larger magnitude changes in part 1 (-1.43% and -1.96% in part 1 versus -1.22% and -1.24% in part 2); the recovery responses following the easy task in each part were both acceleratory with the larger response occurring in part 2 (-0.66% versus -2.63%); and the recovery responses following the personal tempo

There was a Sex x Part x Condition x Task (F(3/78)=3.14, p<.03) found for post-task recovery in PWV. That interaction is shown in Figure S. (The corresponding task data is shown in Appendix 1, Figure G).

There was a Main Effect of Condition (F(1/26)=5.98, p<.02) found for post-task recovery in SBP. Caffeine consumption was associated with an increase in SBP during recovery, no caffeine consumption was associated with a decrease (+0.70mmHg for caffeine versus -0.79mmHg for no caffeine).
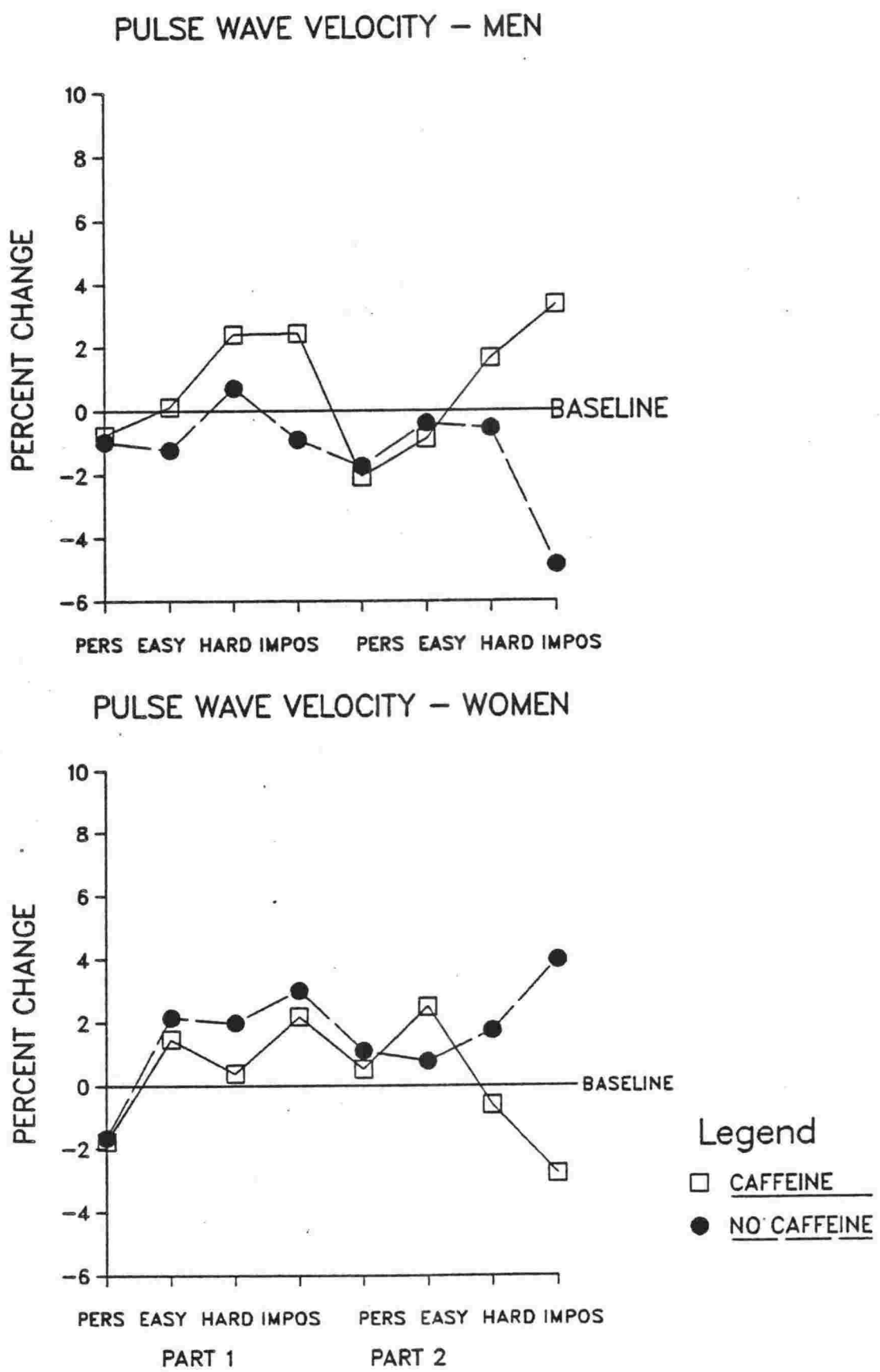
Figure S. Pulse Wave Velocity post–task recovery response during Experiment 4 showing the Sex X Part X Condition X Task Interaction reported in the text (PERS=personal tempo; EASY=easy problems; HARD=hard problems; IMPOS=impossible problems).

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