CHAPTER 1 -- INTRODUCTION
1.1 SETTING

The subject of this thesis is an examination of the possible association between child blood pressure (BP) and exposure to noise emanating from overflights of passenger jet aircraft. The study participants were primary school children attending a sample of schools within a 20 km radius of Sydney (Kingsford-Smith) Airport. The BPs of the children were measured longitudinally in 1994-95 and again in 1997, and were related to measured aircraft noise exposures at school and at home at both time points. This investigation of child BP and aircraft noise has a precedent in a 1980 study which found mean resting BP to be ≈3 mmHg higher in children from aircraft noise-affected schools around Los Angeles Airport, compared to children in quieter schools [Cohen et al, 1980]. A later study on child BP and aircraft noise was also conducted in the mid-1990s in relation to the new Munich International Airport [Evans et al, 1998].

The Inner Sydney Child BP study, the official rubric of this study, was prompted and funded by the then Federal Airports Corporation as part of an Environmental Impact Study (EIS) into the construction of a new runway at Sydney Airport. The EIS covered broad environmental, economic and health issues associated with the new runway. The new runway opened 6 months earlier than scheduled, on 3 November 1994, and provided natural experimental conditions for the study of possible effects of aircraft noise on populations subject to changing aircraft noise exposures.

The commencement of jet aircraft operations on commercial air routes in the 1950s was associated with major increases in noise and disruption to residents living near large airports. Since the 1960s, when jet aircraft came to dominate domestic air traffic, increases in complaints to public officials and airports have been a catalyst for research into the effects of aircraft overflights [Bullen, 1984]. The growth in the literature has reflected continuing and increasing concerns about the effects of aircraft noise on the physical and mental health of exposed populations. Populations living near or under the flight paths of Heathrow (London), Los Angeles and Schiphol (Amsterdam) airports, in particular, have been studied in some detail.

Aircraft noise may produce a variety of psycho-social and economic effects on humans which include: interference with quality of life and amenity [Stockbridge et al, 1973] [Bronzaft et al, 1998; Moran et al, 1981], declines in property values [Kryter, 1994]
Research on the health effects of aircraft noise has several points of departure. In the least rigorous studies, exposed individuals relate not only what they consider to be health concerns but also attribute their cause to aircraft noise exposure (e.g., complaint hotlines, etc). For stronger evidence, health practitioners may put together case series of instances in which an adverse health effect may plausibly be attributable to environmental noise exposure on the basis of known patho-physiology. The best approach is open-ended or hypothesis-driven studies which seek to identify possible adverse health outcomes in populations (or population samples) by separately measuring noise exposure and hypothesised health effects in individuals, and changes in these over time. Precluding randomised controlled trials on ethical and practical grounds, the strongest possible study design for establishing evidence for a causal relationship in populations is the natural experiment whereby some populations become newly exposed to the noise source, other populations become less- or non-exposed, and where before-and-after measures of the hypothesised health outcomes in these populations are available.

Opportunities to conduct a natural experiment are limited to situations where major changes in aircraft noise exposure regimes occur, usually as a result of newly built airports or major reconfigurations of existing facilities (e.g., runway extensions, additions). Consequently, cross-sectional studies, which examine health outcomes in populations experiencing only static noise exposure differences, or where noise exposures and health outcomes are measured once only and compared, are much less able to establish a causal relationship between the study or exposure variable and outcome variable. Establishing causation in such studies is difficult because changes in exposure and corresponding changes in outcome are not measured in the same experimental subjects. Even established cross-sectional associations between different subjects exposed to different conditions are likely to be severely affected by selection bias, particularly in ecological (aggregate) cross-sectional observational comparisons, where health effects purportedly associated with an environmental exposure are confounded by socio-economic and other differences in the groups under comparison.

The following sections outline the extant literature pertaining to blood pressure and its
measurement, aircraft noise and its measurement, and the evidence for a relationship between exposure to aircraft noise and blood pressure.

For this literature review, the possible (non-auditory) health effects of noise have been classified into psychological effects, and acute physiological effects with reference to acute and possibly chronic cardiovascular effects, effects on general morbidity and mortality of populations, and perinatal effects. The main explanatory pathways proposed for physical health effects of noise is that they may be mediated by “stress” (including psycho-social stress), anxiety and/or perceived lack of control over the source of exposure [Littman, 1993]. Furthermore, annoyance and cognitive effects of noise, and ideation about the possibility of an airline crash in the context of aircraft noise may merge into psychological effects, particularly in the presence of additional effect modifiers (eg, property ownership, attitudes) which may then manifest as acute and possibly chronic physical health effects.

However, populations living proximate to aircraft flight paths tend to be of lower socio-economic status (SES) than those that do not, with property values serving as the chief selection mechanism. Levels of morbidity and mortality among low SES populations have long been known to be significantly higher than in populations of average or high SES [Morris et al., 1944]. As a major source of potential confounding, SES is particularly difficult to correct, especially in ecological studies. Again, complicating the picture is the possible influence of modifying factors such as, in adults, ownership of residence (and concern with property values); individual sensitivity to noise; the role of aircraft noise in interfering with other activities; and the fear of an airline crash. Moreover, as a result of the combination of exposure and modifying factors on population migration, people living under flight paths in the long term could well be a selected population in that they would consist of those who most successfully adapted to, or were least adversely affected by, the exposure. Where populations undergo changes in exposure to aircraft noise, the opportunity arises for studying possible health effects in the affected population associated with exposure change. The chief advantage of such a quasi-experimental study design is that the same population is studied under changing exposure conditions, with the study subjects serving as their own controls. Confounders of a noise and health relationship, present under stable or static exposure conditions, will not necessarily change in concert with changes in the exposure condition. This is because changes in aircraft noise exposure usually occur faster than the rate of significant out-migration and
replacement because of the need to sell or find alternative accommodation in less exposed areas (ie, less selection bias).

Adverse health effects may also ensue from measures taken to reduce exposure to noise in the domestic environment, such as reductions in outdoor physical and social activities. Sealing and closure of residences for sound insulation may reduce ventilation and increase the spread of airborne infections, or encourage the proliferation of dustmite and thus increase the prevalence of asthma.

Finally, an important scenario to consider is that physical effects of noise exposure may affect susceptible subgroups through psychologically mediated aggravation of existing physical or mental conditions or disorders, or precipitate complications of such conditions. Likely scenarios include triggering of dysrhythmias in those with heart disease, or acute psychotic episodes in those with mental illness. Tinnitus sufferers and others sensitive to noise may also experience increased psychological distress or exacerbation of existing conditions when exposed to unwanted noise. On the other hand, deafness may be an advantage. For instance, in relation to blood pressure, a study by Wu et al found BP to be significantly lower in deaf mutes compared to subjects with normal hearing, after adjusting for age [Wu et al, 1993].

Elevated blood pressure has been shown to be associated with increased risk for stroke and cardiovascular disease. The strongest evidence for this comes from randomised trials of antihypertensive medications and from prospective observational studies where highly significant inverse relationships have been established [Collins et al, 1990]. From prospective studies for instance, a long-term reduction of 5-6 mm Hg in diastolic BP has been associated with about 35-40% less stroke and 20-25% less coronary heart disease (CHD) [Collins et al, 1990].

Exposure to noise has been linked to elevated blood pressure, both in the laboratory and in more naturalistic settings, longitudinally in the same individuals and cross-sectionally in different individuals, compared to no exposure to the noise. Ample laboratory evidence exists for increased sympathetic nervous activity and hormonal output in subjects exposed to noise, especially as acute effects [Andren, 1982; Slob et al, 1973], but also as residual effects lasting up to an hour after cessation of exposure to the noise [Andren et al, 1980]. Observational studies of both the general population and occupational
groups also have found exposure to noise to be associated with elevated blood pressure [Thompson, 1983]. The findings have not been consistent [Cartwright et al, 1975; Thompson, 1993], but have been more consistent in cross-sectional than in longitudinal studies [Thompson, 1983]. Overall, the magnitude of acute BP elevations found to be associated with noise exposure have ranged between 0 and 10 mmHg (systolic) [Thompson, 1993].

The effects to amenity by exposure to aircraft overflights, for example television reception, may precipitate hostility and anger, which may in turn lead to psychological morbidity. Furthermore, such morbidity may become associated with exposure to aircraft noise itself, by associative learning, without interference with amenity necessarily occurring on each overflight occasion.

The quality of evidence for the role of noise exposure on blood pressure, in light of the other documented influences on blood pressure, is examined in the following sections.

1.2 Review of studies of aircraft noise and non-aural health effects

This section has 3 parts. The first examines the role of blood pressure in health and factors associated with BP variability; the second examines noise, its measurement and its relation to health; the third section examines the theoretical basis for noise exposure being associated with health, particularly the psycho-physiological mechanisms for an association between noise exposure and blood pressure.

1.2.1 Blood Pressure: historical antecedents, definitions

Blood pressure has been studied as a vital sign for centuries. When measured non-invasively, blood pressure represents a measure of both the force of the cardiac stroke and of the stiffness of the main arterial blood vessels, particularly the aorta (systole), and of peripheral resistance (diastole) [Seeley et al, 1989](pp.594-598).

1.2.1.1 Brief historical sketch of blood circulation, pulse and blood pressure

The earliest known written record alluding to the biological role of the heart is the Edwin Smith Surgical Papyrus (named after the papyrus’s purchaser, an American Egyptologist
who acquired it in Luxor in 1862). The papyrus dates from c.1500 before the current era (BCE), and is believed to be a copy of an earlier manuscript from c.3000 BCE [Venzmer, 1972](p.38). The content of the papyrus centred on detailed instructions for setting broken bones, especially cranial injuries and spine trauma, presented in 48 case studies. The papyrus was overwhelmingly empirical and attempted a rational organisation of the bodily systems, rejected demonic explanations of illness in particular, but not necessarily magical invocations. The papyrus also contained references to the role of the heart as a pump and the use of pulse to determine the state of health of the patient. The heartbeat was assumed to be connected with the pulse beat [Venzmer, 1972](pp.38-39), and a feeble pulse with fever, for example, was associated with hopeless injuries that should not be treated [Horrax, 1952]. The Egyptians did not connect the pulse or heartbeat with circulation because they did not know of the circulation’s existence [Venzmer, 1972](p.38).

According to *The Theory of the Body’s Interior or Nei-ching*, thought to be written in c.2600 BCE by the Chinese emperor Huang-Ti, two kinds of blood existed, each coinciding with either Yin or Yang which circulated continuously as ‘The river of blood traces a circle and never ends...’ [Venzmer, 1972](p.42). The *Nan-ching*, probably written by the renowned Chinese doctor Pien Ch’io under the Chou dynasty in c.500-600 BCE, describes in poetic and bucolic imagery the variety of pulses and their implicit diagnoses. For example, pulses could be described variously as “beating deep and strong like a thrown stone”, as “loose and slow like a willow dancing in a spring breeze”, or as “sounding like a sickle, first exuberant then dying away”. Chinese medicine worked under the strict autopsy prohibition of ancestor worship which, combined with centuries of relative geographic isolation, led to the development, and arguably over-development, of a variety of non-invasive and indirect diagnostic and therapeutic methods. These include acupuncture and moxa, and early systems of pathology and pharmacy.

Pien Ch’io founded the complicated system of Chinese sphygmology which was supposed to diagnose any illness from the pulse alone [Venzmer, 1972](pp.44-45). Each organ was supposed to have its own pulse and if the overall pulse was not ‘harmonious’ then this reflected illness [Osler, 1921](Ch.6). Sphygmology was but one indirect method, and nowhere more developed than in China. Following the construction of the Great Wall under the Han emperors, Chinese sphygmology developed further, to the extent that a pulse had to be taken from 11 different parts of the body, with 51 different
types of pulse delineated [Venzmer, 1972](pp.45-46). By c.100 BCE, contemporaneous to the establishment of the Old Silk Road, the Chinese version of the circulation system was more advanced than anything in the West up until William Harvey [Osler, 1921](Ch.6).

The heart itself was regarded as the seat of wisdom in the Nei-ching, and by c.100 BCE was classified by the Chinese as one of the five ‘firm’ organs [Venzmer, 1972](pp.43-44). Yet the heart still was not described in any physically or biologically meaningful way despite the lacunae of 2,500 years.

The Greek philosopher, and charlatan, Empedocles (ca. 492-440 BCE) is credited as the originator of the tenet that all matter is composed of the four ‘elements’ earth, air, fire and water, along with Love and Strife. Empedocles was the first to show that air was a definite substance [Russell, 1974](pp.71-75). Empedocles was also the first to attempt to explain respiration in physiological terms [Derenne et al, 1994], and is credited as the first to recognise the heart as the centre of the circulation system [Osler, 1921](Ch.7). Empedocles saw the heart along with blood as the ‘humour’ representing the ‘element’ fire in the body. However, it was Aristotle’s views of the functioning of the heart which influenced Western thinking right up until the 17th century [Derenne et al, 1994].

Aristotle saw the heart as central to respiration, but not circulation, where he contended that it caused the lungs to heat up and consequently expand the thorax which resulted in breathing [Derenne et al, 1994]. Praxagoras (c.345 BCE) probably was the first to make the distinction between veins and arteries [Venzmer, 1972](p.88), believing arteries to be hollow vessels for carrying an air-like substance (‘pneuma’) throughout the body [Osler, 1921](Ch.7). By about 275 BCE the Greek physician Erasistratus, whose main claim to fame was to distinguish between the sensory and motor nervous systems, recognised the link between the lungs and the circulatory system [Osler, 1921](Ch.10). Like Aristotle, Erasistratus believed the arteries to be filled with air [Osler, 1921](Ch.10).

Herophilus, a pupil of Praxagoras and a slightly older contemporary of Erasistratus, used a water clock to count pulse and erected a ‘pulse lore’ around the musical theories of the time [Osler, 1921](Ch.10). Herophilus’s sphygmology is regarded as nearly as complicated as that employed by the Chinese [Venzmer, 1972](pp.88-89).

By about 175 AD the “Roman” physician Galen (Galen was of Greek origin, from
Pergamon) was advocating the use of pulse as a diagnostic aid, but failed to recognise the heart as a pump of blood despite his knowledge of heart valves, of the source of the pulse being the walls of the heart and arteries, and of the difference between diastole as an expansion of the heart, and systole as a contraction of the heart [Osler, 1921](Ch.11). Nevertheless, Galen demonstrated that arteries were filled with blood, not air, in spite of arteries emptying of blood on death [Venzmer, 1972] (pp.96-97). Galen taught that the lungs were responsible for charging the blood with ‘vital spirit’ as it passed through them and believed that the pumping heart governed ebb and flow of bodily ‘humours’ and fluids in general, not merely blood. Galen maintained the cardiac septum to be porous which allowed blood to seep through for further transport around the body. Galen distinguished between the venous and arterial systems and observed the arterial/venous connexion (anastomosis) without understanding the communication [Osler, 1921](Ch.11).

While Galen based his knowledge on animal work (human dissection was strictly forbidden by the Romans, unlike the Alexandrians when ruled by Ptolemy), he contended that the heart and lungs in humans were connected by the trachea in order to infuse the blood in the left part of the heart with ‘vital spirit’ [Venzmer, 1972](p.97). As Galen’s empirical work was limited to animal studies (chiefly pigs, monkeys, cats and dogs), the vast majority of his mistaken notions came from erroneously projecting animal findings onto humans. Worse, when Galen died all anatomical and physiological research virtually ceased, due largely to the long term decline of the Roman Empire then reaching its nadir. This situation was further reinforced by the Roman Catholic Church who upheld Galen’s precepts as dogma for the next millennium, to be trifled with at one’s peril [Venzmer, 1972](p.99).

Following the long hiatus of the Dark and Middle Ages, interest in anatomy and the human body in general revived amidst the artistic and cultural ferment of the Italian Renaissance. In the 1530s, under the nose of the Inquisition, Andreas Vesalius (also known as van Wesele and later acknowledged as the father of modern anatomy) in Paris began secretly dissecting the bodies of recently executed prisoners and found that many of Galen’s contentions to be fallacious. However, the heat of the Inquisition drove Vesalius to the University of Padua where his work culminated in the first serious breach of Galen with the publication of his seven volume opus *De Humani Corporus Fabrica* in 1543. Vesalius was able to confirm that air could not directly reach the heart via the
lungs, by pumping air under high pressure into the lungs and observing no air in the heart as a result. Vesalius also studied the heart valves and concluded that they served to allow blood to flow in one direction only [Venzmer, 1972](pp.140-145).

In 1553 Vesalius’s fellow-student Miguel Serveto, doctor and amateur astronomer, geographer, mathematician and theologian, anonymously published a theological work which included his findings that the blood circulated from the heart to the lungs then back to the heart. Not via a ‘porous’ septum but as follows:

That vital spirit, the arterial blood, leaves the lungs laden with strength, heat, air, water and fire and enters the left ventricle. The vital spirit is formed as result of the mixing of air in the lungs with the blood which reaches the lungs from the right ventricle and is afterwards hurled into the left ventricle. This connection is not made across the wall of the heart, but very cunningly: the blood is pumped out of the right ventricle and conducted to the lungs. The lungs make the blood bright and fresh, and then, via the arteries it is passed to the veins, from which it is pumped into the left ventricle and so reaches all arteries in the body [Venzmer, 1972](p.168).

When his authorship was discovered in the same year, John Calvin denounced Serveto to the Catholic Inquisition in the hope he would meet his end via the Papacy. Serveto’s capture by the Inquisition was short-lived but the somewhat foolhardy Serveto returned to Geneva where he was promptly discovered and burnt slowly in damp straw at the stake by the vindictive Calvin after enduring 3 months of torture [Venzmer, 1972](pp.167-170). In 1559 the Italian anatomist Colombo, a successor to Vesalius’s chair at the University of Padua, demonstrated Serveto’s proposal by showing that blood circulated from the right ventricle of the heart to the lungs, then back to the left ventricle. Colombo’s description of pulmonary circulation was the best prior to Harvey [Eknoyan et al, 1997]. In 1603 Girolamo Fabricio of Aquapendente, another successor to Vesalius’s Padua chair, presented a detailed study of venous valves, but with little understanding of their function, describing them as ‘pocket-like’ arrangements whose function was to prevent the blood from collecting ‘in the extremities’ [Venzmer, 1972](p.170).

William Harvey was one of Fabrizio’s students but the function of venous valves would not become known publicly until 1616, when Harvey began lecturing to the Royal College of Physicians [Venzmer, 1972](pp.170-175). Even then Harvey’s lectures appeared not to have had much impact and his findings would only do so with the
publication of his seminal work in 1628 [Harvey, 1628; Osler, 1921](Ch.23). Harvey sought to answer the key question: How much blood is sent into the body after each spasm of the heart? After dissections of hundreds of animals he estimated this volume to be 60 cubic centimetres in humans which translated into 250-270 litres pumped per hour. Clearly such a volume could not be contained in the human body in toto, let alone ‘seep’ through thick tissue like the cardiac septum, and therefore such a finding could be explained only by the blood travelling from the heart through the body and then back again to the heart in a cycle [Venzmer, 1972](pp.172-173):

I began to think whether there might not be A MOVEMENT, AS IT WERE, IN A CIRCLE. Now this I afterwards found to be true; and I finally saw that the blood, forced by the action of the left ventricle into the arteries, was distributed to the body at large, and its several parts, in the same manner as it is sent through the lungs, impelled by the right ventricle into the pulmonary artery, and that it then passed through the veins and along the vena cava, and so round to the left ventricle in the manner already indicated [Osler, 1921](Ch.23).[capitals in original]

Harvey was able to explain the function of venous valves, which eluded Fabrizio, as directing the blood toward the heart, but he was unable to explain how veins and arteries communicated with each other. However, Harvey was the first to use the experimental method to demonstrate conclusively blood’s cyclical travels and thereby completed the overthrow of Galen begun by Vesalius.

The puzzle of artery-vein communication was solved by Malpighi, who in 1660 described in detail the air pockets of the lungs and the surrounding complex of blood capillaries. Malpighi showed definitively that air did not enter blood vessels and he established the communication between the veins and arteries [Mezzogiorno et al, 1995]. By 1669 Richard Lower had described the structure of the heart as a muscle and observed the change of colour of blood in the lungs [Larner, 1992]. In 1707, John Floyer published The physician0’s pulse watch [sic] in which he described the elaborate Chinese system of sphygmology [Osler, 1921](Ch.6). Floyer advocated the use of pulse rates for monitoring of general health, and proposed a special physicianO’s [sic] watch for pulse measurement [Townsend, 1967].

By 1726 the Reverend Stephen Hales had described a cycle of blood pressure variability after directly measuring the blood pressure of a horse. Hales inserted a brass pipe into the left crural (leg) artery of the animal and reported blood rushing ‘in an instant’ to a height
of about four feet above the left ventricle of the horse’s heart [Hales, 1733](cited in [Weder et al, 1987]). In a series of pulses which varied periodically, the column of blood eventually reached a maximum height of 8 feet 3 inches [Weder et al, 1987]. Hales reported a regular blood pressure ‘cycle’ in the horse of about 40-50 pulses, and is thereby credited with the first account of periodic variations in blood pressure unrelated to the pulse rate [Weder et al, 1987]. That is, Hales was able to detect non-pulsatile variations in BP [Weder et al, 1987]. Hales reported his findings in 1733, along with his work on the hydrostatics of plants.

In 1847 Karl Friedrich Willhelm Ludwig, who in 1856 was the first to keep animal organs alive outside the body by maintaining their blood supply, invented a device for the continuous monitoring of blood pulse called the kymograph which drew curves of pulse on a rotating drum of paper [Venzmer, 1972](pp.278-279). With the device Ludwig was able to show that blood circulation was a purely mechanical phenomenon [Schroer, 1996]. Many of Ludwig’s contributions to the understanding of circulation and micro circulation in particular remain valid today [Schroer, 1996].

A device for indirectly (non-invasively) measuring blood pulse, called a sphygmograph (a precursor to the sphygmomanometer), was in use by the mid-19th century, but its invention is credited to the French physiologist Etienne-Jules Marey in 1863 [Lawrence, 1978]. The sphygmograph which proved to be cumbersome and inaccurate was operated by a spring-loaded lever pressing against the wrist compressing an artery. With each pulse the lever moved and the lever’s movements were traced on smoked paper [Howard Hughes Medical Institute, 1998]. The sphygmomanometer succeeded the sphygmograph, was devised by the Italian physician Scipione Riva-Rocci in about 1895, and was an early version of the modern blood pressure cuff [Crenner, 1998]. The early sphygmomanometer was more accurate than the sphygmograph but was still inaccurate by today’s standards. The early cuffs were quite narrow (≈2cm) and physicians used their fingers to detect arterial blood flow as they watched BP changes [Howard Hughes Medical Institute, 1998].

In 1905 Nikolai Korotkov, a Russian surgeon, developed the modern method of using a stethoscope to listen for the sound of blood flowing through the artery and this method proved to be extremely accurate [Howard Hughes Medical Institute, 1998]. The sounds reflecting the phases of a blood pressure pulse are named in honour of Korotkov.
One of the aims of the remaining literature review is to show that the weight of evidence indicates the reverse of Empedocles’ original contention: emotion is at the seat of the heart. That is, blood pressure, as prone as it is to many internal and external influences, can be subject to these sources of variation through the effect modifiers of attitude, annoyance and the ability to cope with events which are commonly regarded as ‘stressful’, in addition to and working through the established physiological mechanisms governing the heart.

1.2.2 Meaning and definitions of blood pressure and BP variability

1.2.2.1 Blood pressure regulation

As a vital sign or function, blood pressure reflects the health status of the circulatory system. The two primary measures of blood pressure are systolic (contracted) and diastolic (relaxed-expanded) blood pressures [Seeley et al, 1989](p.594). Two derived BP measures also are often used. One is the so-called pulse pressure, equal to systolic BP minus diastolic BP [Seeley et al, 1989](p.641). The other is the mean arterial pressure (MAP), approximately equal to (systolic BP + diastolic BP)/2 [Seeley et al, 1989](p.648), although alternative definitions of MAP exist. These blood pressure measures reflect the pressure changes occurring in the aorta rather than in the left ventricle of the heart [Seeley et al, 1989](pp.637-640). The MAP in the aorta is equal to cardiac output × peripheral resistance, where cardiac output is the total amount of blood pumped in a unit of time, usually a minute, and peripheral resistance is the total resistance against which the blood is pumped [Seeley et al, 1989](p.648).

Regulation of cardiac output

Blood pressure regulation occurs through hormonal and nervous-system regulation of cardiac output and through peripheral resistance. Cardiac output is governed by regulatory mechanisms classified as intrinsic and extrinsic. Intrinsic regulation occurs chiefly through venous return, in which blood flowing into the right atrium from the veins causes the ventricles to fill more when relaxed/expanded (diastole), which then stretches the ventricular muscle further than normal, in turn leading to a stronger muscular contraction (systole) and greater stroke volume (Starling’s Law) [Seeley et al, 1989](p.598). An increased volume of blood is pumped out with greater force. The heart rate also increases in response to increased venous return, by about 10-30%, through
stretching of the right atrium wall which in turn stretches the sinoatrial (SA) node leading to an increase in the rate of action potential generation through increased permeability of the SA cell membranes to calcium and sodium ions [Seeley *et al*, 1989](p.591). The heart rate is more sensitive to changes in venous return than to changes in arterial blood pressure, since the latter is isolated from ventricle pressure by the closed semi-lunar valves during diastole [Seeley *et al*, 1989](pp.598-600).

Extrinsic cardiac output is regulated by the sympathetic and parasympathetic nervous system and by hormones. Parasympathetic nervous control of the heart is via the vagus nerve synapsing with parasympathetic ganglia in the heart wall. Post-synaptically, these extend to the SA node, the atrioventricular (AV) node, coronary arteries and atrial myocardium [Seeley *et al*, 1989](p.600). Parasympathetic innervation has the primary effect of inhibiting the heart rate, with little impact on the stroke volume, through the production of post-synaptic acetylcholine. Acetylcholine has the effect of making cardiac cell membranes more permeable to potassium which lowers the resting membrane potential (ie, makes it more negative) which reduces the rate of depolarisation, resulting in a reduction in the heart rate. Cardiac output can be reduced by 20-30 beats per minute from parasympathetic stimulation [Seeley *et al*, 1989](p.600).

Sympathetic nervous control of the heart originates in the thoracic region of the spinal cord where emerging neurons synapse with post-ganglionic neurons of the cervical sympathetic chain ganglia. The post-synaptic neurons form the cardiac nerves which innervate the SA and AV nodes, coronary vessels and the atrial and ventricle myocardium. Sympathetic innervation stimulates the heart both through increased heart rate and force of muscular contraction. The heart rate can reach up to 250-300 beats per minute under sympathetic stimulation, but beyond a certain point the cardiac output does not increase because the ventricular diastole is not long enough to allow complete ventricular filling. The increase in force of contraction is of the order of 20% under sympathetic stimulation, through innervation of the ventricular myocardium [Seeley *et al*, 1989](pp.600-601).

Norepinephrine is the main post-ganglionic neurotransmitter involved in sympathetic cardiac stimulation. Norepinephrine is taken up by heart muscle cell surface beta adrenergic receptors leading to an accumulation of cyclic AMP (adenosine monophosphate) in the cytoplasm, which in turn increases the permeability of the cell
membrane to calcium and sodium ions. As a result, the rate and degree of cardiac muscle depolarisation is increased, leading to increased heart rate and greater force of contraction [Seeley et al, 1989](p.601). Cardiac output can be increased by 50%-100% through sympathetic stimulation. Other regulatory influences on the heart rate include blood pH, oxygen and carbon dioxide levels. These are detected by chemoreceptors located in the oblongata medulla itself (sensitive to pH and carbon dioxide) and in the carotid body (sensitive to oxygen). Decreases in pH or oxygen, or an increase in carbon dioxide, will result in sympathetic stimulation of the heart. Sympathetic stimulation is decreased when these levels act in the opposite direction.

The heart rate is also influenced by levels of potassium, sodium and calcium ions. An excess of extracellular potassium ions in the cardiac tissue results in a decreased heart rate through the partial depolarisation of the resting membrane potential leading to lowered amplitude and rate of muscle fibre action potentials. Increases in potassium levels also lead to decreased calcium entering the sarcoplasm of the cardiac muscle cells which decreases the strength of ventral or atrial contraction [Seeley et al, 1989]. Increases in extracellular calcium leads to increase in contraction force, but increased plasma levels of calcium also leads to a lower heart rate because of calcium’s role in reducing the frequency of action potentials in nerve fibres. Lowered blood calcium levels lead to an increase in heart rate, but the levels must be quite low (= 10% of normal) for the effect to be detected, by which time the organism has died from tetany in the skeletal muscles. Extracellular levels of sodium rarely deviate far enough from their normal values to affect cardiac output [Seeley et al, 1989](p.603).

**Hormonal blood pressure regulation**

Hormonal regulation of cardiac output and blood pressure occurs chiefly through 4 pathways: (i) the adrenal medullary mechanism; (ii) the renin-angiotensin-aldosterone mechanism; (iii) the vasopressin mechanism; and (iv) the natriuretic mechanism [Seeley et al, 1989](pp.650-651).

The adrenal medullary mechanism works as a consequence of increased sympathetic stimulation of the heart and blood vessels which also causes increased stimulation of the adrenal medulla resulting in increased secretion of epinephrine and norepinephrine. Both epinephrine and norepinephrine act to increase cardiac output by stimulating the cardiac
muscle, as does norepinephrine when the latter acts as a sympathetic neurotransmitter. As hormones, epinephrine and norepinephrine are delivered to the cardiac muscle via the blood. The effects of epinephrine on cardiac output are slower than sympathetic stimulation, but more prolonged [Seeley et al, 1989](p.601).

In the renin-angiotensin-aldosterone mechanism the kidney releases the renin enzyme into the circulatory system via specialised structures called the juxtaglomerular apparatuses. Renin secretion is stimulated by reduced blood pressure, elevated plasma concentration of potassium and reduced plasma concentration of sodium ions, although the effect of the latter is not as strong as the stimulus of BP reduction or potassium ion elevation. Renin acts on a substrate of plasma proteins called angiotensinogen to produce angiotensin I which in turn is acted upon by enzymes situated mainly in the small blood vessels of the lung to produce angiotensin II (referred to as active angiotensin). Angiotensin II causes vasoconstriction in arterioles and to a lesser extent in veins, causing increased BP from peripheral resistance and from venous return to the heart. Angiotensin II also stimulates the release of aldosterone from the adrenal cortex which reduces urine production, in turn decreasing blood volume loss acting to conserve blood pressure. The renin-angiotensin-aldosterone mechanism acts more slowly on blood pressure than sympathetic nervous stimulation or the adrenal medullary mechanism, and is longer lasting. Renin remains active for about an hour after its release into the bloodstream [Seeley et al, 1989](pp.650-651).

Renal blood flow regulation is not confined to the renin-angiotensin-aldosterone mechanism, however. At least one animal study has shown variations in renal blood flow to occur directly via renal sympathetic nerve activity (RSNA) [Malpas et al, 1998]. In the Malpas et al study, variously stimulated RSNA in rabbits, via 3 different stressors of noise, air jet and hypoxia (10% \(O_2\)), were accompanied by corresponding changes in levels of renal blood flow. Mean levels of RSNA were increased by the 3 stimuli, by 12%, 31% and 14% respectively, which were “mirrored” by mean decreases in renal blood flow of 8%, 10% and 8% respectively. Changes in renal blood flow were also accompanied by changes in plasma renin activity. The authors also found that the hypoxia stimulus caused efferent stimuli to increase in amplitude only (more nerve fibres involved), while the noise and air jet stimuli produced increases in both amplitude and frequency of nervous discharges associated with RSNA. The RSNA mechanism was confirmed in a subsample of denervated rabbits where renal blood flow was not altered.
when exposed to the stimuli.

The vasopressin mechanism is initiated from increased nervous stimulus of the posterior pituitary gland by the hypothalamus, either as a result of decreased blood pressure signalled from baroreceptors (‘stretch’ receptors) located in the aortic arch and carotid sinus, or directly and more sensitively from increased plasma concentrations of solutes. The stimulated posterior lobe of the pituitary gland in turn secretes vasopressin or antidiuretic hormone (ADH) which acts directly on blood vessels as a vasoconstrictor and on the kidney to reduce urine production [Seeley et al, 1989](p.652).

The atrial natriuretic mechanism originates from the release of atrial natriuretic factor from cells located in the atria of the heart and is stimulated by elevated atrial blood pressure. The atrial natriuretic factor in turn acts on the kidney to increase urine production to lower blood volume and therefore blood pressure. The atrial natriuretic factor is also thought to play a role in the suppression of ADH production [Seeley et al, 1989](p.652).

Two additional mechanisms, the fluid shift mechanism and the stress-relaxation response, also operate to regulate blood pressure under normal conditions. The fluid shift mechanism operates when changes in pressure across capillary walls occur. A rapid increase in blood pressure will force some fluid from blood vessels into the interstitial spaces, preventing extreme blood pressures being reached. Conversely, a rapid decrease in blood pressure will cause interstitial fluid to pass into the capillaries. Since the interstitial fluid forms a large reservoir, the fluid shift mechanism is a powerful if passive regulator of blood pressure [Seeley et al, 1989](p.652).

The stress-relaxation response is a consequence of the way smooth muscle cells operate inside blood vessel walls. Within a few minutes to an hour following a sudden reduction in blood pressure, the consequent reduction in pressure on smooth muscle cells lining blood vessels will cause the muscle cells to contract which reduces the volume of the blood vessels in turn preventing a further decline in blood pressure.

**Regulation of peripheral resistance**

The rate of fluid flow along a length of tubing is proportional to the difference in pressure
at each end of the tube’s length. In general,

\[ \text{Flow} = \frac{(P_1 - P_2)}{R}, \]

where \( P_1 \) and \( P_2 \) = pressures at two given points along the tube,
and \( R \) = resistance

Blood flow is governed by Poiseuille’s law which takes account of the internal radius of the blood vessel and blood viscosity, and characterises resistance as proportional to the length of the vessel:

\[ \text{Flow} = \frac{(P_1 - P_2)}{(8\nu L/r^4) \equiv (P_1 - P_2) r^4 / 8\nu L} \]

where \( P_1 \) & \( P_2 \) are the pressures at each end of the length of the vessel \( L \), with
\( \nu \) = blood viscosity, and
\( r \) = radius of the blood vessel

As evident from the above formula, blood flow is reduced by higher blood viscosity and greater vessel length, but is much more sensitive to the radius of the blood vessel (since the latter acts on blood flow in proportion to the fourth power of the vessel radius). For example, holding everything else constant and halving the radius of the blood vessel results in 1/16th of the original blood flow.

Nevertheless, blood vessels are not solid inflexible pipes -- the volume of a blood vessel normally increases (passively) in response to increased blood pressure, referred to as compliance. Vascular compliance is expressed as the ratio,

\[ \text{Vascular Compliance} = \frac{\text{Volume increase (ml)}}{\text{BP increase (mmHg)}} \]

For a given increase in blood vessel volume, the smaller the subsequent increase in BP the greater the vascular compliance. Venous compliance is about 24 times that of arteries, so the veins act as a blood storage system, such that approximately 64% of blood volume at any one time resides in the venous system, 15% in the arteries, 9% in the pulmonary vessels, 7% in the heart and 5% in the capillaries [Seeley et al, 1989](pp.638-639).

Regulation of peripheral circulation/resistance occurs through activation of pre-capillary sphincters and metarterioles by metabolic demand; by the sympathetic nervous system
via vasodilator and vasoconstrictor nerves originating in the vasomotor centre in the medulla oblongata; and by hormones or other vasodilator substances such as $CO_2$, lactic acid, potassium, and hydrogen ions, and/or in response to a lack of oxygen or other nutrients.

Minute-by-minute peripheral circulatory system regulation of the heart begins from sensory receptors called baroreceptors (or “stretch receptors”) in the walls of the large vessels such as the aortic arch and carotid sinus. Signals from these neurons are carried by afferent neurons in the glossopharyngeal and vagus nerves to the cardioregulatory centre situated in the medulla oblongata. Efferent signals are transmitted back to the heart via the sympathetic and parasympathetic neurons in the vagus nerve, the sympathetic cardiac nerve, and to the adrenal medulla (for regulation of catecholamine release into the blood stream for uptake by the heart muscle) [Seeley et al, 1989](pp.601-602).

As blood pressure in the aorta and internal carotid arteries increases, the walls of these vessels stretch, which leads to an increase in the frequency of the afferent impulse. Increased stimulation of the baroreceptors results in decreased sympathetic and increased parasympathetic stimulation, leading to a decrease in the heart rate. This negative feedback loop is the main mechanism for maintaining homeostasis, keeping blood pressure within narrow bounds [Seeley et al, 1989](pp.601-602). A positive feedback loop also operates -- the so-called Bainbridge reflex. As the right atrium wall stretches with increased venous input, the stretch receptors in the right atrium send afferent signals via the vagus nerve to the cardioregulatory centre which acts to increase sympathetic stimulation leading to an increase in the heart rate. The Bainbridge reflex operates in conjunction with the intrinsic regulation mechanism expressed by Starling’s Law (see above) [Seeley et al, 1989](p.602).

Baroreceptors do not change mean BP on a long term basis, but instead adapt to a new mean BP level within 1-3 days. If BP is elevated for a few days, the baroreceptors will adapt to the new level and not reduce BP to its original value [Seeley et al, 1989](p.650). Baroreceptor adaptation is common in hypertensives and represents one possible mechanism for acute BP effects from exposure to BP-raising agents becoming permanent (chronic). Baroreceptor activity may also be of relevance to ethnic-racial differences in BP. For instance, Modesti et al [1999] showed significant differences in the baroreceptor
reflex between recent normotensive migrants from Mogadishu to the US and age- and sex-matched US whites in an experimental study of cardiovascular responses to changes in dietary salt intake. Baroreceptor changes may also play a part in long term, permanent BP increases from exposure to continuous BP-altering stimuli lasting for days.

Longer-term BP regulation, from day-to-day, week-to-week, year-to-year, centres on the renal response to BP variations via the renin-angiotensin-aldosterone system, and on production of the atrial natriuretic factor. Urine production in the kidney varies directly with BP to regulate blood volume. At the same time as the occurrence of a BP increase renin secretion decreases which in turn reduces the production of angiotensin II from the angiotensin substrate. This leads to vasodilation and increased filtering of blood by the kidney and to increased urine production. Aldosterone production is also decreased by reduced renin secretion which in turn leads to increased secretion of water and salt from the kidney. Increased secretion of natriuretic factor from the atrial wall in response to increased BP in the atria of the heart also leads to increased urine production in the kidneys [Seeley et al., 1989](pp.653-654).

### 1.2.2.2 Haemodynamicity/BP variability

Haemodynamicity, or within-subject blood pressure variability, is defined by Weder and Julius as ‘the deviation of measured blood pressures from a baseline or reference value, such as the mean of a series of blood pressure measures’ [Weder et al., 1987]. Factors associated with individual blood pressure variability can be classified into three broad areas. The first is related to nervous system control of stimuli to the heart muscle and blood vessels. The second to the frequency and intensity of external stimuli, and how these are processed by the individual. The third (possibly) as an expression of a transition from normotension to hypertension, since a number of studies have linked elevated blood pressure variability with hypertension [Weder et al., 1987].

Individual BP variations can be due to many factors including seasonal and diurnal cycles, changes in ambient temperature, pharmacological factors, body mass index, smoking status, diet, physical fitness, sub-clinical bacterial, viral or immunologic disease, and predisposing factors including sensitivity to salt and a family history of hypertension. Blood pressure in humans, like Hales’ horse, varies continuously minute-by-minute. The range of temporal variability in human blood pressure appears to be constant when
regarded as a proportional function of the individual’s diastolic blood pressure. That is, with all other factors held equal, the variability of blood pressure is similar across different individuals when this variability is measured as a proportion of each individual’s mean diastolic blood pressure. For example, a proportionate measure of BP variability, the coefficient of variation of a sequence of mean arterial pressure (MAP) measurements, has been shown by Mancia et al to be not significantly different between adult hypertensives and normotensives [Mancia et al, 1980], but the standard deviation of the MAP measures was greater in the hypertensives than the normotensives. The Mancia et al study in fact found that the proportional measure of BP variability, the coefficient of variation, was slightly lower, but not significantly, in the hypertensives than the normotensives. Accordingly, absolute measures of within-subject BP variation, such as the standard deviation of successive MAPs, appear to be more strongly correlated with BP than proportional.

BP variability has also been found to be associated with elevated resting BP and has been posited as a marker for possible future hypertension. In the area of nervous system control of the heart and blood vessels, sympathetic overactivity in particular has been regarded as the main mechanism for BP variability being associated with hypertension, particularly incipient hypertension [Julius, 1996].

Whether BP variability predicts future elevated blood pressure is not certain, as a high proportion of hypertensives may have high BP variability but a low proportion of those with high BP variability may be hypertensive. And in children it is not certain that BP variability is a characteristic of hypertensives. For instance, a study of hypertension and blood pressure variability in a small sample of 11 children aged under 18 years with essential hypertension, by Uhari et al [Uhari et al, 1982], found elevated peripheral resistance in 4 of the children only, elevated cardiac indices in 2 only, and elevated stroke volume in 3 subjects only. The authors concluded that hypertension was not always preceded by increases in cardiac output and that total peripheral resistance can be present already at the onset of hypertension. In other words, sympathetic overactivity did not seem to be associated with child hypertension.

An examination of peripheral resistance in children of hypertensive and normotensive parents, with specific focus on the regulatory role of the kidney in the angiotensin-renin-aldosterone pathway, found renal vascular resistance to be higher in the children of
hypertensive parents than in children from normotensive parents [van Hooft et al, 1991]. The authors concluded that “Renal vasoconstriction is increased and renin and aldosterone secretion is decreased in young persons at risk for hypertension (viz, children with hypertensive parents). These findings support the hypothesis that alterations in renal hemodynamics occur at an early stage in the development of familial hypertension” [van Hooft et al, 1991].

Further, increased blood pressure lability or cardiovascular reactivity in response to long-term exposure to intermittent stressors has not been shown to be a precursor to sustained hypertension [Horan et al, 1990; Julius et al, 1971]. Peaks of blood pressure, for example, have been found to be less significant for predicting hypertension than the average 24-hour blood pressure level [Pickering, 1978].

In their review article of behaviour and blood pressure variability, Weder et al [Weder et al, 1987] identify the following areas of agreement in the literature: (i) blood pressure variability is positively correlated with age (see also [Mancia et al, 1980]) and is not significantly different between hypertensives or normotensives. (ii) experimentally induced increases in blood pressure variability have not been found to cause hypertension. (iii) hyper reactivity of blood pressure in young borderline hypertensives and in normotensives with a family history of hypertension has been shown to occur almost exclusively in response to specific mental/cognitive tasks. Such hyper reactivity has not been shown to be an independent precursor or predictor of future hypertension. (iv) psychological and behavioural traits such as hostility and aggressiveness are associated with BP variability and with hypertension. (v) no reliable marker (biochemical, physiological or behavioural) exists for ‘prehypertension’.

Areas of disagreement centre on (i) increased short-term blood pressure variability, induced in the laboratory, predicts/does not predict future hypertension. (ii) repeated pressor episodes (episodes which cause blood pressure to rise) do/do not lead to hypertension. (iii) increased blood pressure variability is/is not a predictor of cardiovascular damage. (iv) psychological and behavioural traits cause/do not cause hypertension or BP variability. (v) genetic factors are/are not the most important predictors of future hypertension.

If evidence is weak for stress manifesting as permanent physiological change through
cardiovascular hyper reactivity, the case for alternative mechanisms is weak also. Studies have yet to establish sustained hypertension to result from stress, regardless of differing response modes to stressors, and despite ample laboratory and naturalistic evidence of transitory blood pressure effects induced by various stressors, including noise [Lipscomb et al., 1976; Gunn, 1978; Andren et al., 1980]. However, there are few if any studies which have examined BP, hypertension or cardiovascular disease outcomes when stimulus-induced blood pressure elevations (under experimental or naturalistic conditions) are not permitted to return to normal levels before the next stimulus was initiated. If Pickering’s finding that average 24-hour blood pressure levels are of more relevance than peak blood pressures, then it would appear reasonable to infer that a permanent effect could result from extended exposure to intermittent stressors whose frequency of occurrence is sufficiently high to not allow a return to normal resting blood pressure levels.

Extensive laboratory evidence points to the likely underlying haemodynamic regulation mechanisms under noise stimulus. For instance, a laboratory study by Andren et al. [Andren et al., 1979 & 1980] sheds some light on the possible mechanisms of haemodynamic regulation of BP under noise stimulus. In this study, 18 healthy experimental subjects were exposed to industrial noise levels of 75, 85, and 95 dB(A), and BP, heart rate, stroke volume and cardiac output measured non-invasively. Mean diastolic BP was found to increase significantly by 12% under noise stimulus, as did peripheral resistance by a similar amount. At the same time, stroke volume and cardiac output decreased by 7% and 5% respectively, while mean systolic BP and mean heart rate did not change significantly. Moreover, no significant changes in plasma adrenaline or noradrenaline occurred during the noise exposure. The BP effects persisted for 5 minutes after cessation of the stimulus and were absent 10 minutes after noise cessation. These and findings from other laboratory studies also suggest that under noise stimulus the mechanism for acute BP increase is through vasoconstriction rather than sympathetic stimulus of the heart.

1.2.2.3 A candidate mechanism from human studies for noise stimulus to affect resting blood pressure

From the outlines of blood pressure and cardiac regulation above, it is possible to hypothesise putative blood pressure mechanisms which may be responsive to stimulus from noise, from short (acute) and longer term (chronic) perspectives. Clearly a directly
physiological reaction to loud impulsive noise such as an explosion occurs as the startle reflex, where no cognition is involved and all organisms with hearing react similarly. From the above, there is also at least one plausible candidate mechanism for an acute BP effect to manifest into elevated resting BP. This is through the property of baroreceptors habituating to new elevated blood pressure levels where these have occurred over a period of about 3 days. From a noise exposure point of view, the noise stimulus would need to be loud enough and sufficiently frequent or constant over a period of days for it to prevent BP returning to normal resting levels and consequently lead to a permanent raising of blood pressure. The re-setting of baroreceptors may be consequent to increased sympathetic stimulation of cardiac output or peripheral vasoconstriction which in turn may be associated with the stress response to a continuous external stressor. Another putative mechanism also exists for noise being associated with elevated resting BP and this will be outlined when the literature on laboratory animal studies is examined.

**1.2.2.4 Issues of measurement of blood pressure**

Issues of blood pressure measurement, generally and in relation to noise exposure, include inter-observer BP variation; observer-subject BP variation (eg, so-called ‘white coat hypertension’); the degree of compatibility of BP readings taken by different BP measurement techniques and instruments; whether 4th or 5th phase Korotkov sounds are used for automated or non-automated determination of diastolic BP; and the degree of machine and human (intra-observer) reliability of BP measurements.

Inter-observer variation has been shown to affect non-automated auscultatory BP readings [Osborne et al, 1983; Burke et al, 1987], and to a lesser extent automated oscillatory BP measurements [Fortmann et al, 1981; Bruce et al, 1988]. ‘Whitecoat hypertension’ (see below) is probably the most commonly known source of inter-observer variation in BP measurement. In non-automated BP measurement, the main sources of inter-observer variability include differences in the point at which observers perceive 4th phase pulse muffling or 5th phase blood flow noise disappearance; digit preference, where a BP reading is rounded up or down to the nearest 5 or 10 mmHg, countered by the use of random-zero sphygmomanometers [Rose et al, 1982]; and how different observers interact with measured subjects [Rose et al, 1982].

In relation to noise and BP, the main measurement issue in naturalistic settings is of
separating possible associations between transitory noise exposure events and BP readings, if these associations exist, so that measured resting BPs can be related validly to long-term mean noise exposure levels.

**4th and 5th Korotkov phase blood pressure measurement**

The 4th phase Korotkov sound occurs with the muffling of the turbulent pulsing sound of the blood flow resulting from easing the complete constriction of the brachial artery by the sphygmomanometer cuff. The 4th Korotkov phase muffling occurs when the pulsing blood flow in the partly constricted brachial artery is in transition to continuous laminar (non-turbulent) blood flow. The disappearance of blood flow sounds, the 5th Korotkov phase, signals the complete return to laminar blood flow [Seeley et al, 1989](p.638). The diastolic BP is taken either as the 4th or 5th phase reading.

Unreliable determination of diastolic BP is minimised in automated BP measurement (oscillometric), or in non-automated auscultation, by using the 5th Korotkov phase (K5) when the sound of blood flow disappears completely, rather than when the pulse is perceived to muffle (4th phase). Fourth phase readings are used more in auscultatory BP measurement by conventional sphygmomanometry.

In children, the difference between the 4th (K4) and K5 BP measurements appears to be associated with a number of factors. For example, a study by Sinaiko et al [Sinaiko et al, 1990] of 19,274 children aged 10-15 years, found no difference between 4th and 5th Korotkov phases in 50% of the children; 1-4 mmHg difference in 15%, 5-10 mmHg in 20%, 11-20 mmHg in 11%, and ≥21 mmHg in the remaining 3 percent. The authors found that the 4th-5th phase difference on average to be greater in boys than girls and in older than younger children. The difference was positively related to height, systolic BP and fourth phase diastolic BP, and negatively related to body mass index and 5th Korotkov phase diastolic BP. Clearly, the validity of comparisons of BP measurements taken at different Korotkov phases can depend on the subjects being measured, but also on the observer.

Differences in inter-observer variation between K4 and K5 measurements has also been found. Elkasabany et al, from the Bogalusa cohort [Elkasabany et al, 1998], found inter-observer variability to be greater with K5 than K4 child BP measurements and found also that the variance in both decreased with age of the child.
Uhari et al [Uhari et al, 1991], in their follow-up BP studies of a cohort of 2,500-3,000 Finnish children aged 6-18 years, found K4 to be consistently absent in 3-6% of respondents and K5 to be absent in 0.2-0.6% of the sample. They concluded that BP measurements were most reliable and repeatable using K5 diastolic measures.

**Observer-subject variation**

With automated BP measurement, Bruce et al [Bruce et al, 1990] compared observer differences in BP measurements made with a Dinamap oscillometric blood pressure recorder on a total of 2,596 male and female subjects aged 25-59 years from nine British towns. After adjusting for age, body mass index and town, significant differences between the two observers were found for systolic pressure, 3.07 mmHg (p = 0.001) for male subjects and 2.08 mmHg (p = 0.036) for female subjects. Observer differences for mean arterial pressure and diastolic pressure were found to be less than 1 mmHg and were not statistically significant. The magnitude of observer differences also varied between towns, and when tested statistically, was significant in male subjects for systolic (p = 0.011), mean arterial (p = 0.009) and diastolic pressures (p = 0.002), but in female subjects was significantly different only for diastolic pressure (p = 0.023). It can be concluded from these studies that although the automated Dinamap kept observer variation within acceptably low limits in field study conditions, the problem was not altogether eliminated. Accordingly, it would still be necessary to adjust for inter-observer variability in analyses when automated BP machines are used.

**Reliability of automated BP measurement versus random-zero sphygmomanometry**

The method developed by Bland and Altman [Bland et al, 1986] has been the most widely used approach to assessing repeatability and agreement in measurement by different instruments. Bland and Altman advance strong arguments for why Pearson product-moment correlation should not be used for assessing the level of agreement between any two tests. This is because high correlation does not mean high agreement (since any two measures may correlate very strongly (eg, vary in unison) but not agree at all). This is the difference between concurrent and criterion validity where the value of one measure can be predicted from the other without transformation. The Bland-Altman technique relies on plotting, at each measurement occasion, the difference between the two measurement methods against each inter-method mean. For normally distributed measurement differences, the ‘limits of agreement’ are defined as 2 standard
errors above and below the overall mean difference of the two measurement methods, ie, the range within which over 95% of readings occur. The standard error of the mean difference is estimated in the usual way as $\sqrt{s^2/n}$, and that of the upper and lower ‘limits of agreement’, defined as $\hat{d} \pm 2s$, as approximately $\sqrt{3s^2/n}$. Repeat measures of a single instrument, to determine its ‘limits of reliability’, are dealt with similarly, except the standard error of the mean repeat measure difference is calculated differently.

Goonasekera et al [Goonasekera et al, 1995] compared systolic and 5th phase diastolic BPs measured by a random zero sphygmomanometer with those from an automated oscillometric BP monitor, the Critikon Dinamap 8100. The authors determined and compared repeatability coefficients of both for systolic and diastolic BP measurements. These were 8.64 and 7.04 respectively for the random zero sphygmomanometer, and 9.72 and 6.62 for the Critikon 8100. The automatic BP monitor differed in its systolic BP by 6.45 mm Hg and diastolic BP by -10.77 mm Hg compared with the random zero sphygmomanometer. The limits of agreement, as estimated by the Bland-Altman method above, were wide enough for a normotensive to be missclassified “as a hypertensive on machine error alone”.

While BP measurements may differ significantly between auscultatory and oscillatory methods, within automated oscillatory BP measurement there is also wide variation by machine. For example, Kaufmann et al [Kaufmann et al, 1996] compared 3 different brands of automated oscillatory BP monitors, with agreements between any two instruments judged according to guidelines of the American Association for Advancement of Medical Instrumentation (AAMI). While the authors found that repeat BP measurements from one of the machines to be reliable, they also found that BP results from the different monitor brands were not interchangeable. The standard deviation of the BP difference between two Dinamap monitors was 7 mm Hg for systolic and 6.3 mm Hg for diastolic blood pressure. The mean BP differences were 0.9 mmHg systolic and 0.2 mmHg diastolic (not statistically significant), thus showing reasonable reliability within the one brand of automated BP machine. Standard deviations and means of BP difference between different brand machines were: SpaceLabs versus Dinamap, 9.1 mmHg for systolic and 8.3 mmHg for diastolic BP with mean differences of 1.6 mmHg ($p = 0.026$) and 7.3mmHg ($p = 0.0001$), systolic and diastolic respectively. Corresponding standard deviations of the BP differences for Marqui versus Dinamap were 11.8 mmHg systolic and 9.7 mmHg diastolic, with corresponding mean BP
differences of 0.8 mmHg systolic (not statistically significant) and 0.3 mmHg diastolic (not statistically significant).

The authors found Dinamap versus Dinamap SBP differences exceeded 10 mmHg in 10% of observations, while the 10 mmHg threshold was exceeded in 31% and 32% of observations for Space Labs versus Dinamap and Marqui versus Dinamap, respectively. The authors concluded, “In view of the variability that exceeds the AAMI guidelines and the one out of three occurrence of individual SBP differences exceeding 10 mm Hg for comparisons of SpaceLabs or Marqui versus Dinamap, measurements by these three oscillometric devices are not interchangeable”.

The above indicates that while automatic oscillometric BP monitor and manual sphygmomanometric BP measurements clearly cannot be compared or used interchangeably in clinical practice, neither can oscillatory BP readings be compared interchangeably if gained from different brands of automated BP monitor. Repeatability coefficients, which ideally should be zero, generally are too large for either type of instrument to be considered as the gold standard for non-invasive BP measurement, although those of automatic oscillometric BP monitors are generally superior to those of random-zero sphygmomanometers.

*How suitable is BP measurement data for pooling in meta-analysis?*

The measurement reliability and validity studies quoted above recommend that BP measurements derived from different BP measurement techniques not be treated as equivalent. In particular, these centre on the non-equivalence of BP results from manual sphygmomanometry versus random-zero sphygmomanometry versus automated oscillatory BP measurement; and whether 4th versus 5th Korotkov phases are used to determine diastolic BP. Clearly, the implication is that for meta-analytic studies of particular factors associated with BP, stratification by the type of BP measurement would be necessary, in conjunction with conversion formulae [Weaver et al, 1990], to render differently measured BP data compatible.

Nuutinen et al [Nuutinen et al, 1996] attempted to pool BP measurements from 30 BP studies of children aged 11-15 years. Due to the heterogeneity of the BP data regarding the method of measurement and whether 4th or 5th Korotkov phase was used, the
authors concluded that, “BP results obtained in different investigations using different BP devices and Korotkoffs [sic] phases as indicator of diastolic BP should not be pooled. More widely accepted recommendations are needed to standardize the measurement of BP in children.”

As there are no BP conversion factors universally available or accepted, an adequate meta-analytic literature review of BP would require development of methodological and statistical analytic tools beyond the scope of this thesis. This issue is compounded further when relating BP findings and noise exposure since there are many noise exposure metrics which do not convert readily to a common unit. Accordingly, an adequate systematic review of BP and noise would require simultaneous stratification by BP measurement mode and by noise metric, then findings from the separate strata pooled on the basis of valid conversion factors between the different aircraft noise exposure metrics and BP measures.

“Regression dilution bias”

The literature on determinants of BP and risk factors for hypertension is extensive. A key issue on factors found to be associated with BP is the effect of variability in these on regression or risk models of BP. In linear, logistic, Poisson or proportional hazards regression models, it is assumed that variation or error in the covariates (ie, the factors under study in the regression model) is zero. This may be true of sex or other factors which do not change with time, such as a family history of high blood pressure. However, when natural variation in other physical measures has not been taken into account, the resulting estimates are biased toward the null in the case of univariate modelling, and can be biased in either direction in the case of multivariate modelling. This bias is referred to as ‘regression dilution bias’, and was first raised as an issue in the medical literature (although not by that name) by Gardner et al [Gardner et al, 1973].

Much of the subsequent literature addressing regression dilution bias has been in reference to blood pressure, either as an outcome or as a predictor of other health outcomes. MacMahon et al [MacMahon et al, 1990] adjusted for regression dilution bias in relation to BP’s variability as a predictor of stroke and coronary heart disease events. Chen et al [Chen et al, 1991] adjusted for regression dilution bias in relation to serum cholesterol variability as a predictor of death from coronary heart disease. Law et al

The main approach to adjusting for regression dilution bias in longitudinal studies centres on re-measuring the risk factor or predictor variable in a study sub-sample at the midpoint of a given follow-up interval and relating these values to the baseline measure to produce a “regression dilution ratio” which is used to correct the regression estimate for baseline values of the study factor [Clarke et al, 1999; MacMahon et al, 1990].

Parametric and non-parametric methods can be used in estimation of the regression dilution ratio [Clarke et al, 1999]. The non-parametric approach assembles pairs of individual baseline and mid-period observations into several groups ordered according to the initial baseline values of the study factor. The range of these initial values, $r_i$, is defined as the difference between the highest group mean baseline value and the lowest group mean baseline value. The corresponding range of midpoint values, $r_u$, is then the difference between the highest mean midpoint value and the lowest group mean midpoint value, as found in these groups. In general, the range of the midpoint means, $r_u$, is less than the range of the baseline means, $r_i$ and the regression dilution ratio is defined as $R = r_u/r_i$. The adjusted regression coefficient for the study factor is then $1/R$ times the “uncorrected” regression coefficient. According to Clarke et al [Clarke et al, 1999], $R$ is “approximately independent of the number of groups chosen” and is an “assumption-free estimate of the importance of regression dilution during the exposure period”. A parametric approach can be used if the variances of the study factor at baseline and midpoint are approximately equal, and the autocorrelation coefficient between baseline and midpoint measures is approximately equal to $R$ [MacMahon et al, 1990], so is limited by the requirement for constant variances.

Regression dilution bias has been found to increase with period. In a comparison of BP and cholesterol measurements from the Framingham and Whitehall cohorts Clarke et al [Clarke et al, 1999] found the ratio of mid-period follow-up to baseline ranges of systolic BP in the Framingham cohort to decline from 0.68 at the first decade midpoint to 0.52 at the second decade midpoint to 0.34 at the third decade midpoint. In the
Whitehall cohort the corresponding ratio at the third decade midpoint was 0.32. Similar findings emerged for diastolic BP and cholesterol levels.

The implicit assumption behind these approaches to correcting for regression dilution bias is that the value of the risk factor measured at the midpoint of the follow-up period is its “usual”, unbiased level for the whole follow-up period (hence the use of the subscript ‘u’ above), and that the baseline value is a biased estimate of the level of the study factor for the period.

In univariate analyses regression dilution bias causes estimates of an association to be biased toward the null [MacMahon et al., 1990]. In multivariate analysis regression dilution bias may affect an association toward or away from the null [MacMahon et al., 1990]. This occurs essentially because as the effects of regression dilution bias are adjusted for in all predictor variables in a model the resulting influence of each on the outcome and on each other will be different.

The increase in regression dilution with follow-up period can be used to establish an upper bound for regression dilution bias where data for the midpoints of follow-up periods are not available. For instance, a cohort study may have only a baseline and single follow-up measurement of a study factor for BP. In such a case the upper bound for regression dilution bias can be estimated by taking the follow-up measurement as the midpoint of a hypothetical period twice the size of the actual follow-up period. The estimate for R would be an overestimate of its true value for the actual follow-up period but would nonetheless be indicative, as an upper bound, of the extent of regression dilution bias in the predictor variable. The extent of regression dilution bias with respect to noise exposure measures will be assessed in the methods section.

“White-coat hypertension” and “White-coat normotension”

A common phenomenon, especially in child blood pressure measurement, is so-called ‘white-coat hypertension’, first documented by Ayman et al. [Ayman et al., 1940]. White coat hypertension (WCH) refers to artificially elevated blood pressure readings in the clinic compared to the subject’s ambulatory or non-clinic readings [Pickering et al., 1990]. WCH has been shown to be more pronounced in subjects measured by physicians rather than by technicians or nurses, and more likely to occur in those who are younger,
female and weigh less [Pickering et al, 1988]. WCH is thought to result from subject anxiety and stress associated with the blood pressure measurement procedure, and has been shown to occur in children. One comparative study of child (5-15 yr, n=159) ambulatory and clinic BP measurements showed WCH to be responsible for 44% of children classified as hypertensive under clinic BP measurements (defined as 95th percentile systolic BP) who were normotensive under ambulatory measurement [Hornsby et al, 1991]. Hornsby et al also noted that WCH children had significantly different ambulatory BP patterns to both normotensives and hypertensives, but WCH has not been shown to correlate with target organ damage [Pickering, 1996].

In terms of taking BP measurements in quick succession WCH effects may manifest as higher readings across all the measurements, or in a slower decline in BP with successive readings as the subject becomes more accustomed to the procedure, but less so than those not prone to WCH. Similar reactions to WCH may occur due to subjective observer-subject interactions.

1.3 Determinants of Child Resting Blood Pressure

1.3.1 Activity and Fitness Levels

Levels of child physical activity and fitness have been shown to be negatively correlated with child blood pressure. In the Australian Schools Health and Fitness Survey, for example, Dwyer et al [Dwyer et al, 1994] found a statistically significant negative correlation between systolic BP and fitness level which was only partly accounted for, in multi-variate models of systolic BP, by child body fatness. The measure of fitness was defined as the ratio of physical work capacity at a heart rate of 170 beats per minute to the estimated lean body mass [Dwyer et al, 1994]. The authors concluded that the negative association found between higher systolic BP in children and fitness level was only weakly linked to plasma lipids and lipoproteins as possible intermediary factors along the causal pathway between fitness level and BP. That is, when skinfold thickness and lean body mass, themselves significant positive predictors of BP, were accounted for, fitness level was found to be a significant (negative) predictor of systolic BP in its own right.

Findings on child activity and fitness levels and BP have not been consistent, however [Dwyer et al, 1994]. Dwyer et al’s findings applied to systolic BP and not diastolic BP.
Harshfield et al also found less fit boys and girls to have higher systolic BPs in most group comparisons than fit counterparts, but also found higher diastolic BP in less fit female African American subjects [Harshfield et al, 1990].

1.3.2 DIET AND EATING HABITS

Dietary surveys are subject to substantial measurement error, such that most of the work on ‘regression dilution bias’ in BP determinants has centred on relating BP to inter-subject variability in dietary intake, after taking into account intra-subject variability in dietary measures such as salt intake and serum cholesterol levels. Findings from child BP and diet studies have been more equivocal, or even negative, compared to adult studies. [Whitten et al, 1980; Trevisan et al, 1981; Howe et al, 1986].

In Australia, Jenner et al found a significant negative association between diastolic BP and energy intake in boys, after adjustment for age, weight, height, socio-economic status and month of examination (a proxy for ambient temperature) [Jenner et al, 1988]. After adjustment for calorie intake (as a control for differences in body size, metabolic efficiency and physical activity which in turn determine the caloric intake of individuals in energy balance) and the above confounders, the effect size of the association between systolic BP and cholesterol intake in girls was large and statistically significant, with a mean increase of 12.9 mmHg for each unit of mean intake of cholesterol. Diastolic BP in boys was also found to be negatively associated with calorie-adjusted fibre intake. Systolic BP in girls was negatively related to calorie-adjusted intakes of protein and cholesterol. No detectable relationships were found between BP and calorie-adjusted intakes of fats, carbohydrates, sodium, potassium, calcium or magnesium. (n = 884, m = 434, f = 450; age = 9-10 yrs).

1.3.3 AMBIENT TEMPERATURE

Australian and overseas child blood pressure studies have found ambient temperature to be significantly and negatively associated with blood pressure [Jenner et al, 1987; Chifamba et al, 1998]. Jenner et al found the negative effect in children aged 9 years, with BP decreasing by 5-7 mmHg per 10°C rise in temperature. Chifamba et al found BP to increase by 32.2 mmHg systolic and by 19.5 mmHg diastolic per 10°C decrease in ambient temperature (from 25°C to 15°C) in African adults.
1.3.4 CHILDHOOD CONDITIONING FACTORS

1.3.4.1 Age

As children grow their blood pressure increases on average by about 1.5 mmHg systolic and 0.7 mmHg diastolic with each year of age until 18 to 20 years [Cresanta et al, 1986]. Thereafter the rate of increase in blood pressure reaches a plateau until late middle age whereupon the increase steepens again. Most of the rise in blood pressure with age in children is associated with increases in height and weight. When children of similar height and weight are grouped together the correlation with age disappears [Cresanta et al, 1986].

1.3.4.2 Sex

In males there are approximately 5.2 million erythrocytes (red blood cells) per cubic millimetre of blood (range: 4.2 to 5.8 million), and 4.5 million erythrocytes per cubic millimetre in females (range: 3.6 to 5.2) million [Seeley et al, 1989](p.561), providing one mechanism for higher blood pressure in males unrelated to differences in body size which may exist between the sexes. That is, greater pressure is required to force blood to flow the higher its viscosity due to a higher haemocrit. While this is a plausible mechanism for higher blood pressure in males than females as adults, most sex differences in BP in children are due to physical differences [Cresanta et al, 1986].

1.3.4.3 Race/Ethnicity

Ethnic or racial BP differences have been shown to exist in population-based studies of blood pressure. Most ethnicity/race studies of BP originate from North America, and a number have shown mean resting BP in African-Americans to be higher than in Caucasians. These findings have not been consistent, particularly after controlling for differences in body size [Gutgesell et al, 1981]. The Bogalusa heart study found black/white BP differences to begin in the early years of childhood after controlling for anthropometric differences [Voors et al, 1976; Berenson et al, 1989]. Some racial/ethnic BP differences may be due to differences in underlying illness prevalences which may contribute to population-based BP differences, and some BP differences have been associated with biological factors (eg, positively with urine sodium levels, negatively with plasma renin activity in African-American children [Berenson et al, 1979]); and with
anthropometric differences, for example body size (height, weight and adiposity). In some studies the BP differences did not remain after the biological or morbidity differences were factored out, while in others the BP differences remained.

1.3.4.4 Body mass, height, adiposity and growth stage

Various measures of weight, height and adiposity have been correlated to blood pressure. In adults the Body Mass Index (BMI) is often used to produce an indicator of obesity from height and weight alone. The BMI is equal to weight(kg)/height(m)². In children 14 years and younger the so-called Ponderal Index has been recommended as a better indicator of obesity. The Ponderal Index is equal to weight(kg)/height(m)³. However, no consistent measures of body size which best correlate with child BP have been found. For instance, one study of 13,700 children 10 years of age found that weight, height and height² had the highest correlation with systolic BP in boys, while in girls height and BMI were most strongly correlated with systolic BP. For diastolic BP, weight only in boys and height and BMI in girls were the most strongly correlated body size measures [Thomas et al, 1989].

Some of the inconsistencies of BP and body size indicators from various studies can be attributed to differences in body composition for a given weight, BMI or Ponderal Index. And methods have been developed to distinguish lean mass from total weight. Also, height can be influenced by prior childhood nutrition (eg, stunting due to low childhood protein intake), so that compound indices like the BMI and Ponderal Index may only approximately correlate with BP. Skinfold thickness as an indicator of fatness has been shown to correlate more with diastolic than systolic BP in some studies [Stine et al, 1975], while other studies found little correlation between skinfold and BP when height and weight were accounted for [eg, Stallones et al, 1982].

Skinfold thickness is prone to measurement error, particularly since repeated skinfold measures cannot be performed at the same location without allowing time for the epidermis to recover from the recent compression of the skinfold calipers. Also, skinfold thickness measurements of the very fat may correlate even more weakly with BP.

BP studies of puberty and precocious puberty have been more consistent, and show that neither puberty nor precocious puberty correlate with BP after body size has been
accounted for [Liker et al, 1988; Thomas et al, 1989].

Finally, at least one major study of multiple predictors of childhood BP, the Minneapolis Children’s Blood Pressure Study (n=9,977), found the strongest single anthropometric predictor of BP to be weight [Prineas et al, 1980].

### 1.3.4.5 Hormonal/metabolic factors

Hormonal influences on blood pressure, aside from normal regulation, can occur through hormonal imbalances occurring for various reasons such as diabetes. Serum insulin levels have been found to be positively correlated with child systolic BP after taking into account adiposity [Florey et al, 1976].

### 1.3.4.6 ‘Tracking’ of BP levels

Blood pressure tracking refers to the tendency of individuals in a percentile of blood pressure to remain in that percentile. Cohort studies have delineated the extent of blood pressure tracking in paediatric populations. For instance in the Bogalusa Heart Study, the previous year’s BP contributed a partial correlation coefficient of $r = 0.6$-$0.7$ to the variability in current BP for each age cohort, after controlling for other determinants in a multiple regression analysis [Voors et al, 1979].

BP tracking in children has been postulated to predict future hypertension as adults [Berenson et al, 1984; Bao et al, 1995], but this has been disputed. For example, a study of a random sample of 596 Dutch children aged 5-19 years who were re-measured 4 years later found 27% of boys and 44% of girls initially in the top systolic BP decile to remain there, compared to 25% and 22% respectively for diastolic BP. The authors concluded from these proportions that it was not possible to predict future adult hypertension from child BP readings alone [Hofman et al, 1985]. The Bao et al study [1995], however, showed high correlation between elevated childhood BP and BP measured 15 years later: of the 116 subjects who developed hypertension at follow-up, 48% and 41% had elevated systolic and diastolic BPs respectively at baseline. In any case, due to the well established and substantial auto-correlation in child BP found, within-subject BP change would need to take account of baseline BP measures in assessing factors contributing to child BP or its changes [Torok et al, 1986].
1.3.4.7 Pulse rate

Child blood pressure has been shown to be related to pulse (heart rate). In the Bogalusa Heart Study, the BPs of white children aged 5-17 years were found to be positively correlated with heart rate [Voors et al, 1982], but the correlation was not found in African-American children. From a longitudinal study of BP tracking in 2,946 Chinese children aged 4-14 years, by Li et al, pulse rate was found to correlate significantly and positively with both systolic and diastolic blood pressure, along with other factors [Li et al, 1995]. It appears that the correlation between pulse rate and BP weakens with age. Heart rates have been shown to decline with age also but differentially by sex: heart rates in adolescent males (12-17 years) appear not to correlate with their BP, while the correlation has been shown to remain in females of the same age [Schall et al, 1985].

1.3.5 Effect modifiers

A number of effect modifiers have been found to affect the blood pressure-stressor relationship. These have been linked mainly to personality types and attitudes to the stressor which are examined below in the context of noise as a stressor.

1.3.5.1 BP and sodium (Na) intake/excretion/sensitivity

There is much evidence to show that some individuals display salt (Na) sensitivity in their blood pressure, while others do not, pointing to a genetic component to BP salt sensitivity [Luft et al, 1988]. In child BP studies Na intake has been found to have little or no correlation with BP, and BP salt sensitivity consequently has been postulated to increase with age [Howe et al, 1991]. Child BP Na sensitivity has also been shown to vary by the degree of obesity of the child, with BPs in more obese children found to be more sensitive to changes in their salt intake [Rocchini et al, 1989]. However, salt levels in individuals are very difficult to measure accurately because of great natural day-to-day variability in urinary salt excretion and/or variability of salt intake from daily nutrients. It should be noted also that most of the work around the issue of regression dilution bias in BP has been due to the measurement error associated with variability in salt intake.
1.4 Noise

1.4.1 Definitions of noise

The physical characteristics of sound can be specified in terms of its frequency, intensity and duration. Noise has often been characterised simply as sound with an uncorrelated or random frequency spectrum. That is, the frequency spectrum of a sample of sound contains very few or no pure tones or harmonics. For example, Hirsh described noise as a “complex sound that has little or no periodicity” [Hirsh, 1952]. This definition takes no account of the volume or intensity of the sound, which when the listener is accounted for, may be sound with many correlated frequencies or harmonics but is perceived as too loud, or not, according to the listener’s “taste”. Conversely, uncorrelated sound such as the background hiss of a radio is not regarded as noisy by the listener because it is quiet enough to be imperceptible or is sub-consciously blocked out from being heard. At the very least, noise can be defined as unwanted sound, as argued by Berglund et al [Berglund et al, 1995](p.13), while loudness can be defined as “the subjective intensity of a sound” [Kryter, 1994](p.54).

Clearly, there is a strong subjective component to any adequate definition of noise, since the sonic input is processed in the cochlear and brain and is therefore subject to psychological interpretation which can vary between and within individuals, and between and within sound contexts. This is perhaps clearer at the ‘sonic borderline’ where there may be a number of harmonics or correlated frequencies in which the overall sound could be regarded by some as noise but by others as music [Hodgson, 1999]. Avant-garde music, for instance, may be unlistenable to most people and accordingly would be regarded by them as noise. However, within the context of a given type of sound, it should still be possible to define what is regarded as ‘noise’ and not noise. That is, aircraft overflights above a certain sound pressure level would be regarded by most as noise, but within the context of, say, chamber music the sound pressure level at which the sound is regarded as ‘noisy’ would be different. In short, comparing different sound sources for noisiness is not particularly useful if there is different cognitive meaning attached to the sound source (eg, transportation noise versus music) [Kryter, 1970]. At its most subjective extreme a sound may be very annoying, but not loud and with few or no uncorrelated frequencies. A good everyday example of this occurs with advertising in which a jingle is played repeatedly on radio or television such that one listener
eventually becomes quite irritated each time the jingle is played again, while another listener remains unaffected or copes by ‘tuning out’. This conceivably can occur in individuals who are not noise sensitive since there is no reason to believe that noise-sensitive individuals have a monopoly on annoyance from unwanted sound. Finally, it should be recalled that learned reaction to a sound source was central to Pavlov’s demonstration of associative learning, where gastrointestinal responses in the experimental dog, initially associated with the smell of food, were elicited by transference to the sound of a bell, sans smell.

Accordingly, noise reaction goes beyond physical noise measurement exposure because of the complexity and variety of sound needs to be considered along with the wide variability of listener reactions within and between individuals. On the one hand, organisms capable of hearing will have a universal startle reaction to a loud explosion or some other intense and sudden burst of uncorrelated sound; on the other hand, sudden or gradual bursts of correlated sounds may also invoke reactions similar in range to divergent musical tastes.

The context of the sound is also important since a noise reaction will occur if the sound is unwanted or unexpected. The same individual may not regard a sound as noise in one situation but may in another because the sound distracts from other tasks, or in some way lowers amenity, and is therefore unwanted. Furthermore, noise may be regarded as acceptable if it is associated with some benefit. Aircraft, road traffic or industrial noise in general may be acceptable to many because of the benefit or savings that modern transport or industry confers on population living standards. But it may also be acceptable only when not perceived, or at least as long as only others are frequently exposed to it (‘NIMBY’ -- Not In My Back Yard). The noise reaction is the result of a complex interplay of cognitive, hormonal and nervous responses mediated by attitude and sensitivity to the noise source and the context of the noise.

Part of the cognitive reaction to noise also relates to the individual making a decision at some level as to the price paid and benefit gained. Sound with high pressure levels and with spectra of uncorrelated frequencies may be noisy but endured if there is a perceived gain. The nearest and best example of this is aircraft passengers who are prepared to accept quite high cabin noise levels as a price for shorter travelling times.
Other components to the assessment of noisiness is the duration of the sound source; whether the onset of the sound is sudden or gradual, whether the noise is expected or unexpected, the number of times the noise event occurs in a given interval, and to a lesser extent Doppler effects (shifts of the whole sound frequency spectrum according to the relative motions of the source and observer) [Kryter, 1994].

In summary, noise or the extent of noisiness is not well characterised without the subjective input of the listener. From this perspective, noise is best described as annoying or unwanted sound that is loud and whose frequency spectrum is uncorrelated. For some types of noise the level of subjective annoyance can be gauged only through the self-report and reactions/annoyance of exposed subjects taking into account the context of their exposure.

### 1.4.2 Human Perception of, and Reaction to, Noise

To objectively measure noise mechanically with sound-level meters or spectrum analysers, account first needs to be taken of the characteristics of human hearing. This is to maximise the correlation between the sound’s objectively measured physical properties (eg, sound pressure level, frequency spectrum) and the psycho-biological (subjectively perceived) characteristics of the sound. Because different noise sources produce different reactions, mechanical noise measurement scales have needed to be supplemented by human noise reaction data, acquired from laboratory and social surveys pertinent to the particular noise source under investigation.

Before this level of ‘tailoring’ of the noise scale, it has nevertheless been possible to incorporate some human hearing reaction characteristics into mechanical noise measurement devices so that the measured sound pressure level correlates reasonably with perceived loudness. In short, the development of source-specific noise scales has involved a three stage process: (i) the development of a noise level scale which captures the human response to sound in terms of its loudness and perceived ‘noisiness’ or unwantedness; (ii) the development of an objective scale based on sound pressure level changes which accounts for onset duration and impulsivity of the noise source; (iii) the development of a composite scale of noisiness which correlates with human reaction to the particular sound source, taking into account subjective attitudes toward the sound, usually derived from community/social surveys and laboratory studies.
1.4.3 MECHANICAL MEASUREMENT OF NOISE

Sound is a phenomenon of alternating pressure propagated longitudinally in an elastic medium (eg, solids, liquids, gases). Its physical measurement is based on detecting changes in the pressure of the medium at a given location over a given time interval and is referred to as the sound pressure level. Sound pressure levels are expressed in decibels (dB). The decibel unit of sound pressure level (SPL) is defined as 10 times the logarithm to base 10 of the ratio of the mean square of measured sound pressure to the mean square of a reference sound pressure level. One reason for the log scale of the decibel is the large range of sound pressure levels relevant to the human listener, 7 orders of magnitude from $10^{-5}$ to $10^2$ Pascals [Berglund et al, 1995](p.4). Equivalently, the SPL is equal to 20 times the log to base 10 of the ratio of the root mean square of the measured sound pressure to the root mean squared reference pressure level [Kryter, 1994](p.5).

$$\text{SPL (dB)} = 10 \log_{10} \left( \frac{P_{\text{meas}}^2}{P_{\text{ref}}^2} \right) = 20 \log_{10} \left( \frac{P_{\text{meas}}}{P_{\text{ref}}} \right),$$

where

$P_{\text{meas}} =$ root mean squared measured sound pressure

$P_{\text{ref}} =$ reference pressure $= 20 \, \mu\text{Pa}$, by international convention (in micro-Pascal units).

[The Pascal unit of pressure is defined as 1 Newton of force acting uniformly on an area of 1 square metre; 1 Newton is defined as the force required to impart an acceleration of 1 meter/second$^2$ to a 1 kg mass]

Squared and mean squared values of sound pressure measurements are used in noise level calculations as it is the absolute amplitude of the pressure level which is important. Conceptually this is exactly analogous to the amplitude of a standing wave being defined as the distance from a peak or a trough of the wave to the mid or level point of the medium.

1.4.3.1 Development of the dB(A) scale

As human hearing responds differently to different sound frequencies for a given constant sonic intensity or sound pressure level, the reaction to a sound, in the first instance, is
conditioned by the frequency-dependent perception of its loudness and or noisiness. Most of the early work in human perception of loudness was conducted by Fletcher and Munson [Fletcher et al, 1933] who plotted perceived loudness versus sound pressure level across puretones of human sound frequency spectrum. Fletcher and Munson used the 1000 Hertz (1 kHz) tone as the standard sound against which other frequencies would be judged for loudness. Perceived loudness contours were produced over the range of audible sound frequencies in which the loudness was perceived to be of equal magnitude. From these contours a weighting of frequencies was produced to give the highest correlation between the perceived loudness and the measured sound pressure level. The resulting sound level scales were designated the dB(A) and dB(B) scales.

The dB(A) scale corresponds to the frequency weights derived from the 40 dB contour of Fletcher and Munson [Fletcher et al, 1933] and was first built into sound measuring meters in 1936 [Berglund et al, 1995]. Stevens [Stevens, 1955] defined a unit of loudness called the sone, based on a sound pressure level of 40 dB (in relation to the 20 μPa reference pressure) for the 1 kHz pure tone. The sone is a scaling measure such that a 2 sone sound is perceived as twice as loud as a 1 sone sound, a 4 sone sound twice as loud as a 2 sone sound, and so on. The relationship between sound pressure and perceived loudness was posited by Stevens as a “psychosocial” power law of the sound pressure level [Berglund et al, 1995]. In the mid-audio frequency range, this is such that a doubling of loudness, for instance, corresponds to a 10-fold increase in sound intensity which converts to a 10 dB increase in the sound pressure level [Stevens, 1957; Berglund et al, 1995]. Robinson et al defined the phon in units similar to dBA such that a tone perceived to be equally loud to the 1 kHz tone has the same phon value as the 1 kHz tone which is identical to the dB value [Robinson, 1956]. This expresses loudness as a sound pressure level equivalent to that occurring at 1 kHz. The relation of S (loudness in sones) to P (loudness level in phons) at phon levels above 40 dB is,

\[ S = 2^{(P-40)/10} \]

That is, to double S it is necessary to increase P by 10 phons, and at 40 phons the loudness is equal to 1 sone, as defined [Berglund et al, 1995].

The dB(B) scale corresponds similarly to the frequency weightings derived from the 70 dB equal loudness contour of Fletcher and Munson, while the dB(C) scale is unweighted across most of the sound frequency spectrum except for the very low and very high (audible) frequencies. All frequencies on the A- and B-weighted scales are weighted in
relation to 1 kHz which is unweighted. As a consequence of this and later work, most sound measuring instruments are calibrated to measure sound intensity on the dB(A), dB(B) and dB(C) scales.

In general, sound measured by the dB(C) scale produces noise intensity measures lower than the perceived loudness. To a lesser extent the dB(A) and dB(B) scales also underestimate perceived loudness, especially if impulsive sounds are being measured. In spite of these drawbacks, the dB(A) scale is considered to be the closest equivalent to the response of the human ear to a range of audible frequencies, and it correlates well with damage risk and reaction to annoying noises [Kryter, 1994].

### 1.4.3.2 The Effective Perceived Noise Level (EPNL)

The Effective Perceived Noise Level (EPNL) measure, first developed by Kryter in the late 1960s [Kryter, 1970], incorporates an objectively measured sound pressure level, dB(A) for example, with correction factors for the onset duration and impulsivity of the sound source. The EPNL was originally motivated by differing perceptions of aircraft noise depending on whether the noise was from propeller or jet aircraft. The EPNL, and a related measure, the sound exposure level (SEL) is at the core of the noise metric used for determining noise exposure in the Inner Sydney Child Blood Pressure Study and is outlined below.

The development of the EPNL begins with determining the physical aspects of a sound source from which the sound is judged subjectively as ‘noise’. A distinction is made between the objectively measurable loudness of a sound and the subjective ‘noisiness’ of the sound. According to Kryter [Kryter, 1994] the physical properties of a sound that can contribute to its perception as ‘noise’ are: (i) spectrum content and level; (ii) spectrum complexity, particularly the concentration of sound energy in narrow frequency bands within the broadband spectrum; (iii) the duration of the sound; (iv) duration of the sound pressure level prior to reaching the maximum level, for non-impulsive sounds; (v) the increase in sound pressure level within a short period (usually taken as 0.5 second, based on the functional characteristics of the auditory system), for impulsive sounds.

However, as Kryter points out [Kryter, 1994],
Defining perceived noisiness as a sensation inherently related to certain physical characteristics of sound appears to be inconsistent with two commonly held physiological and psychological tenets of hearing. The first is that all peripheral neural information is presumed to be coded for loudness and pitch discrimination; as a result, there is no neural information available from the cochlear on which to base a sensation such as noisiness. Second, and accordingly, a feeling of noisiness from stimulation must mean that a cognitive, learned, association is involved, so that the “feeling” is not a sensation as such; it is conventionally presumed that a learned relationship is not involved in the sensations of loudness and pitch.

Answers, if available, to such questions are to be found in empirical data.

Accordingly, the EPNL is constructed by correlating the spectrum properties of the particular sound source to perceived noisiness for given levels of loudness.

1.4.4 What is aircraft noise?

The type of aircraft noise that has generated the most reaction in the community is that originating from domestic jet aircraft. The introduction of jet aircraft on commercial routes in the 1950s was associated with major increases in noise and disruption to residents around large airports. Since the 1960s, when jet aircraft came to dominate air traffic around airports, increases in complaints to public officials and airports have been a catalyst for research into the effects of aircraft overflights in Australia and elsewhere [Bullen, 1984]. The growth in the literature has reflected continuing and increasing concerns about the effects of aircraft noise on the physical and mental health of exposed populations. Populations living near or under the flight paths of Heathrow (London), Los Angeles and Schiphol (Amsterdam) airports, in particular, have been studied in some detail.

Aircraft noise comprises a number of components originating in the mechanics of the aircraft and in its flight characteristics. Table 1.1 shows a breakdown of the components of aircraft noise. To these components may be added the conditions under which the aircraft is flying. For instance, aircraft noise from take-offs is different to that of landings, and within landings or take-offs conditions affecting the amount of noise produced also vary. Aircraft noise from take-offs can vary according to wind conditions, load, and distance to the first stopover of the journey (referred to as ‘stage length’). The heavier the load and/or the longer the stage length, the noisier will be the take-off, as the jet engines must run under greater power for take-off. Light wind conditions and hotter
weather where the air is less dense also mean that more power is needed for takeoffs since there is little or no aid to flight from a headwind. Noise from aircraft landings is subject to less variation, but is influenced by prevailing winds and the consequent use of thrust and reverse thrust for landing. Aircraft are also lower for longer during landings, so exposed populations will experience the noise from a landing event for longer than a take-off event.

Table 1.1 Selection of noise sources in domestic aircraft operation, the mechanism for their generation and areas/people affected (reproduced with modification from [Rodda, 1967]).

<table>
<thead>
<tr>
<th>Noise source</th>
<th>Noise generating mechanism</th>
<th>People affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jet engines</td>
<td>Turbulence aft of jet nozzle</td>
<td>People in vicinity and ground; passengers, crew</td>
</tr>
<tr>
<td></td>
<td>Interaction of turbulence and shock within jet and airframe</td>
<td></td>
</tr>
<tr>
<td>Axial-flow compressors and fans</td>
<td>Turbulence over blades; boundary-layer pressure fluctuations on blades; wake of compressor blades</td>
<td>People in vicinity and on ground; passengers, crew</td>
</tr>
<tr>
<td>Turbines</td>
<td>Turbulence and flow fluctuations over turbine blades</td>
<td>People on ground and passengers, crew</td>
</tr>
<tr>
<td>Centrifugal compressors</td>
<td>Turbulence, separated flows, and unsteady flow over vanes</td>
<td></td>
</tr>
<tr>
<td>Fuselage boundary layer</td>
<td>Wall fluctuating pressure exciting structure; turbulence in boundary layer</td>
<td>Passengers, crew</td>
</tr>
<tr>
<td>Propellers</td>
<td>Rotation effect of blades (blade thickness); rotation effect of blades under lift and torque forces</td>
<td>Passengers, crew</td>
</tr>
</tbody>
</table>

Overarching these factors is the politics under which aircraft operations take place, particularly the amount of noise-related community complaint generated. Flight paths, approach and take-off glide angles and other characteristics relating to flight operations at Sydney Airport have been subject to change according to political developments arising from community reaction to aircraft noise. Table 2.1 (in Chapter 2) shows a chronology of changes to flight patterns in response to community reaction since the opening of the new parallel runway at Sydney Airport on 4 November 1994.
1.4.4.1 Social surveys of community reaction to noise

The chief aim of social surveys of noise is to develop objective scales of noise measurement which correlate most closely with subjective annoyance reactions to the particular sound source. Various factors which may modify a reaction to sound are taken into account and include time of day (daytime, evening or nighttime); the type of sound source (eg, road traffic, industrial, aircraft); and the geographic location of the neighbourhood (rural, industrial/commercial, suburban). The social survey aims to establish weights for each of the above factors (in combination) so that when the physical properties of the particular sound source are measured objectively with sound measuring instruments the level of reaction or annoyance in a population can be predicted from the physical measurement by simply plugging the A-weighted sound level reading into a formula based on a combination of this and the survey-derived weighting factors.

Social surveys on noise annoyance have been conducted in England, France, Switzerland, Sweden, United States of America, West Germany, and Australia [Bullen, 1984]. Borsky [Borsky, 1961] is credited as the first to assess annoyance in terms of reported disturbance of specific activities including conversation, watching television, and sleeping [Bullen, 1984]. Subsequent surveys have also provided data on general annoyance, without direct reference to specific activities.

The predictive validity of noise metrics vis-a-vis annoyance was examined by Schultz when he synthesised the results of 11 international surveys conducted between 1961 and 1974 -- six studies of aircraft noise, four of traffic noise, and one railroad survey. Schultz found a consistent relationship between non-impulsive noise exposure (measured by \( L_{dn} \), an average day-night sound level) and community annoyance, irrespective of the source [Schultz, 1978], and concluded that different types of transportation noise could be accommodated in the same noise metric. The dose-response relationship has been reproduced in more recent community surveys [Hede et al, 1982; Schomer, 1983]. Support for Schultz's synthesis has not been unanimous, for example, Griffiths [Griffiths, 1983].

Other studies have shown that the association between noise exposure and annoyance does vary according to source [Hall, 1981; Kryter, 1982; Hall, 1984; Miedema et al, 1998], while the level of background (eg, road traffic) noise may not influence reported

For their own 1981 population survey of residents around Sydney, Adelaide, Perth and Melbourne airports, Hede and Bullen proposed using a lower level of reaction as a cut point, one more broadly based on affectedness, dissatisfaction, three annoyance ratings, and fear of an aircraft crash [Hede et al, 1982]. Their outcome measure was designated as the percentage ‘seriously affected’, rather than percentage ‘highly annoyed’. The investigators found that at ANEF (Australian Noise Exposure Forecast, an ‘energy averaged’ noise metric) of 20, 12% of residents reported being seriously affected by aircraft noise, and 38% were at least moderately effected. At 35 ANEF, 36% of residents reported being seriously affected and 73% were at least moderately affected. For details on the ANEF, see sections 1.4.4.3. and 1.4.4.4.

Bjorkman et al [Bjorkman et al, 1995] found a statistically significant and positive correlation between exposure and annoyance when exposure was measured as the number of noise events above 70dB(A), but only when these events occurred more than three times daily. The finding suggests that annoyance/reaction may be more highly correlated with exposure when using metrics with counts of extreme noise events as their basis rather than energy-averaged metrics, also supporting previous findings of Bjorkman et al [Bjorkman et al, 1992]. The findings also suggest that a minimum threshold number of noise events may need to occur before measurable noise reactions also occur.

Despite the predictive value of noise measures using aggregated data, noise metrics fail to predict individual responses to noise accurately, signifying that noise exposure is not the only factor involved in annoyance reactions. With regard to generic type of noise, community reaction/annoyance has been shown to be higher for impulsive than for non-impulsive noise sources [Bullen, 1991], but the correlation of individual reaction levels to impulsive noise also has been shown to be lower than for non-impulsive noise [Job, 1988]. Job estimates that approximately 20% of variation in reaction to noise is due to the noise exposure, and that attitude and sensitivity to the noise accounts for greater variation in noise reaction. While attitudes and sensitivity to the noise source have been shown to be correlated with reaction to the noise, this correlation has been stronger with impulsive noise than non-impulsive noise [Job, 1988]. In short, attitude and sensitivity to the noise source plays a larger role in modifying individual reactions to impulsive noise than to non-impulsive noise.
Other psycho-social factors have been found to affect noise perception and annoyance reactions to noise, depending on the noise source, as established in community surveys [McKennell, 1963; MIL Research Ltd, 1971; Schultz, 1978; Borsky, 1979].

With regard to aircraft noise, annoyance reactions have been found to be greater in people who indicate a fear of aircraft crashes, are concerned about the health effects of noise, or report interference with activities such as watching television, talking and sleeping [Schomer, 1983]. Recorded aircraft noise when it interfered with tasks requiring concentration (for instance, proof-reading and figure-tracing) was perceived to be more annoying and less pleasant than the same noise when these tasks were not being attempted [Moran et al, 1977]. People who report that they are sensitive to noise, so-called “noise sensitive” individuals, are also more likely to indicate intense annoyance reactions [Fiedler, 1975; Moran et al, 1977].

Surveys have reported that: (i) socio-demographic factors of age, sex, marital status and socioeconomic status have low correlation with individual reactions to noise, and that SES correlates more strongly and positively with complaint behaviour [Schomer, 1983] and (ii) complaint behaviour showed lower correlation with noise exposure than did noise reaction [Schomer, 1983]. Bullen concluded from similar findings that, “In general, the number of complaints received is a very poor guide to the extent of noise reaction in the community...”[Bullen, 1984].

The relationship between aircraft noise annoyance and population health has been examined is some detail in surveys of people living proximate to London’s Heathrow Airport and Amsterdam’s Schiphol Airport in particular. Much of the Heathrow evidence has come from a residential survey (n=6,000) of aircraft noise and mental health conducted in areas around Heathrow Airport [Tarnopolsky et al, 1980; Jenkins et al, 1981]. Watkins et al found a significant association between annoyance level and reporting of individual psycho-social and physical symptoms and the use of medications, which was independent of noise exposure level [Watkins et al, 1981]. From the same survey Watkins et al concluded, “The uptake of psychotropic drugs, and the use of GP and outpatient services, increase with increasing annoyance in both low- and high-noise areas, though the increases do not in all cases reach the 5% level of statistical significance. The use of non-prescribed drugs, on the other hand, increases significantly (p < 0.001)
with annoyance in high noise, but shows no relation with annoyance in low noise conditions.” [Watkins et al, 1981]

Further analysis of the same community survey by Kryter showed that the psychiatric patients were disproportionately annoyed by aircraft noise, indicating that aircraft noise may effect these people more adversely than the general community [Kryter, 1990].

A study by Graeven found that a significant correlation between annoyance from aircraft noise and the number of symptom complaints reported in the previous week was independent of other factors, including level of noise exposure [Graeven, 1974]. Fiedler et al reported that about half the people bothered by aircraft noise attributed some kind of personal effect to it [Fiedler et al, 1975]. More than half of these effects were psychological in nature, centring on reported nervousness and irritability, and the frequency of these complaints did not differ according to the airport noise zone.

A complaints hotline set up by the New South Wales Health Department to monitor community reaction to the opening of the new parallel runway in 1994 at Sydney airport produced the following results [Senate Select Committee on Aircraft Noise in Sydney, 1995]: of about 1,700 health-related complaints attributed to aircraft noise (from = 450 callers), ≈ 20% were for sleeping difficulties; a similar proportion were for increased (mental) tension; 15% increased anxiety; and ≈ 10% fatigue. Other complaints included headache, pollution effects, tinnitus, breathing difficulties, and child sleeping and concentration difficulties. No information on exposure was reported.

Results reported by Lercher [Lercher, 1995] of a survey of 1,989 respondents from 5 villages in rural Austria suggest an association between aircraft noise exposure above 55dBA (assigned levels), and higher reporting rates of tiredness, nervousness, “loss of well-being and safety”, sleep disturbance, headache and palpitations [Lercher, 1995]. These rates were adjusted for age, sex and education. When annoyance was taken into account the association between the exposure and reporting rates was stronger. The participation rate for this study (≈ 62%) could render some of its findings biased in that the remaining ≈ 38% of the population could conceivably contain a lower proportion of noise-sensitive individuals.

The findings of Björkman et al [Bjorkman et al, 1992] and Lercher [Lercher, 1995] appear to contradict the findings of Job [1988] where the former found annoyance and
reaction to be correlated more highly with exposure when the latter was measured as
counts of extreme events rather than as energy-averaged noise levels. These findings are
also in conflict with those of Hede et al [Hede et al, 1982](p.11) who concluded from
the Australia-wide social survey of airport noise that ‘...“equal-energy” indices, eg NEF
[Noise Exposure Forecast], show a significantly stronger relationship with community
reaction than other types of index tested, including “peak-level” indices and indices which
are independent of the number of overflights per day.’ It remains to be established if
noise metrics based partly on annoyance/reaction levels are appropriate for correlating
with possible physical health outcomes such as blood pressure. Certainly there appears
to be different findings according to how ‘annoyance’ is distinguished from ‘reaction’ to
the noise.

1.4.4.2 Event-based noise metrics

Two event-based measures of noise exposure have been most commonly used. The first
is the Noise Number Index (NNI) which has been used mainly in the UK and Europe and
is based on the number of aircraft noise events. The second commonly used event-based
noise measure is the “N70” metric which is defined as the number of noise events
occurring per hour at sound pressure levels of 70 dB(A) or above, usually averaged over
24 hours [Bjorkman et al, 1992].

1.4.4.3 Energy-averaged noise metrics

The measure of aircraft noise used in this study is an energy equivalent type of noise
metric, used in conjunction with aircraft noise certification data as part of the Integrated
Noise Model (INM) [Peploe, 1996]. That is, each aircraft type has a characteristic noise
signature under different flight conditions (landing, take-off, powering up or down, and
so on) which is determined at the time of the aircraft’s manufacture. Aircraft-type noise
information contained in the INM is used in conjunction with empirical flight track data
to characterise and estimate quantitatively the noise exposure. The INM is used
commonly throughout Australia [Peploe, 1996]. However, the INM itself does not
adequately model noise transmission to affected areas, tends to be less accurate at lower
noise levels, and is unable to account for variations in terrain and other local noise
propagation conditions [Peploe, 1996]. The INM may also inadequately characterise the
extent of sideline attenuation of aircraft noise. In other words, the INM can adequately
characterise residential noise exposure vertically underneath a given aircraft type operating under given flight conditions, but not for exposures lateral to the aircraft flight path. For the Sydney Airport study supplementary noise measurements were taken to check the INM’s accuracy overall and to assess the extent of sideline attenuation in non-vertically exposed residents, by the so-called ‘slant distance’ [Peploe, 1996].

The approach underlying the construction of an energy equivalent noise metric essentially equates the perceived intensity of a single noise event of given sound pressure, duration and frequency with multiple noise events of different durations, frequencies or intensities. Energy-averaged noise levels are calculated for a given location and period of exposure to flights of differing aircraft types at various altitudes, bearings and under different fuel and power settings according to stage length (distance to first landing stopover).

Most noise metrics, including cumulative energy averaged measures, have at their core an aggregation of a single noise event measure, the maximum of either a construct called the Perceived Noise Level (PNL), or a development of the PNL called the Effective Perceived Noise Level (EPNL). The PNL was first developed by Kryter and others in the late 1950s and attempted to capture on a single scale levels of human annoyance with unwanted noise across different frequencies, durations, frequency of occurrences, and intensities [Kryter, 1959]. The PNL attempted to distinguish between objectively measured loudness (for example, physically measurable sound pressure level on the dBA scale), annoyance (subjective human reaction specific to the type of noise and its context, along with its loudness); and noisiness, conceived of as lying between loudness and annoyance, “intended to describe the inherent undesirability of a given sound, but without being influenced by the context in which the sound is heard” [Berglund et al, 1995]. The PNL is defined as “being numerically equal to the sound pressure level of a reference sound (defined as a frequency band limited random noise signal from 910 to 1090 Hz) judged by listeners to have the same perceived noisiness as the given sound” [Berglund et al, 1995].

The EPNL unit was developed to incorporate adjustment for the duration of overflight events [Berglund et al, 1995], and was later incorporated into a cumulative noise metric called the Noise Exposure Forecast (NEF) [Berglund et al, 1995]. The Australian Noise Energy Index, Concept and Forecast metrics (ANEI, ANEC, ANEF respectively) are developments of the NEF and take into account day/night loadings from social surveys.
conducted in Australia.

In general, a noise index has the form:

\[
\text{Noise Index} = \text{Energy level of noise events [dB(A)]} + k \cdot \log(\text{number of events})
\]

Noise levels are usually added on an energy basis. That is, a 10-fold increase in the number of events at a given level is equivalent to a 10dB increase to that level (ie, setting \( k=10 \), in the above formula). Different social surveys have established different values for \( k \). That is, the number of events of noise at a particular intensity required to equal an event of a given standard intensity is determined by community surveys of affected populations. Hede and Bullen’s work in Australia established a value of \( k=12 \) [Hede et al, 1982]. In principle, scales of subjective reaction to noise are used in noise reaction survey instruments to assess the relationship between noise intensity and the number of such events that occur in a given time interval. For instance, the noise of a given intensity and frequency band associated with an adverse reaction in subjects can be equated to noise of a different intensity and frequency band which generates the same level of reaction. Consequently, based on empirically derived values for \( k \) from survey data, the noise index for a given geographic point can be inferred directly from objectively measured noise intensity in conjunction with the number of times events of such magnitude occurred over a given time interval.

A number of attempts have been made to incorporate evening as well as night weightings into energy-averaged noise indices. A day/night weighting value of 4 was derived by Bullen et al for the ANEI/ANEC/ANEF noise metric in Australia, based on their survey work [Bullen et al, 1983]. It should be noted that at the time of writing no weighting has been developed in Australia for geographic variation: airports situated in quiet rural areas versus noisy urban environments, for example, might produce higher levels of community reaction to aircraft noise for given sound intensities and frequency of overflight occurrences.

Overall, noise metrics based on community surveys explain about 15-20% of individual variation in reaction to noise [Job, 1988]. It is thought that much of the unexplained variance is due to individual differences which often are assumed to be distributed non-differentially according to noise exposure level. Based on this assumption it has been
argued that aggregate measures of reaction cancel out some of these individual variations [Job, 1988]. Accordingly, it has been shown that a stronger correlation exists between reaction to noise when both are measured at an aggregate rather than at an individual level [Job, 1988]. Some of this correlation may be due to artefact, due to sample clustering and/or assignment of single noise exposure values to whole populations. On the other hand, stronger correlation at an aggregate than individual level may reflect regression dilution bias where the error in a predictor variable (noise exposure) at an individual level leads to an underestimate of the true magnitude of association between the predictor and outcome variable at an individual level [Gardner et al, 1973](see also section on regression dilution bias, above).

Nevertheless, the assumption that individual reactions to noise should not be distributed differentially according to measured noise level may not necessarily hold. It is feasible that noise-sensitive individuals will tend to avoid living in high noise areas, or would tend to live in such areas for shorter time periods. For example, the follow-up of schoolchildren in the Los Angeles Airport BP study [Cohen et al, 1981] found that the children with the highest BP at baseline had moved away from noise-affected schools. These individuals, or their parents, may have been more sensitive to aircraft noise than the remaining sample, and moved to quieter locales at the first opportunity. This argument does assume that populations can move at will, whereas noisy areas tend to be of lower socio-economic status (through lower real estate values), so that populations in these areas are less mobile through economic compulsion. Accordingly, while it would be unsafe to assume that in general noise sensitivity is non-differentially distributed across different noise exposures, this is modified by socio-economic reality. The effect of such differential distribution of noise-sensitive individuals would in principle be a partial neutralisation of possible associations of noise with physical or psychological parameters -- that is, a bias toward the null. The extent of this bias is difficult to determine.

1.4.4.4 Steps in construction of an ANEI (or ANEC or ANEF) value at a given grid point

The steps for constructing the noise metric used in Australian aircraft noise measurements are as follows:

1. Calculate distance from the grid point to the closest point of approach of the flight track flown by the aircraft.
2. Calculate the distance along the flight track from the start of takeoff, or runway threshold for landings, to the closest point of approach.

3. Using the distance calculated in 2, in conjunction with the take-off or landing profile of each aircraft type (including stage for takeoffs), obtain from flight tracking records the aircraft’s power setting, altitude and speed at the closest point of approach.

4. Using the distance calculated in step 1 and the altitude from 3, calculate the slant distance from the grid point to the aircraft at the nearest approach point on the flight track.

5. Using the slant distance from 4 and the power settings from 3, obtain the aircraft’s unadjusted noise levels to which the grid point is exposed (from the aircraft type noise certification data).

6. Adjust the noise levels using data obtained in 3 to account for the aircraft’s speed, elevation angle and whether the closest point of approach is on a straight line or a curve. (Obviously, more complicated flight tracks pose further computational difficulties).

7. Once the adjusted noise level has been derived, add the daily number of operations on each track by the aircraft type.

8. Add logarithmically the noise levels obtained in 7.

9. Repeat the process for all aircraft types on that flight track for that grid point. Add these levels logarithmically

10. Repeat process for all flight tracks in relation to the grid point.

11. Repeat process for all grid points.

The partial ANEF at a grid point is given by:

\[ \text{ANEF}_{ij} = \text{EPNdB}_{ij} + 10 \times \log(N_{\text{day}} + 4 \times N_{\text{night}}) - 88 \]
where \( \text{ANEF}_{ij} \) = noise exposure due to aircraft type \( i \) and flight path \( j \).
\( \text{EPNdB}_{ij} \) = noise level of aircraft type \( i \) and flight track \( j \).
\( N_{\text{day}}, N_{\text{night}} \) = number of flights during the day and night respectively of aircraft type \( i \) and flight path \( j \).

The total ANEF at a grid point is given by:
\[
\text{ANEF}_i = 10 \log \left( \sum_{i=1}^{i=t} \sum_{j=1}^{j=n} \text{antilog} (\text{ANEF}_{ij}/10) \right)
\]

where, \( i \) = total number of aircraft types
\( j \) = total number of flight tracks
\( \text{ANEF}_i \) = total noise exposure forecast at that point

The EPNL can also be derived empirically from Sound Exposure Levels (SELs) from tape recordings and 3rd octave measurements of aircraft flyovers, and compared with or used as an alternative to the EPNLs in aircraft certifications contained in the Integrated Noise Model (INM) data base [Peploe, 1996]. The SEL also has the advantage of simplicity of measurement.

In practice for the Sydney Airport study due to their numbers, individual flight tracks were aggregated into ‘mean tracks’ for each aircraft type, and noise levels were calculated according to the above algorithm from the aggregated flight tracks [Peploe, 1996].

1.5 Noise and Health

1.5.1 Definitions of Health

Of the three major types of definitions of health, that most commonly quoted is by the World Health Organisation, “health is not merely the absence of disease or infirmity but is a positive state of physical, mental and social well-being” [WHO, 1994]. This definition is inadequate for population-based studies since it does not include premature death of those who otherwise may be in a “positive state of physical, mental and social well-being” (although it does include illness and disability), and there is a lack of agreed measures of well-being.
Health can also be considered as successful adaptation of individuals or groups to environmental circumstances. This requires that “successful adaptation” be defined. On an individual level, this is usually considered to be independent living and normal social interaction, and thus can include well-adjusted people with severe physical handicap. On a population level, it may be considered as perpetuation of the species at near zero population growth with minimal environmental disturbance, or adjustment to environmental changes with little or no social dislocation. In relation to aircraft noise, those who adapt would be considered healthy, while those who did not might be considered unhealthy or as potential candidates for future ill-health.

The third or “classical” approach is to consider that individuals are healthy until they are determined not to be so, and to use a range of comparative population measures of mortality, morbidity and impairment to determine the relative health of various groups. This approach has the advantage of using routinely available data, but there is difficulty in defining ‘disease’ at the margins. For example, in mental health the definition and separation are indistinct between clinical anxiety and depression on the one hand, and anger, annoyance, irritation, sadness, loss of morale and other normal sensations on the other. Furthermore, disagreement in the medical literature on the role played by “stress” in the aetiology of illness indicates current uncertainty regarding plausible biological pathways for mental and emotional states in ‘determining’ or ‘pre-determining’ physical health [Freeman, 1990].

1.5.2 “Stress” and health

Despite some lack of consensus on its definition, “stress” has been suggested as the primary mechanism through which external stimuli can affect mental and physical health, including from noise sources of stress. Physiologically, stress has been shown to manifest as a complex of autonomic endocrine processes centred on the pituitary-adrenocortical axis [Krantz et al, 1987], but under certain conditions the physical outcomes of stress have been shown to be modified by the context of the source of stress and by the individual’s attitude to the stressor.

1.5.2.1 Development of stress as a determinant of health

“Stress” was studied and systematised by Hans Selye who in 1936 employed the term to
refer to the reaction of the body to an environmental threat or challenge [Selye, 1950]. Selye proposed a three stage ‘general adaptation syndrome’ (GAS) model of the stress process [Selye, 1950].

The first ‘Alarm’ stage in the GAS model was characterised by secretion of adrenal hormones as a response to the presence of the noxious agent. A ‘Resistance’ stage followed where the organism adapts to the stressor, with attenuation or elimination of symptoms achieved by elevated but stable outputs of corticosteroids. The third ‘Exhaustion’ phase occurs when the ‘stressor’ (a term also coined by Selye to refer to the external agents causing the stress [Selye, 1950]) is severe or prolonged enough to overpower somatic defences. The adrenal cortex and anterior pituitary glands are unable to maintain high hormonal secretion levels and symptoms reappear. Organs specifically vulnerable to the particular stressor can be damaged if exposure to the stressor continues unabated [Krantz et al., 1987].

Selye defined the GAS as: “the sum of all non-specific systemic reactions of the body which ensue upon long-continued exposure to systemic stress” [Selye, 1950]. Central to the GAS are three key notions:

(i) that different alarming stimuli, or ‘stressors’ (viz, the alarm stimuli responsible for a biological response) produce similar systemic biological effects. That is, both the stressor is non-specific in that more or less extended areas of the body are affected by exposure to the stressor; and the body’s reactions to the stressor are also non-specific in that the same somatic reactions occur in the presence of different stimuli.

(ii) that the 3 stages of the GAS are biologically distinct.

(iii) that the pathological effects of a given stressor are the result of an organism reaching the exhaustion phase, when the defences of the adaptation phase are no longer sufficient to withstand exposure to the stressor, leading to physical damage.

Each stage in the GAS is characterised by somatic changes conjointly occurring as a syndrome characteristic of each stage. In some stress reactions, there is ambiguity at the boundaries of Selye’s stages, but the stress reaction in general can be identified as a common set of somatic responses that can occur under the action of a variety of different
stressors.

The first stage of a reaction to a stressor, the alarm stage, was regarded by Selye as comprising a ‘shock’ phase, followed by an ‘counter-shock’ phase. Conditions associated with the shock phase include hypothermia, hypotension, depression of the nervous system, hemoconcentration, changes in capillary and cell membrane permeability, hypochloremia and hypoglycemia. Selye regarded this phase as not entirely ‘shock-like’, since some of the above occur in an alarm reaction without the classic indicators of shock, while some may be absent within an overall shock condition.

According to Selye, the ensuing ‘counter-shock’ phase of the alarm stage represented the transition to the adaptation/resistance stage, and is often indistinguishable from the latter in the case of chronic exposure to a stressor. A number of the conditions associated with shock are reversed in ‘counter-shock’ (eg, hypoglycemia → hyperglycemia, etc), mainly through release of corticosteroids from an enlarged adrenal cortex.

According to Selye, the basis for pathology resulting from stress is a failure of the body to adapt to the stressor. Selye classified the failure to adapt into hypo-, hyper- and dysadaptation (ie, insufficient, excessive, and abnormal function) [Selye, 1950](p.12). The excess of anterior pituitary and adrenal-cortical hormones produced during the GAS, over and above their normal levels, effectively defend the body’s various systems for relatively short time periods, but the presence of such hormonal excess for extended periods can lead to cardiovascular, renal or joint diseases [Selye, 1950](p.14). Such conditions Selye labelled as diseases of adaptation, and subdivided these further into primary and secondary diseases of adaptation [Selye, 1950](p.15). Primary diseases of adaptation were defined as those resulting from lesions in the organs of adaptation themselves, for example abnormally functioning pituitary gland (eg Cushings disease as pituitary hyperfunction, Simmonds disease as pituitary hypofunction); or adrenal glands (Cushing’s syndrome as adrenal hyperfunction, Addison’s disease as adrenal hypofunction); or renal damage leading to renal hypertension. Secondary diseases of adaptation are the consequences of malfunctioning of the organs of adaptation. Selye included among these conditions some types of hypertension, nephritis and diabetes, rheumatic diseases, and various psychosomatic disorders.

Selye’s theories of cardiovascular system participation in the stress reaction centres on
the neuro-hormonal regulators of the stress reaction (GAS), including sympathetic 
stimulation of hormonal secretion from the adrenal medulla or the action of 
adrenocorticotropin hormone (ACTH) on the adrenal cortex, and the secretion of 
vasopressin by the posterior lobe of the pituitary gland.

Selye elaborated his schema with numerous examples from the extant literature.

Selye's GAS model has been criticised as being too general in that different stressors can 
have distinct, sometimes opposite effects on hormone response levels (‘specificity’) 
[Krantz et al, 1987], despite many examples of different types of stressors producing a 
similar generalised reaction. For instance, Mason showed that secretions of 17-
hydroxycorticosteroid in monkeys increased, decreased or remained unchanged 
according to the type of stressor [Mason, 1971]. While the physical mechanisms for 
stress are now well documented, the mechanisms for effect modification by cognitive, 
personality, attitudinal and contextual variations are less so, in spite of mounting 
evidence for effect modification, not least in the area of noise research.

1.5.2.2 Stress and personality type

The physiological stress response has been found to be modified by innate or learned 
predispositions of the organism [Seligman et al, 1971; Dembroski et al, 1985]. 
Investigations into stress and personality, coping mechanisms, and ability to control 
exposure to the stressor, have yielded significant variations in individual physiological 
reactions to stress.

For example, so-called Type-A personality individuals have been shown to respond more 
actively to stressors including noise than Type-B personality individuals [Weidner et al, 
1978; Glass et al, 1980; Lovallo et al, 1980], and including children [Hunter et al, 1982; 
Lawler et al, 1981; Murray et al, 1985]. Type-A personality individuals are classified as 
being more aggressive, hostile, and competitive, more prone to time urgency, and tend 
to cope more actively with stressors than Type-B counterparts. Type-A physiological 
response patterns are characterised by excess catecholamine secretions and 
cardiocirculatory reactivity, compared to so-called Type-B personalities [Henrotte et al, 
1985].
However, studies of blood pressure and personality type have yielded mixed results. Haynes et al, from the Framingham Heart Study, found Type-A personality to independently predict incidence of coronary artery disease and myocardial infarction--but only in white-collar males and females aged 45-64 years [Haynes et al, 1980]. A 22-year follow up by Ragland et al of the Western Collaborative Group Study cohort (WCGS, N = 3,154), found Type-A hypertension rates to be not significantly different from those for the Type-B personality group [Ragland et al, 1988]. This was despite an earlier finding on the same subjects (after 8.5 years follow up) [Rosenman et al, 1975]. The Rosenman et al study appeared to show a statistically significant relative risk of $= 2$ for Type-A patients acquiring coronary artery disease. After controlling statistically for traditional risk factors, Ragland et al’s re-analysis of the 8.5 year follow up data of the WCGS also reversed Rosenman et al’s original finding [Ragland et al, 1988].

In the mid-to-late 1970s angiographic studies showed Type-A patients to have more extensive coronary artery disease than those classified as Type-B [Zyzanski et al, 1976; Blumenthal et al, 1978; Frank et al, 1978]. An angiographic study of 2,289 respondents by Williams et al found that Type-A personality was significantly associated with the severity of coronary artery disease--but only in those aged 45 years or younger [Williams et al, 1988].

From the early 1980s, further negative findings on personality, stress reaction and cardiovascular outcomes emerged from prospective studies such as MRFIT [Schwalbe, 1990], the Aspirin Myocardial Infarction Study [Shekelle et al, 1985] and the Multicenter Postinfarction Program [Case et al, 1985]. In the latter investigation, for example, the negative result confirmed other studies that showed no personality effect on subsequent coronary artery disease (CAD) events in those who had already suffered a CAD event; or in those who were at high risk for CAD from other factors [Case et al, 1985]. A higher proportion of negative findings appear to have arisen from studies using a self-administered questionnaire for personality assessment than in studies using semi-structured interviews [Matthews et al, 1986;Dimsdale, 1988 (quoted in Littman, 1993)].

In relation to children, from the Minneapolis Children’s Blood Pressure Study, Lee et al found that from childhood to adolescence Type-A individuals did not have significantly higher blood pressure than Type-B individuals, nor did the BP of Type-A individuals increase more rapidly than Type-B individuals over a 10 year period [Lee et al, 1996].
With the shift in focus to a subset of traits within the Type-A personality construct, more consistent cardiovascular findings have emerged. Re-analysis of the MRFIT and WCGS data showed that hostility was strongly predictive of coronary artery disease and coronary events [Dembroski et al, 1989]. Also, angiographic studies by MacDougall et al have shown hostility to be significantly related to coronary artery disease [MacDougall et al, 1985].

In children, data from the Bogalusa (Louisiana, USA) Childrens Cohort suggest that in comparisons between highest and lowest ‘Eagery’ factor quintiles (from the Hunter-Wolf A-B scale [Hunter et al, 1982]), the upper quintile group had mean readings of triglycerides, β-lipoprotein cholesterol and total cholesterol = 10 mg/dl higher than the lower quintile [Hunter et al, 1982]. These differences were statistically significant.

A study of child personality and cardiovascular reactivity carried out in Germany indicated that type-A school children were no different from type-Bs in blood pressure or heart rate when at rest. But during video game stress tasks type-A boys had pronounced diastolic blood pressure reactions compared to type-B boys, while the girls showed no difference [Schmidt et al, 1986].

Contrary findings with regard to the Type-A personality in adolescents were reported by Seigel et al, where systolic BP variation and peaks in systolic BP were positively associated with Type-A personality as well as Type-A subsets of hostility and quick speech latency. In contrast, diastolic BP was not significantly associated with Type-A or Type-A traits [Siegel et al, 1983].

In summary, despite the findings on personality and BP not being consistent, systolic BP and, to a lesser extent, diastolic BP appear under differing stimulus conditions to be positively associated with personality type, in particular with the aggression/hostility subsets of the Type A personality construct.

### 1.5.2.3 Stress and “locus of control”

The pioneering studies of Seligman established controllability of exposure to the stressor (as distinct from the ability to eliminate harmful effects of the stimulus) as an important effect modifier of the stress response [Seligman et al, 1971; Seligman, 1972]. Seligman’s
concept of ‘learned helplessness’ was a construct for impaired cognitive performance and depressed mood frequently associated with physiological responses such as elevated catecholamine levels. ‘Learned helplessness’ has been shown to occur in a wide variety of experimental and naturalistic settings where respondents have limited or no control over their exposure to the stressor, and has been shown to be accompanied by the classic somatic stress responses [Murison et al, 1993]. It should be noted that ‘learned helplessness’ appears to parallel Selye’s Exhaustion stage in the GAS, as the outward psycho-social/behavioural manifestation of the Exhaustion stage.

Somatic stress responses have also been linked to the amount of control a subject has over their exposure to stressors in occupational settings. Locus of control studies by Karasek et al have suggested that people in demanding work with little intellectual scope for control over the work suffer higher rates of morbidity and mortality from coronary artery disease [Karasek et al, 1981]. Marmot et al came to similar conclusions in their study of British civil servants in which it was found that those with less control over their work had higher rates of cardiovascular disease after controlling for confounders such as age, smoking status and physical fitness [Marmot et al, 1978].

Cardiovascular outcomes may occur as a result from situations which are perceived to be a psychological threat. For example, a prospective cohort study of self-perceived psychological stress in 6,935 Swedish males aged 47 to 55 years, by Rosengren et al, found those at baseline who reported suffering ‘permanent’ stress for the past 1 or 5 years, to have a relative risk of 1.5 of a non-fatal myocardial infarction or fatal coronary artery disease later on, compared to all the other groups who reported less or no stress. Age and other risk factors were controlled for, and a similar effect was found for risk of stroke. No dose-response relationship was evident. Relative risk of death from cardiovascular diseases was found to be significantly higher in the two highest stress groups only [Rosengren et al, 1991].

Contrariwise, in a study of 1,040 normotensive bus drivers in San Francisco by Winkleby et al, self-reported stress was inversely related to levels of hypertension, but related positively to gastrointestinal, respiratory and musculoskeletal ailments [Winkleby et al, 1988].

In a prospective study of cardiovascular effects of anxiety, Kawachi et al followed
39,999 male health care professionals aged 42-77 years for two years. The investigators found that the relative risk of fatal coronary heart disease in those who reported high levels of phobic anxiety at baseline was $\approx 3$, compared to those with low levels of phobic anxiety. Moreover, the authors found a consistent dose-response relationship between levels of anxiety and relative risk of fatal CHD. Non-fatal myocardial infarction rates remained unaffected by levels of anxiety, however [Kawachi et al, 1994].

Studies relying on self-reported stress or anxiety suffer from inherent measurement problems: to one individual subjectively-felt stress may not be stress to another, even though both could be experiencing similar autonomic responses to stressors. Yet, as the Swedish and health professionals studies cited above illustrate, self-assessment of health appears to be a better predictor of an objective outcome such as myocardial infarction or sudden death than the more objective clinically based studies. Whether the relationship is consistent with hypertension or elevated BP remains unclear, however, especially given the inverse finding from the San Francisco bus driver study.

1.5.2.4 Stress, locus of control and personality type

Coping reactions to stressors have been investigated in terms of learned helplessness and personality type, shedding light on the relationship between the ‘coronary-prone’ type-A personality and learned helplessness. A study by Krantz et al showed Type-A personality subjects to rate themselves as more helpless than Type-B subjects when exposed to intense uncontrollable noise, but not when exposed to moderate uncontrolled noise [Krantz et al, 1974]. Moreover, for a given intensity of noise stressor, the Type-A subjects did not rate the noise stressor as more unpleasant than did the Type-B subjects. In short, the psychological stress in Type-A personalities manifests from lack of control rather than greater sensitivity to stressors.

In conclusion, biological mechanisms for acute somatic effects from exposure to stressors have been well established and documented. Reactions to stressors have also been shown to be modified by context (controllability/learned helplessness, other tasks being performed), by perceptions of stress, and by personality. Cardiovascular reactions to stressors have been shown to occur, more so acute than chronic, and to also be modified by context and personality. The evidence for long term exposure to a stressor resulting in permanent or chronic cardiovascular effects has not been established in human studies.
As a consequence, none of a number of biologically plausible mechanisms for chronic cardiovascular effects from long term exposure to stressors have been shown to be activated as a result of long term exposure to stressors known to provoke acute cardiovascular responses.

1.5.3 Noise as a Stressor

Noise has long been recognised as a source of physiologic stress, such that noise is the stressor of choice in laboratory-based stress reaction studies. Reactions to noise can depend on the noise source itself, and individual differences in reaction to noise in turn can depend on the individual attitude to the noise source. As a consequence, different measures of reactivity to noise have been developed according to the noise source. Most measures of noise reactivity, generally called noise ‘metrics’, have been derived from community surveys which attempt to marry an objective measure of noise intensity (eg, sound pressure in dB(A)) with other aspects of the noise source including pitch or change in pitch, impulsivity, frequency (all combined into the so-called Effective Perceived Noise Level, EPNL). The EPNL in turn is combined with factors such as the number of noise events, weightings for day/night noise events, residential location and restrictions in amenity or activities attributable to the noise to produce ‘metrics’ which predict a level of reaction in a community for a given sound intensity and number of noise events.

The possible role of stress in relation to aircraft noise and cardio/cerebrovascular disease outcomes can be summarised as follows:

(i) Individuals who perceive that they are undergoing continual psychological stress as a result of exposure to aircraft noise may be at higher risk of becoming hypertensive, suffering a stroke, or suddenly dying from coronary artery disease. It is possible that the effect is more pronounced among middle-aged or vulnerable subgroups or in those at the extremely high end of a self-reported stress scale. Physiologically, this may correspond to the Exhaustion stage in Selye’s GAS scenario, where the stressor remains unabated and the body’s autonomic system is no longer able to suppress symptoms.

(ii) In terms of transitory/intermittent stimulus, in populations exposed to stressors of sufficient magnitude as to cause transient changes in cardiovascular function, and the stressor occurs frequently enough to prevent a return to resting cardiovascular status,
then habituation to the stressor may result and manifest as sustained elevated blood pressure after the stimulus is removed.

(iii) Individuals in situations with little or no control over exposure to the stressor may be more prone to permanent health effects.

(iv) Interaction effects may occur between the stressor, hostility toward it and controllability of exposure to it. Interaction effects are key to the schema, since physiological responses can be minimised if exposure to, or hostility and anxiety toward, the stressor can be minimised.

The general scheme for stress and its hypothesised effect on BP is shown in Figure 1.1. Figure 1.2 summarises the complex of biological pathways for non-auditory response mechanisms to noise stimulus.

**Figure 1.1.** Schema for pathways between childhood stress and raised blood pressure and hypertension in adulthood. Adapted from Cresanta et al [1980].
Figure 1.2 Major response mechanisms of the body and their interconnections with the central and autonomic nervous-glandular systems. Source: Kryter [1994]
1.5.3.1 Cardiovascular effects and noise exposure

The bulk of noise and cardiovascular effect studies suggest that if persistent or long-term cardiovascular effects exist due to noise exposure then these are probably weak [Berglund et al, 1995; Babisch et al, 1999]. The extant laboratory and population evidence remains suggestive, but most of the study designs have not been strong enough to produce compelling evidence for long-term or permanent cardiovascular effects in humans. It should be pointed out, however, that if there were large long-term cardiovascular effects due to noise exposure then these would have manifested even in study designs incapable of establishing causation, and consequently there would have been more urgency (and funding) to examine the relationship using study designs capable of establishing causation. For example, cross-sectional community or occupational health studies would have picked up any sizeable chronic cardiovascular effects associated with noise exposure, attempted to eliminate these by controlling for known confounders, and if the effects remained then this would have provided compelling evidence to research funding bodies to resource stronger studies (eg, prospective cohort studies). Large cohort studies of populations or occupation groups, for example, would have been undertaken and relationships established. Usually if an effect size is large enough it will be discovered by accident, perhaps in an unrelated study with a different purpose, then quantified with the appropriate subsequent study design. Also, a large BP effect size associated with noise exposure would have been replicated, but to date no studies of aircraft noise and blood pressure have produced the same or similar results.

Certainly acute cardiovascular effects from noise exposure have been established in numerous laboratory studies. These effects include peripheral vasoconstriction or vasodilation, as well as elevations in blood pressure.

Laboratory & epidemiological studies of cardiovascular reactions to noise

In general, studies of cardiovascular reactions to noise have found acute or transient reactions to noise stimulus but none have produced conclusive evidence of permanent residual cardiovascular effects in humans after cessation of the stimulus.
Animal studies

The main drawback of animal studies is that animal biological systems, particularly those relating to processing and interpretation of noise in higher brain centres, are not the same or, arguably, as developed as in humans so that while findings from animal studies may be suggestive, their generalisability to humans is not.

The bulk of the animal-study literature on physiological responses to noise stems from the 1970s on and is mostly with regard to rats, although some primate noise studies have been conducted. In a study of monkeys exposed to 85-90 dBA industrial noise for up to 9 months, Peterson et al found that the exposed monkeys experienced changes in diurnal heart rate patterns and in blood pressure, compared to a control group of unexposed monkeys. These changes persisted for a month following exposure cessation [Peterson et al, 1984a].

Animal studies suggest that there may be permanent BP changes resulting from exposure to noise, but the mechanisms for this are not clear and animals may not have the cognitive mechanisms for suppressing the sound beyond those in the olivocochlear bundle, the chief physical mechanism for protecting the inner ear from noise overexposure via aural and cochlear inhibitory reflex [Kryter, 1994].

Exaggerated BP responses have been invoked in spontaneously hypertensive rats using 30 seconds of noise stimulation, with smaller BP rises in renal hypertensive and normotensive rat controls [Hallbäck et al, 1974]. Correspondingly, the noise threshold necessary to generate a BP effect was found to be lower in the spontaneously hypertensive rats, and the BP effect took longer to subside [Hallbäck et al, 1974]. Whether there are BP effects due to long term exposure to noise is in some dispute. For example, no BP effects were found in rats exposed to broadband noise for 10 hours per day all their lives [Borg et al, 1978]. On the other hand, Rhesus monkeys exposed to long-term industrial noise responded with elevated blood pressure which lasted for at least one month after the cessation of noise exposure [Peterson et al, 1981].

Another study, by Fisher et al [Fisher et al, 1991], attempted to establish a physiological mechanism for elevated BP found in borderline hypertensive rats during 10 weeks of exposure to recorded aircraft noise. This study was suggestive of the mechanism related
to changes in the structure of blood vessels rather than changes in baroreceptor sensitivity or autonomic influences. The investigators made this conclusion from three key experimental findings:

(i) no statistically significant difference was found between exposed and control rats in brachycardiac responses to graded doses of phenylephrine (which has the effect of disabling the baroreceptor mechanism), so that BP differences in exposed versus non-exposed rats could not be attributed to changes in baroreceptor sensitivity.

(ii) no significant difference in autonomic (mainly sympathetic) influences on BP were found after ganglion blockade by chlorisondamine, so that BP differences between the exposed and non-exposed could not be attributed to heightened sensitivity to epinephrine and other sympathetic stimuli.

(iii) diastolic BP in the exposed rats was higher than the unexposed rats (62 mmHg versus 49 mmHg) after maximum vasodilation was induced with hydralazine, although this difference was only borderline statistically significant (p = 0.08).

Wu et al [Wu et al, 1992] produced similar findings in rats and concluded similarly that “enhanced response to vasoconstrictors and the attenuation to endothelium-dependent vasodilators may account for elevations in blood pressure during noise stress. This indicates that the elevation in blood pressure by noise stress may be partly due to the deterioration of endothelial function”. These findings were also reproduced in another rat study of BP and exposure to aircraft noise in conjunction with the effects of elevations in dietary sodium [Tucker et al, 1993].

Primate studies of BP effects of noise exposure suggest that the BP elevations persist beyond the noise exposure. Peterson et al [Peterson et al, 1984b] found in Macaque monkeys exposed to 97 days of 4 and 8 hours-per-day exposures to recorded industrial noise that heart rates responded immediately to noise exposure commencements (heart rate rise) and cessations (heart rate decrease), while elevated mean arterial pressure levels persisted after exposure cessation.

An avenue which animal studies could explore would be to attempt to separate cognitive and learned mediation of noise reaction from direct physiologic reactions to noise.
exposure, by using a classic Pavlovian approach. For example, in addition to the usual control and exposed groups, 2 further exposure conditions could be investigated. These exposure groups would comprise one set of subjects being pre-conditioned to expect an unpleasant experience when the noise exposure occurs (eg, electric shock) and another group pre-conditioned to receive a reward (eg, food) or some other pleasant experience with exposure to the noise. The extent of BP differences between the 4 different arms would then be an indication of the extent of influence of cognitive and learned reactions as effect modifiers. In particular, it would be instructive if there is a learned, cognitively-mediated BP reaction to noise exposure that is differentially associated with pre-conditioning to unpleasant versus pleasant experiences [Bersh et al, 1986]. The animal mechanism underlying this possibility would shed light on possible human cognitive mediating mechanisms for BP reactions to noise exposure, especially between noise-sensitive individuals, individuals annoyed by noise versus individuals not sensitive to or easily annoyed by noise.

In summary, animal studies appear to suggest that structural changes to vascular systems are associated with noise exposure and elevated BP, but not so much via changes in baroreceptor sensitivity or autonomic/sympathetic influences. However, understandably, the animal studies also have concentrated largely on physiology and not on mechanisms of cognitively mediated BP reactions associated with noise exposure.

**Human studies -- laboratory based**

Laboratory studies have contributed enormously to the understanding of hearing and underlying mechanisms of response to noise. Moreover, much of the development of the Perceived Noise Decibel (PNdB), Effective Perceived Noise Decibel (EPNdB) and EPNL, the bases of the NEF method of rating community response to aircraft noise, were defined and established from judgements made by experimental subjects in the laboratory. Laboratory studies have provided the insights and foundations for the underlying mechanisms for hearing and health effects found in human and animal populations.

However, laboratory studies of human blood pressure responses to noise are limited by: (i) the realness of the noise exposure, particularly its loudness, duration, regularity or whether ambient noise sources are present; (ii) the realness of options available for the subject to minimise their exposure to the noise (iii) the transient nature of the
cardiovascular response; (iv) the realness of the context of the noise; and (v) ethical limits on the extent of experimentation on human subjects, particularly regarding possible permanent effects.

(i) There is particularly wide variation in the duration and sound pressure levels of noise across different experimental settings to which subjects are exposed. Exposure durations range from seconds to hours, while sound pressure levels can vary from 65 dB to 95dB or higher. Consequently, it is difficult to assess whether a dose-response relationship established under one set of duration-loudness combinations is equivalent to another occurring under a different combination of duration and loudness. Noise experiments based on a single noise source, such as domestic jet aircraft noise, are not so circumscribed as such noise events are limited in their frequency and duration.

(ii) Experimentally evoked cardiovascular responses to recorded noise in the laboratory setting often are produced in subjects who are unable to alter their exposure to the sound. Even if the noise exposure were a true reflection of exposure in naturalistic settings the opportunity for a subject to lessen their exposure is limited by the need for each experimental noise exposure to be kept constant so that accurate dose-response relationships between different sound pressure levels and cardiovascular reactions can be established. As a consequence, it is probable that cardiovascular effect sizes found in the laboratory are higher than in naturalistic settings due to lack of subject control over the exposure.

(iii) Acute cardiovascular responses to noise are readily evoked in the laboratory, but the extent of longer lasting effects on the cardiovascular system, either as a result of the acute cardiovascular reaction or of other mechanisms, largely remains outside the purview of the investigator.

(iv) The context of noise exposure is a significant effect modifier of cardiovascular response to noise. This well-established phenomenon has underpinned a plethora of laboratory studies of cardiovascular responses to noise exposure in subjects who perform specific cognitive or other tasks while exposed to the noise.

(v) Ethical issues limit the extent to which subjects can be exposed to noise experimentally. On the one hand, if permanent cardiovascular effects were found from
laboratory experimentation then limits to the amount of exposure to noise *vis-a-vis* cardiovascular effects may well be determined, possibly at the expense of damage to other organs including hearing.

Human laboratory-based studies have produced equivocal cardiovascular responses under exposure to noise. For example, Etholm *et al* found no effects on cardiac output, heart rate, stroke volume or pulmonary artery pressure in subjects exposed to 29 minutes of 90 dB white noise [Etholm *et al*, 1964 (cited in Berglund *et al*, 1995)]. On the other hand, Klein *et al* found the pulse rate of the internal carotid artery to increase in some but decrease in other subjects when exposed to 92-96 dB noise for 10 seconds [Klein *et al*, 1969 (cited in Berglund *et al*, 1995)]. Ising found similar results in a sample of hospital patients experimentally exposed to traffic noise (65 dB LAeq) for 12 hours: some patients showed increased BP, others decreased BP, compared to when they were not exposed to the noise [Ising, 1983 (cited in Berglund *et al*, 1995)]. A similar study by Ising [1983] of policemen exposed to traffic noise for several hours per day produced analogous results to the hospital study.

These findings suggest either that there is not a strong association between noise exposure and BP since they are explicable simply by stochastic variation in BP; or, at most, that changes in the BP of some susceptible individuals is associated with noise exposure.

*Human studies -- occupational health*

Epidemiological studies of BP and occupational noise exposure have shown similarly equivocal results to the laboratory studies. A study by Kent *et al* of US Air force flight crews whose noise exposure was measured by the extent of high tone hearing loss, found no association between blood pressure and the hearing loss [Kent *et al*, 1986]. On the other hand, a study by Talbott *et al* found an association between hearing loss and diastolic BP exceeding 90 mmHg, in a study of 245 retired metal workers occupationally exposed to noise for 30 years or more [Talbott *et al*, 1990]. The association was statistically significant (*p* ≤ 0.05) only in the older retirees aged 64-68 years but not the younger group aged 56-63 years.

A larger study, by Nowak, examined the BPs of 2,599 workers occupationally exposed
to 90 dB or more of intermittent noise and compared these to 2,454 control workers not so exposed. Pre-work and at-work BPs were measured. Factors found to be significantly associated with the at-work BP measurements were the initial pre-work BP, age, duration of employment and exposure to noise. The strength of association of noise exposure with BP was found to be strongest with length of employment in the noise-exposed group and in borderline hypertensives [Nowak, 1996].

A later study by Talbott et al compared 329 male workers in a noisy manufacturing plant (noise levels ≥ 89 dBA) with 314 male workers in a quieter plant (noise levels ≤ 83 dBA), all with 15 or more years seniority, and found mean systolic BP in the noisy plant to be 123.3 mmHg compared to 120.8 mmHg in the quiet plant, marginally statistically significant (p = 0.06); and a statistically significantly higher diastolic BP in the noisy plant of 80.3 mmHg versus 77.8 mmHg in the quiet plant (p = 0.014). An association between cumulative noise exposure and BP was found in the noisy plant but not in the quiet plant [Talbott et al, 1999].

In summary, occupational studies of noise exposure and blood pressure are confounded by many factors, not the least of which are the usual risk factors for elevated blood pressure and exposure measurement, and, particularly in North American studies, differences in proportions of African American workers between exposure groups.

**Human epidemiological studies: ecological evidence -- hospital admission studies**

In 1969 an ecological retrospective study of populations living in boroughs surrounding London's Heathrow Airport reported significantly higher rates of admissions to the Springfield psychiatric hospital among the noise-exposed population than those living in low aircraft noise exposed areas [Abey-Wickrama et al, 1969]. Total and first-time admissions over a two year period (mid-1966 to mid-1968) were examined. Admission rates for all groups taken together, all females (both total and first time admissions), all females over 45 years of age (both total and first time), and all widows (both total and first time admissions) were significantly higher in the noise-exposed population than in the low noise region. Observed admission rates were consistently higher than expected values for the high noise areas, even if statistical significance was not reached in some strata. Potential confounders -- for example, ease of access to mental health care facilities, or differences in age structure and socioeconomic status of the populations being compared -- were not adjusted for.
The above admissions study was repeated for the years 1970 to 1972 [Gattoni et al, 1973]. After adjustments were made between the high exposure and low exposure populations for age, sex and marital status, the findings of the earlier study were not replicated.

A further study of psychiatric hospital admissions around Heathrow airport by Jenkins et al, after partial control of several measures of socioeconomic status, failed to find a consistent relationship between noise exposure and admission rate by hospital - there was a possible trend for higher admission rates with noise exposure at two hospitals, but a negative trend for the remaining hospital [Jenkins et al, 1981].

In an attempt to resolve the apparent contradiction between the negative dose-response relationship in admission rates by hospital, Kryter re-analysed the Jenkins et al data and showed that immigrant status in the study population was strongly and negatively correlated with psychiatric hospital admissions; that the proportion of immigrants in one hospital catchment area was significantly higher; and the distribution of immigrants skewed more toward the higher noise contours for that hospital’s catchment area than in the catchment areas for the other two hospitals. According to Kryter, the negative dose-response result of Jenkins et al was thus an artefact because insufficient allowance was taken for immigrant status of the population for that particular hospital [Kryter, 1990].

Among residents near Los Angeles international airport, admissions to mental hospitals from a high noise area were found to be 29% higher than in low noise controls (significant at the 10% level) [Meecham et al, 1977]. Potential effects of a number of confounders were not allowed for -- African-Americans made up 25% of the exposed area, but only 2% of the control area, for example.

The reworking by Kryter of the London psychiatric hospital admission data illustrates the importance of accounting for cultural differences in population sub-groups regarding mental illness and how mental health services are used. The Meecham et al study may have been affected by differential psychiatric admission rates between African-Americans and non African-Americans, quite possibly biasing their results toward the null if psychiatric admission rates in African-Americans are lower than non African-Americans.
In summary, there appears to be some evidence suggesting that aircraft noise may be positively associated with psychiatric hospital admissions but the evidence is ecological.

**Human studies: survey evidence -- psychological surveys**

If aircraft noise contributes to mental illness, it is probable that differences in non-institutional psychological or psychiatric morbidity would be evident.

The community survey of 6,000 people from four noise exposure zones living near Heathrow Airport (see above also) produced variable results [Tarnopolsky et al, 1969]: significantly higher prevalence of recent onset of night waking, depression, irritability (along with swollen ankles, minor accidents including burns and cuts, and skin problems) occurred in those from higher noise zones (based on energy-averaged noise contours). Significantly lower prevalence of chronic irritability was noted in lower noise zones. However, psychototropic drug intake was found to have a significant negative trend in relation to noise exposure [Watkins et al, 1981], inconsistent with a noise-psychiatric illness/symptomology hypothesis. These findings failed to replicate results of the prior pilot study by Tarnopolsky et al, where psychiatric measures showed an association with noise only in more highly educated respondents [Tarnopolsky et al, 1978].

In a postal follow-up survey of annoyance and noise sensitivity of a subsample of 77 women from the same survey, repeated measures of levels of annoyance were found to be more highly correlated in those sensitive to aircraft noise \( r = 0.85 \) than in those sensitive to road traffic or other noise \( r = 0.51 \). Sensitivity to noise was not a significant predictor of psychological morbidity (as measured by the 30-item General Health Questionnaire [GHQ]) [Stansfeld, 1992].

In the Netherlands, Knipschild found significantly higher General Practitioner-patient contact rates for psychological problems, ‘mental disorders’ (consistent across degrees of severity), and some ‘psychosomatic complaints’ (spastic colon and lower back pain) in the 2 noisiest of 4 exposure zones around Schiphol airport (near Amsterdam) [Knipschild, 1977]. However, some of the differences in contact rates could have been explained by differences in socio-economic status between exposure zones rather than the exposure itself since socio-economic status was not controlled for.
In 552 residents surveyed from five different noise zones (including a quiet control zone) around San Francisco Airport, significant correlations were found between noise awareness and annoyance, and the number of health problems reported from a symptoms checklist [Graeven, 1974]. Fear of an aircraft crash explained most of the variance in the quiet control zone, while in the noise-exposed areas most of the variance was explained by noise awareness and annoyance. Kryter reports an ecological study of people living near airports in the former Soviet Union finding a higher incidence of ‘nervous diseases’, among other conditions [Karogodina et al, 1969 (cited in Kryter, 1994)].

Evidence for an association between psychological symptoms and aircraft noise exposure is based mostly on self-report survey data which appears to show such associations to be contingent on noise sensitivity and annoyance in respondents. Health service contact studies give more objective evidence of higher symptom rates in noise-exposed areas but some of this may be due to differences in socio-economic status between exposed and unexposed populations. Community-based mental health and psychological studies of aircraft noise, while not central to the topic of this thesis, are summarised in Table 1.2.
<table>
<thead>
<tr>
<th>Authors/Location</th>
<th>Health Effect</th>
<th>Noise Measure</th>
<th>Study type</th>
<th>Confounding Factors</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abey-Wickrama <em>et al</em> (1969) London/Heathrow, UK</td>
<td>Admission to mental hospital</td>
<td>&gt;55 NNI or PNdB&gt;100</td>
<td>Ecological</td>
<td>Sex specific. No control for age or SES factors</td>
<td>Significantly higher admission rates in exposed population</td>
</tr>
<tr>
<td>Gattoni and Tarnopolsky (1973) London/Heathrow, UK</td>
<td>Admission to mental hospital</td>
<td>&gt;50 NNI &lt;50 NNI</td>
<td>Ecological (replication of Abey-Wickrama (1969))</td>
<td>Age-standardisation sex/marital specific rates. No control for SES</td>
<td>Non-significant positive result</td>
</tr>
<tr>
<td>Meecham &amp; Smith (1977) Los Angeles airport, USA</td>
<td>Admission to mental hospital</td>
<td>&gt;90 dB &lt;90 dB</td>
<td>Ecological</td>
<td>No adjustment for race</td>
<td>29% higher in high noise area (p&lt;0.1)</td>
</tr>
<tr>
<td>Tarnopolsky <em>et al</em> (1978) London/Heathrow, UK</td>
<td>Annoyance, possible/confirmed psychiatric cases</td>
<td>&gt;55 NNI =35 NNI</td>
<td>Cross-sectional population survey</td>
<td>Sub-group analyses including by sex, educational level</td>
<td>Correlation between annoyance and psych symptoms. No significant noise effect except in high education group</td>
</tr>
<tr>
<td>Jenkins <em>et al</em> (1979) London/Heathrow, UK</td>
<td>Admissions to mental hospital</td>
<td>25-34 35-44 45-54 55+ NNI</td>
<td>Ecological</td>
<td>Age-standardised, sex/marital specific</td>
<td>Non-significant result</td>
</tr>
<tr>
<td>Tarnopolsky (1980) London/Heathrow, UK</td>
<td>Psychiatric illness other acute/chronic symptoms</td>
<td>&lt;35 35-44 45-54 55+ NNI</td>
<td>Cross-sectional population survey</td>
<td>Age/sex standardised, SES taken into account</td>
<td>Some acute symptom increase with noise no obvious threshold</td>
</tr>
<tr>
<td>Jenkins <em>et al</em> (1981) London/Heathrow, UK</td>
<td>Admissions to mental hospital</td>
<td>25-34 35-44 45-54 55+ NNI</td>
<td>Ecological</td>
<td>Age standardised sex/marital specific control of measures of SES included</td>
<td>Mixed result: 2 hospitals positive, 1 hospital negative</td>
</tr>
<tr>
<td>Authors/Location</td>
<td>Health Effect</td>
<td>Noise Measure</td>
<td>Study type</td>
<td>Confounding Factors</td>
<td>Findings</td>
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<tr>
<td>Watkins et al. (1981) London/Heathrow, UK</td>
<td>Use of medications and health services</td>
<td>&lt;35 35-44 45-54 55+ NNI</td>
<td>Cross-sectional population survey</td>
<td>Age, sex, SES</td>
<td>No significant higher uptake of medications in high noise areas. Higher psychotropic drug use and GP utilisation with annoyance. Variable response of health service use with noise exposure.</td>
</tr>
<tr>
<td>Kryter (1990) London/Heathrow, UK</td>
<td>Admissions to mental hospital</td>
<td>25-34 35-44 45-54 55+ NNI</td>
<td>Ecological (Re-analysis of Jenkins' [1981] data)</td>
<td>Migrant status, SES, age, sex</td>
<td>Significant results: = 40% higher admission rates in higher aircraft noise exposed groups</td>
</tr>
<tr>
<td>Stansfeld (1992) London/Heathrow, UK</td>
<td>Annoyance/sensitivity and psychological morbidity</td>
<td>&lt;45 &gt;45 NNI</td>
<td>Longitudinal population study (postal follow-up of subsample of females from Tarnopolsky sample)</td>
<td>Sex, migrant status</td>
<td>Noise sensitivity not a predictor of psychological morbidity. Noise annoyance in noise sensitive subjects significantly higher in high aircraft noise zones</td>
</tr>
</tbody>
</table>

dB = Decibels  
NNI = Noise Number Index  
PNdB = Perceived Noise Decibels  
SES = socioeconomic status  
Ecological = group-based geographic study
1.6 Aircraft noise and child BP

The main naturalistic studies of associations between child BP and exposure to aircraft noise have come from studies of school children proximate to Los Angeles Airport, by Cohen *et al* [1980, 1981], and of children proximate to the new Munich International Airport, by Evans *et al* [1998]. The Cohen *et al* study was a cohort design with baseline and follow-up one year later. The Cohen *et al* study is the most widely quoted and is examined in some detail below. The Evans *et al* study was a cohort study also but occurred under changing exposure conditions as a natural experiment with the construction of a new airport in Munich.

1.6.1 Los Angeles Airport child BP study

Cohen *et al* reported an (unadjusted) mean excess of 2.91 mmHg systolic and 2.68 mmHg diastolic BP in schoolchildren from elementary schools exposed to high levels of aircraft noise around Los Angeles Airport, compared with children in control schools not exposed to the noise [Cohen *et al*, 1980]. This study also found that the BP difference was greatest between children whose period of exposure to the noise was shortest, as measured by the period of attendance at the school, suggestive of apparent adaptation effects. The study sample comprised 262 Grade 3 and 4 schoolchildren, with 142 subjects from 4 schools exposed to aircraft noise and 120 from 3 quiet schools. Control schools were matched to exposed schools on grade level, ethnic and racial distribution of the children, education levels and occupations of parents, and on the proportion of children whose families received child assistance welfare payments.

Despite the effort in matching exposed and control schools on the above criteria, the noisy schools had a significantly higher proportion of African-American children (32% versus 18%) and a significantly lower proportion of Hispanic children (33% versus 50%). The proportions of white children were similar. Other significant differences between the noisy and control schools included shorter mean period of residency (41.4 months versus 49.6 months) and a shorter mean period of attending the particular school (43.2 versus 36 months) in the noisy schools. The above differences were controlled for statistically in regression analyses of BP and aircraft noise exposure.

Noise exposure in the Los Angeles Airport study was determined by peak noise level
measurements, with the mean peak level in the noisy schools determined at 74 dB, compared to 56 dB in the quiet schools. Noise exposure at home in children from the noisy schools was approximated from noise contour maps compiled by Los Angeles International Airport.

In addition to the overall BP excess found in children from noisy schools, children from noisy schools who lived in quiet homes were found to have marginally significantly higher systolic BP (p < 0.06) and significantly higher diastolic BP (p < 0.02), compared to children from the quiet schools. The magnitudes of these BP differences were not reported, however. The Cohen et al study is considered further in the discussion section.

In the longitudinal follow-up to the Los Angeles Airport study [Cohen et al, 1981], the same children had their BPs re-measured 1 year later (1979). The cross-sectional BP differences between children in the noisy versus quiet schools found at baseline were not reproduced in the follow-up study. Cohen et al attributed this negative finding to sample attrition in children with higher BPs in the noisy schools since it was children with higher BP from the noisy schools who were lost to follow-up [Cohen et al, 1981]. Cohen et al speculated on the possible processes for the sample attrition as:

“(a) parents of children with elevated blood pressure were sensitive to their children’s experience of stress and as a consequence moved to a less noisy neighborhood; (b) because of a familial bias (either genetic or environmentally determined), parents of children with noise-induced blood pressure elevations experienced similar stress-related reactions that motivated them to move from the neighborhood; (c) the children’s elevated blood pressures were a response not to the noise itself but to their parents own noise-induced stress, which was motivating the parents to move from the neighborhood; and (d) some unknown third factor is related to mobility, high blood pressure, and living in a noisy neighborhood.”

The authors’ conclusion, “that selective attrition, not adaptation, is responsible for the decrease of the difference between the blood pressure of noisy-school and quiet-school children” [emphasis in original], is consistent with the initial cross-sectional findings of decreasing BP differences between noisy and quiet schools with the number of years children were enrolled at the school. This is because if sample attrition were occurring due to any or some of the reasons listed above then this would also have been occurring before the initial study was commenced and would therefore have produced the decrease
in BP differences found with increasing years of attendance, since individuals purportedly less affected by the noise tend to remain in the noise-exposed schools. From an aircraft noise-child BP perspective, the evidence for apparent ‘adaptation’ of child BP to noise is most probably an artefact of sample attrition. To estimate the extent of BP ‘adaptation’ to aircraft noise exposure adequately it would be necessary to follow the same individuals over time and under changing exposure conditions.

These results and further aspects of the follow-up study of Cohen et al are discussed further in the discussion chapter.

1.6.2 The Munich Airport Study (1998)

With the opening of a new airport in a rural area 35 km outside Munich, BPs and hormonal levels of school children were measured at 3 separate times at affected schools and compared with similar measurements of children from otherwise unaffected rural schools matched on socio-economic status. The first measurements were taken 6 months prior to the opening of the new airport (‘wave 1’), the second 6 months after the airport’s opening (‘wave 2’) and the third measurements 18 months following the opening (‘wave 3’) [Evans et al, 1998]. At each measurement, 217 Year 3 and 4 children participated in the study. Attrition bias was minimal, with 10 and 14 children lost to follow-up in the noisy and quiet schools respectively. Evans et al found that the attrition was unrelated to the BP outcome variable, unlike the Los Angeles study. That is, there was not a higher likelihood of sample attrition if BP was initially higher.

The main BP findings of the Munich study were: (i) lower mean BPs were found in the children who subsequently became exposed to the noise compared to the unexposed children; (ii) mean BPs in the newly exposed children approached the higher BP levels of the unexposed children with time following the opening of the new airport. At baseline, 6 months prior to the airport’s opening, the mean systolic BP in control children not subsequently exposed to the noise from the new airport was 100.8 mmHg, compared to 97.2 mmHg in children who became exposed to noise from the new airport. Corresponding mean diastolic BPs were 60.5 mmHg versus 62.6 mmHg. Six months after the opening of the airport, the mean systolic BP in children exposed to the noise was 101.6 mmHg, versus 102.2 mmHg in the unexposed children. Corresponding mean diastolic BPs were 63.2 mmHg versus 63.6 mmHg. One year after the airport’s opening,
the mean systolic BP in the noise exposed children was 102.4 mmHg, compared to 102.6 mmHg in the unexposed children. Corresponding mean diastolic BPs were 64.4 mmHg versus 64.8 mmHg. That is, BP in the noise-exposed children increased significantly more than in the children from the quiet areas.

While the Munich results are not directly comparable with those from the Los Angeles study, they do raise a number of issues since they were related to changes in exposure, not merely cross-sectional differences. The first issue is the sensitivity of initial samples to differences in BP. That is, cross-sectional BP differences between groups under existing noise conditions may be due to underlying BP differences in those populations unrelated to noise exposure, as the Munich study showed in the pre-exposure BP (Wave 1) measurements. Accordingly, in studies without measurements of BP in subjects prior to them becoming exposed to noise it is always possible that BP differences between noise exposure groups are due to other factors (since the only BP measures of comparison groups are cross-sectional, after exposure to the noise). In this regard, it is possible that the results of Cohen et al (1980) are due, at least in part, to initial or underlying (unrecorded) BP differences between the comparison groups.

The second issue raised by the Munich study is that the exposed group at each measurement stage continued to show lower mean BPs than the unexposed group. That is, if the reduced BP differences with time can be entirely attributed to the time-related change in noise exposure, then the magnitude of the effect of the noise exposure on BP is small. The Evans et al study will be considered further in relation to results from the Sydney study in the discussion chapter.

1.7 Conclusions

Despite the establishment of strong theoretical frameworks for examining the effects and underlying biological mechanisms for exposure to noise and cardiovascular effects, the lack of laboratory evidence for non-acute long-lasting or permanent cardiovascular effects from noise exposure suggests that if such associations between BP and noise exposure are found in the field in naturalistic settings, then these are most probably due to confounding factors. Thus far, no epidemiological study of child BP and aircraft noise exposure has replicated the results of another. As only two child studies specifically examining BP and aircraft noise exposure have been conducted this is understandable.
Certainly some acute cardiovascular effects due to noise exposure have been found in laboratory settings, but not all these have been unequivocal or consistent. The least inconsistent field-based evidence for BP effects from noise exposure has come from task-related studies taking into account personality type and coping styles. But the effects found generally have been acute and reversible once exposure to the combination of task and stressor has ceased.

The situation of children exposed to aircraft noise in the school setting, can be viewed as a naturalistic replication of task-related studies in some ways, since children are doing classroom tasks for much of the time as part of their learning. Years of exposure to aircraft noise in the classroom setting under aircraft noise exposure conceivably would reproduce permanent cardiovascular effects if these were found in the laboratory. The field evidence for permanent BP effects is not strong and this appears to reflect the laboratory evidence. However, the weak field evidence is based to an extent on weak study designs, with little control of confounding and effect modification factors of BP and with crude exposure measurements. This situation creates uncertainty as to whether there is a link between aircraft noise exposure and child BP, perhaps a weak one, while evidence based on stronger study designs would at least point toward evidence of weak or no child BP effects associated with exposure to aircraft noise.