Physiological Measurement

Portfolio submitted to the
Institute of Physics and Engineering in Medicine

in partial fulfilment for the award of
Clinical Science Diploma

by

Graham Webb

Department of Medical Physics and Bioengineering
St George's Hospital
London

October 2006 – March 2007

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SUMMARY

The placement was carried out with the Department of Medical Physics and Bioengineering of St George's Hospital, which is part of the St George's Healthcare NHS Trust. The training was carried out over a 22-week period during 26th October – 16th March 2007, under the supervision of Fred Mitchell (Principal Clinical Scientist and Training Coordinator).

I attended monthly seminars in the Department and a Medical Devices Study Day to gain a wider view of medical device training in the hospital, during which I received training from Nutricia Clinical Care. I also attended the 6th Annual Education Symposium for Clinical and Biomedical Engineers at the Barbican London, which covered a broad range of physiological measurement subjects.

This portfolio documents my experiences in the following areas:

- Audiology[*†]
- Lung Function[*†]
- Neurology[*†]
- Neonatal Intensive Care[†]
- Urodynamics[†]
- Vascular Lab.[†]

[*] Experienced as part of this Physiological Measurement placement.
[†] Experienced as an acquaintanceship.

List of Competencies

The following tables list the competencies set out by IPEM and the suggested chapter and section headings that go towards demonstrating competence in those areas.

Table 1 Evidence of Specific professional competency.

SPECIFIC Competencies		Where Demonstrated
Use of Equipment and Clinical Applications		
PM 1.1	demonstrate a broad understanding of the principles of common physiological measurement procedures, the physiological processes involved and the significance of the measurements, covering examples from (i) electrophysiological signals and processing, (ii) pressure measurements, and (iii) flow measurements;	Throughout
PM 1.2	demonstrate a detailed understanding of the principles, physiological processes and measurement significance in three examples of physiological measurement covering, where possible, very different physiological processes and/or clinical areas;	Sections 1.3, 2.3, 3.2 and 4.2
PM 1.3	identify the measurement of one parameter from each of the three examples of physiological measurement chosen and be able to:	
PM 1.3.1	identify and justify the choice of equipment, including the placement / selection of the electrodes/transducer;	Sections 1.3, 2.3, 3.2 and 4.2
PM 1.3.2	use the equipment to obtain clinical measurements from patients;	Sections 1.5, 1.6, 2.6, 3.4
PM 1.3.3	apply signal conditioning techniques, including methods to optimise the signal, improving signal to noise ratios and the role of filtering;	Sections 1.3, 1.5, 3.2, 3.3, 4.2
PM 1.3.4	identify artefacts and be aware of sources of error;	Sections 1.3.4, 2.35, 2.4.5, 3.2.4, 3.3.5, 4.4
PM 1.3.5	undertake calibration procedures (applicable, where appropriate, to both stimulus and measurement equipment);	Sections 1.3.2, 2.3.3, 2.4.3, 2.5.4, 4.4
PM 1.3.6	apply appropriate statistical methods to draw conclusions as to the normality/abnormality of the data and/or the significance of any changes detected in response to stimulation or other intervention.	Sections 1.5, 1.6, 2.6, 3.4
Quality Assurance and Safety		
PM 2.1	identify the risks associated with the use of the equipment and the precautions to be taken with it (this must cover all three of the chosen physiological measurements);	Sections 1.7, 2.7, 3.3.5, 4.3 and Chapter 5
PM 2.2	perform measurements to ensure that the equipment used complies with all relevant safety standards (this must cover at least one of the chosen physiological measurements);	Section 5.7
PM 2.3	demonstrate implementation of current safety standards for the safe use of the measurement system in its clinical setting (this must cover at least one of the chosen physiological measurements);	Throughout
PM 2.4	carry out a formal risk assessment of a clinical procedure, device or software (this must cover at least one of the chosen physiological measurements).	Section 5.7

1. AUDIOLOGY

1.1 Introduction

The Physiological Measurement placement began with six weeks in the Audiology department of St George's Hospital. During my time in the clinic I conducted some hearing and vestibular measurements and carried out practical work. I witnessed a range of psycho-acoustic and neuro-otological tests. This chapter describes the experiences gained in clinic and through that project work. The measurement types cover electrophysiological signals arising from electroencephalographic (EEG) and electronystagmographic (ENG). I observed motion being assessed in the Vestibular Testing Laboratory.

1.2 Background

Mechanical pressure waves enter the ear through the pinna, travel through the external auditory meatus and strike the tympanic membrane, causing it to vibrate.

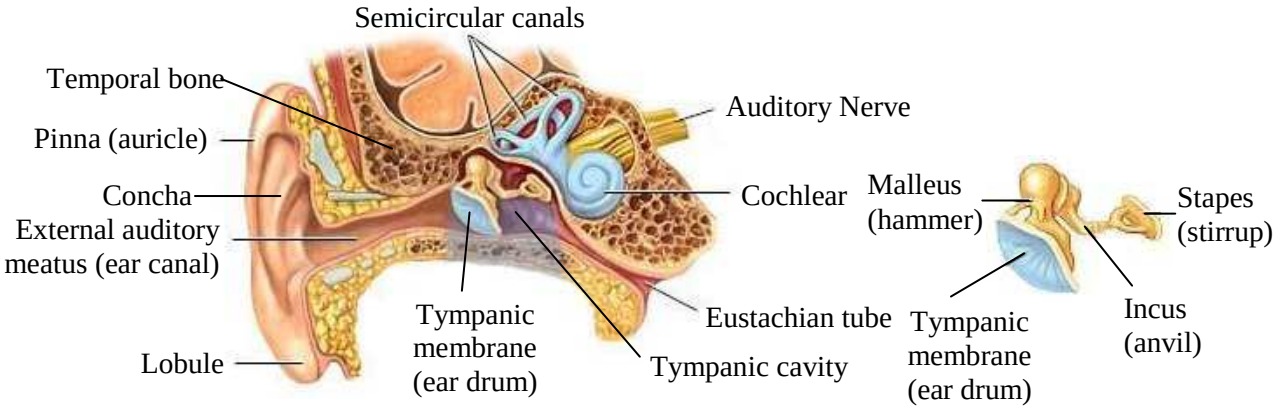


Figure 1 Anatomy of Ear detailing the ossicles [1].

The middle ear transduces energy from these weak vibrations using the mechanical advantage of the ossicles. The ossicles are the smallest and first fully formed bones in the human body, comprising: the malleus, incus and stapes [2]. Two mechanisms protect the middle ear from some damage caused by prolonged exposure to high amplitude sounds: 1) air is equalised via the Eustachian tube and 2) the damping action on the stapes by the tensor tympani and stapedius muscles, in a feedback process known as the acoustic reflex [3].

The inner ear houses the transducers for balance (semicircular canals and vestibule) and hearing (cochlear). The cochlear contains three fluid-filled channels separated from each other by the tectorial and basilar membranes. Vibration of the oval wall is transmitted through these channels causing the basilar and tectorial membranes to vibrate. The organ of Corti sits between the tectorial and basilar membranes with rows of two types of cell, inner and outer hair cells. Each hair cell supports bundles of stereocillia which protrude into the tectorial membrane and synapse with a complex network of afferent and efferent neurons. The differential vibration between the basilar and tectorial membranes causes the stereocillia to move and create an action potential.

The central auditory system is defined by the auditory structures medial to the cochlear nerve. Afferent nerve cell bodies are found in spiral ganglia within the cochlear. The fibres make up the VIII cranial nerve and travel through the temporal bone to synapse with the ventral and dorsal regions of the cochlear nucleus. These regions lie within the medulla of the brainstem. The afferent pathway ascends to the superior olivary cortex within the medulla, the location of efferent nerve cell bodies and the origin of the motor pathway of the middle ear muscles. The afferent pathway continues superiorly through the lateral lemniscus, a structure that covers the superior olivary cortex, pons and the inferior colliculus of the midbrain. Afferent signals then conduct to the medial geniculate body, before reaching the primary auditory cortex, located within the superior temporal gyrus of the cerebral cortex.

Hearing loss or deafness affects about 8.7 million people in the UK. A third of the population will have significant hearing loss by the age of retirement. A loss of the ability to discriminate different sounds is attributed to sensorineural loss (inner ear and/or nerve pathology), whereas a loss of perceived loudness (loss in sensitivity) can be attributed to sensorineural or conductive hearing loss (outer and/or middle ear pathology). Conductive hearing loss may result from obstruction to the outer ear. Viral or bacterial inflammation causes attenuation in the outer or middle ear. Traumatic damage to the ear may cause perforation of the membrane. Otosclerosis results in increased impedance of the stapes. Cholesteatoma is the growth of skin in the middle ear, may follow otitis media and membrane perforation and lead to erosion of the ossicles. Sensorineural hearing loss results from loss or absence of inner hair cells and may result from biological or chemical assault. Noise-induced hearing loss is the prolonged exposure to high amplitude sounds resulting in permanently flexing inner hair cells. Multiple sclerosis destroys the myelin sheath of the vestibulocochlear nerve. Tumours can also grow on the vestibulocochlear nerve in neurofibromatosis and restrict the development and growth of nerve cells.

Over fifty tests are in use throughout the UK to assess hearing, tinnitus and balance disorders [4]. The following is a summary of the major tests employed within the department at St George's Hospital.

Pure tone audiometry (PTA): PTA is the gold standard subjective test to assess hearing threshold, recording the patient's response to a range of air and bone conducted audio stimulus, with varying frequency (Hz) and intensity (dB SPL). PTA is a subjective measure relying on the patient's ability to decide if a stimulus is audible and to respond accordingly. False negatives are a concern for cases involving insurance claims. The audiologist must also consider false positives due to patient generated sounds, such as tinnitus. There are no single or double blind controls in PTA [5].

Tympanometry (or middle ear impedance audiometry): Tympanometry is a middle ear function test that measures membrane compliance as a function of static pressure. This varies with effusion, ossicle trauma, otosclerosis, or perforation. Membrane perforations and otitis media will produce results that could mask secondary conditions involving the ossicles [6].

Acoustic reflex (or stapedius reflex): The stapedius muscles contracts, stiffening the stapes when a stimulus above approximately 80dB is presented [6]. The acoustic

reflex test provides an objective measure of this action by assessing acoustic impedance changes. The amplitude, latency and timing of the response can be recorded using a tympanometer. Absence of the reflex or activation at lower intensities could indicate sensorineural loss or dysfunction.

Preventative healthcare in audiology today places an increased emphasis on early detection of hearing defects. This is facilitated under the NHS Newborn Hearing Screening Programme (NHSP). Along with the unconscious and uncooperative patients, neonates often fall into the category of hard-to-measure patients. The following tests are useful in these situations:

Visual reinforcement audiometry (VRA): An enhancement to PTA for neonatal and paediatric threshold assessment, where patient feedback is indicated by a conditioned response. A visual re-enforcement is used to train the patient. This is a fascinating modern day application of Pavlov's famous experiments [5]. Low VRA thresholds correlating with abnormal tympanograms or absent oto-acoustic emissions indicate a hearing loss. An acoustically calibrated room is used with testers on either side of a one way mirror. Speakers present free field tones. Visual re-enforcement boxes containing toys are hidden and controlled by the PTA tester. PTA hardware is used. The baby is conditioned to respond to the audio stimulus. Tester 1 (or parent) distracts the baby. Tester 2 conducts a PTA out of sight, observing responses by the direction of head turns towards the visual rewards. This is a subjective psycho-acoustic test used within a false (trained) psychological framework. The test can lead to both false positives and false negatives.

Oto-acoustic emissions (OAEs): Outer hair cells generate a sound which can be evoked and picked up with a microphone. These are oto-acoustic emissions. The precise mechanism of the emissions is unknown, but their presence or absence has been qualitatively documented in different pathologies, making them clinically useful. They can benefit diagnosis of: auditory neuropathy, vestibular schwannoma, ototoxic drug action and noise induced hearing loss. OAEs are used in neonatal screening [7].

Vestibular Testing: Although the majority of examinations in the department are audiological, there is a vestibular testing laboratory that provides the following tests:

1. Eye gaze (using video-nystagmography or electro-nystagmography)
2. Eye tracking or smooth pursuit (using VNG or ENG)
3. Saccadic eye motion (using VNG or ENG)
4. Rotary chair
5. Caloric

The semi-circular canals provide feedback information to the vestibuloocular reflex (VOR). VOR maintains visual acuity and a stable visual environment during motion. Nystagmus can be found in patients with a non-functioning VOR. Nystagmus are rhythmic involuntary oscillations of the eyes with clearly defined fast and slow phases, referred to as right-beating and left-beating. The objective quantification of nystagmus is the role of the vestibular laboratory. Each test is a variation on a theme of recording the patient's eye motion before, during and after a defined patient task.

Auditory Evoked Potentials (AEPs): Auditory evoked potentials are bio-potentials with amplitude of 1-10 μV, which originate from different anatomical structures along the central auditory pathway (Figure 2) [8]. Different AEPs are elicited by the application of different external stimuli and present peaks with characteristic amplitudes and onset latencies following the stimulus. The full range of potentials is shown in Figure 3. The brainstem and late cortical responses are used in St George's Hospital Audiology department and will be discussed in more detail.

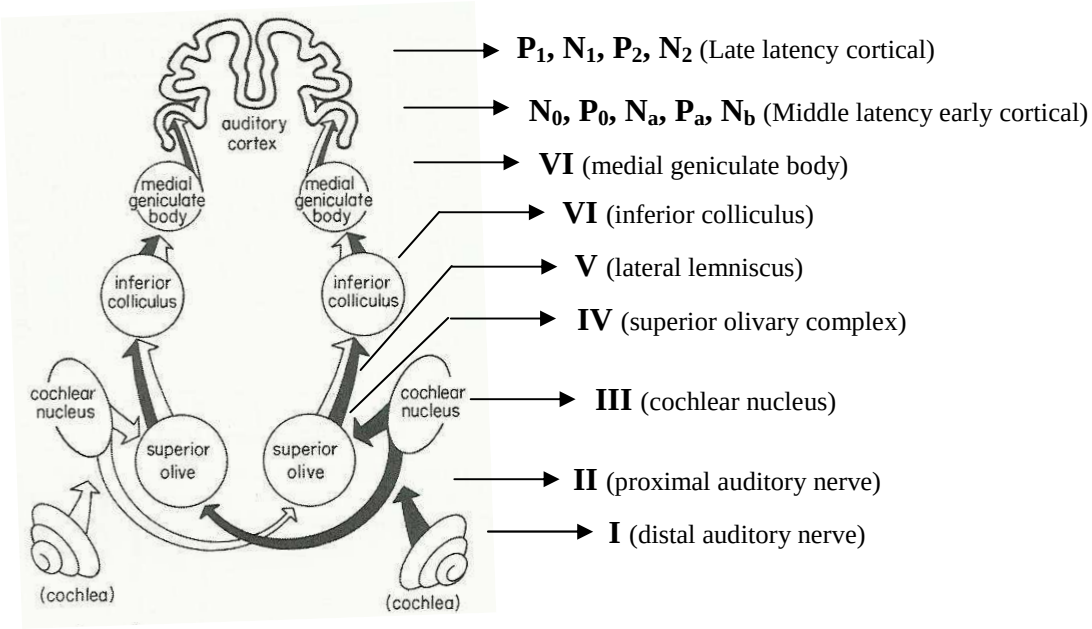


Figure 2 Auditory evoked response landmarks

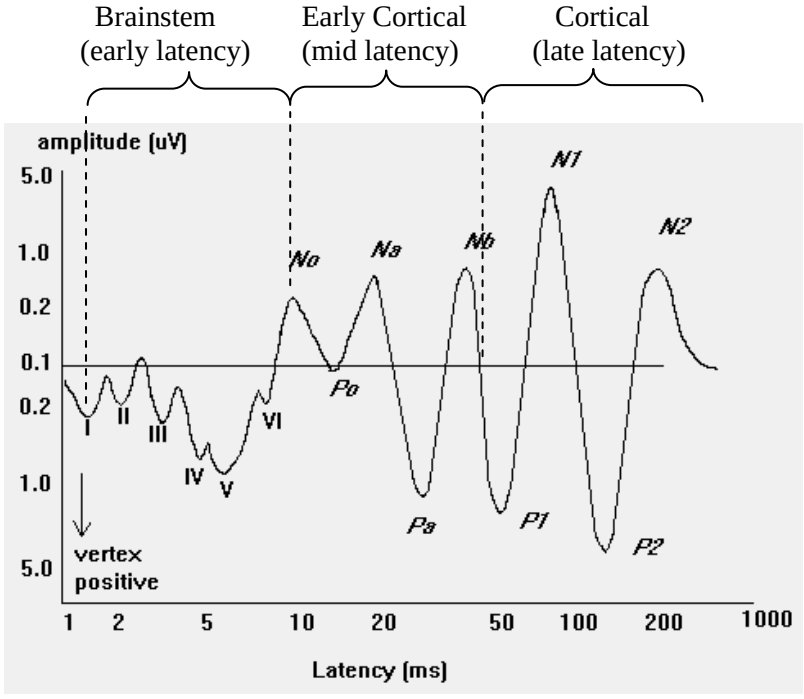


Figure 3 Full range of normal auditory evoked response

The Roman numeral labelling convention in use today was developed by the pioneers of the AEP test, Jewett and Williston. 'N' and 'P' labels refer to positive and negative peaks. Some interesting responses are labelled according to the time of appearance following stimulus onset, for example: P3, P300 or N400. The late cortical response N400 is a cognitive one elicited by the presentation of a semantic incongruity, such as the phrase "I am going to take my house for a walk". Given this specificity and the small scale of the signals amid the total background EEG, this begs the question – what other signals are available for clinical use that have not been identified?

1.3 Auditory Brainstem Response (ABR)

The Auditory Brainstem response is an early latency response originating from the brainstem and is the most widely used for clinical assessment. The signal is picked up with skin surface electrodes and the peak and inter-peak relative and absolute latencies are compared to normative reference values. The reference database has been built up from an ongoing longitudinal study in the department, based on an 80dBHTL stimulus, and is summarised in a lookup table (Table 2). It is used clinically to screen within ± 2 standard deviations. See section 1.5 for my work on a normal ABR database.

Table 2 Normative values for peak and inter-peak ABR latencies

Wave peak	Ipsi-lateral recording latency (ms)	Contra-lateral recording latency (ms)
I	1.28-1.73	N/A
III	3.40-4.16	3.40-4.12
V	5.24-6.01	5.38-6.03
I-III	1.90-2.58	N/A
III-V	1.53-2.17	1.68-2.28
I-V	3.70-4.49	N/A

Note: Contra-lateral peaks do not emerge from sites I or II because these structures (proximal and distal auditory nucleus) are not directly stimulated. Hence there are no inter-peak latencies. The signal crosses at the Superior Olivary complex (III), so late contra-lateral signals exist.

The ABR has three major applications:

- ***Identification of retrocochlear pathology:*** If outer and middle ear tests eliminate the possibility of conduction loss, the presence or absence of a brainstem response is used to review retrocochlear and sensorineural pathologies. A CN VIII lesion will show an absence of characteristic wave peaks. A tumour (acoustic neuroma or vestibular schwannoma) compressing CN VIII will slow down conduction velocity producing a higher inter-peak latency.
- ***Newborn hearing screening:*** Since a cognitive response is not required, the ABR is used in the NHS newborn hearing screening program. For the same reason it also finds use with uncooperative and unconscious patients.
- ***Interoperative monitoring***: ABR is used interoperatively (as is electrocochleography), to monitor brainstem and cochlear integrity. Ischemia is a primary cause of surgery-related hearing loss, wave I is monitored to provide the surgical team with information regarding blood flow to the cochlear.

1.3.1 ABR Measuring equipment

Principle of operation: Auditory evoked signals are small (1-10µV) amid the surrounding bioelectric signals: EEG (5-300µV), EOG (50-3500µV) and EMG (100-5000µV). The signal appears for only a brief duration (a few ms). Two processing techniques are employed to capture it: 1) The stimulus generator is time locked with the ABR signal sampler as shown below. This effectively acts as a trigger input for the detection hardware and aids signal detection temporally. 2) The signal is amplified with a gain of approximately 30000 and band limited. Following amplification, a number of sweeps are made to generate data for a signal average to be taken. Aperiodic noise is mathematically thrown away and periodic AEPs remain.

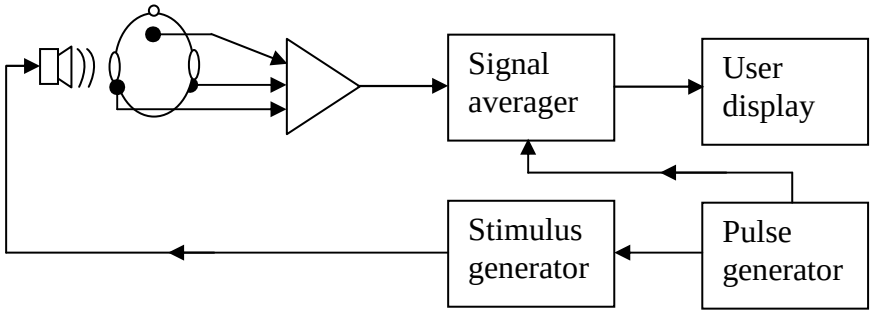


Figure 4 Schematic of ABR equipment

The Audiology department at St George's Hospital use a portable Biologic Navigator Pro system and laptop in clinic, for work in the theatres and surgical assessment and recovery wards. The unit presents a 100µs duration click stimulus to each ear drum through an ear insert, or to both cochlear through the mastoid bone, with a bone vibrator. In the time domain this is the closest representation of a delta dirac function, it has an infinite spectra in the frequency domain. To stimulate specific areas of the basilar membrane, the frequency bandwidth is narrowed using filtering, masking or a narrow band tone burst. Parameters of concern for the tone burst wave shape are the number of cycles during rise time, plateau and fall time. A typical stimulus would be of the format 2-2-2 µs. The Biologic Navigator Pro is capable of two channel recording. The connections are as follows:

Single Channel Recording: for a left ear ipsilateral test, A_2 is common and the stimulus is picked up between F_{pz} and A_1. The stimulus is given to the right ear for a contralateral test. For a right ear ipsilateral test A_1 is common and F_{pz}-A_2 carries the potential.

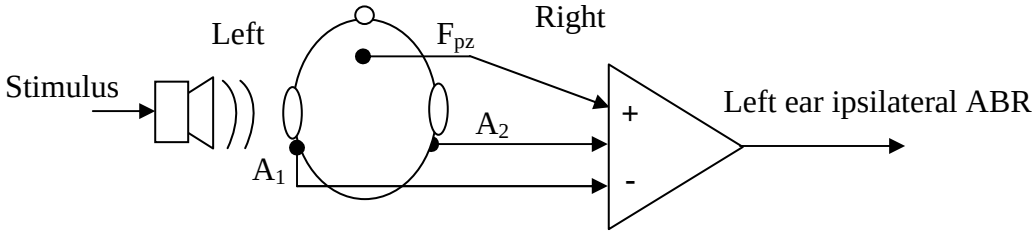


Figure 5 single channel ABR hardware setup

Dual Channel Recording: Both A_1-C_Z and A_2-C_Z are measured with respect to the high forehead electrode F_{PZ}, as follows. The stimulus is transferred between ears for ipsi- or contra-lateral recordings:

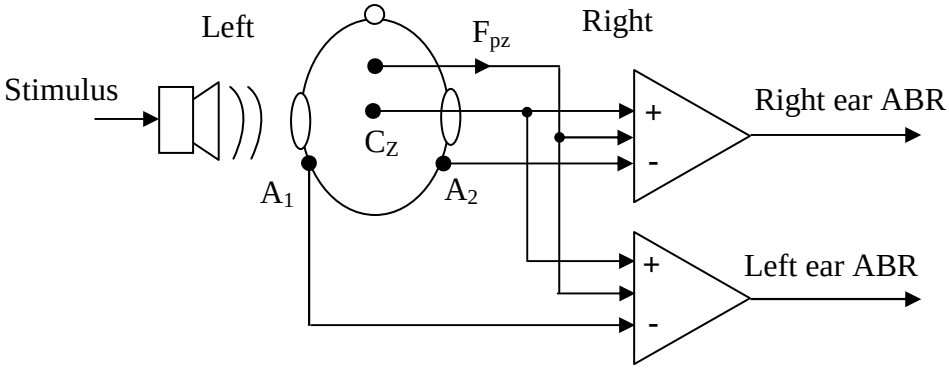


Figure 6 Amplifier – electrode configurations

Electrodes: The electrode positions conform to the internationally recognised 10-20 system, shown below. A combination of electrodes pairs is called a *montage*. Three types of surface electrode are used in the Audiology Department to collect ABR depending on the contact surface: Small self-adhesive jelly button electrodes are used for neonates, slightly larger and stickier 'tiptrode' electrodes are used for adults and Ag/AgCl cup electrodes are used for hairy surfaces such as the high forehead and vertex recording sites, as required. This electrode type is held in place with a piece of tape. Care is taken when placing the electrodes over the fontanel of neonates. All of the electrodes and leads are disposable. A minimal voltage variation at the skin-surface contact, across the electrode montage, is required to minimise the variation in induced noise. To impedance match the montage, the skin is prepared with a slight abrasion with clinical emery paper and the application of a saline or aluminium oxide solution called *nuprep*. A conductive adhesion gel called *collodion* is used for the vertex cup electrode.

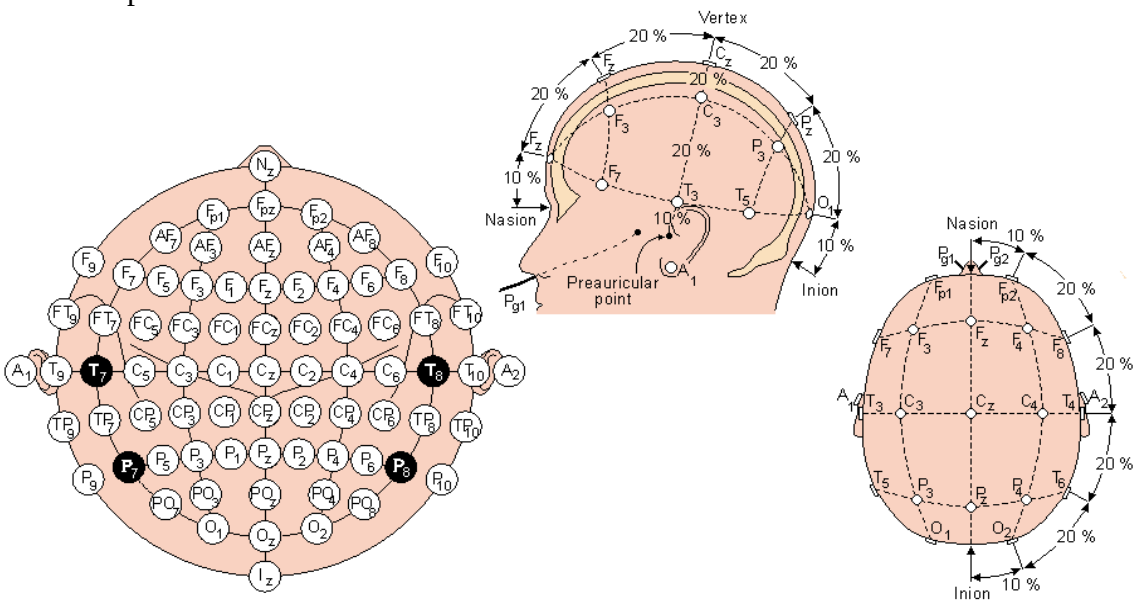
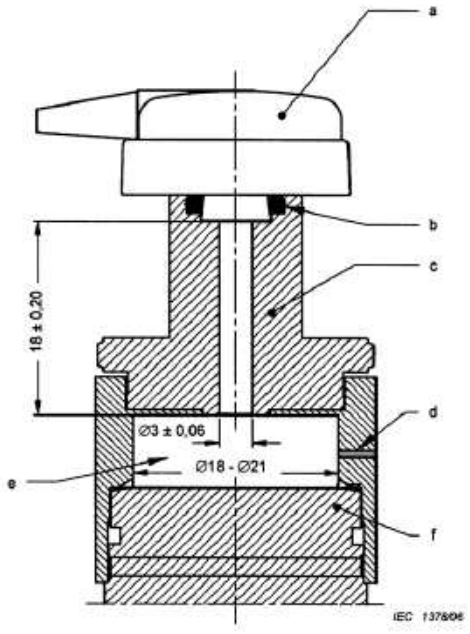


Figure 7 10-20 electrode placement system

1.3.2 ABR Equipment calibration

The click stimulus used to evoke the ABR is calibrated annually according to BS-EN-ISO 389-1:2000. The calibration is required to determine the absolute sound pressure level (SPL) of a particular stimulus emitted through a set of headphones. The calibration also ensures the intensity levels of the required stimulus and emitted sounds are comparable. The relationship between absolute SPL in an acoustic coupler and output from different headphones is variable. For a given earphone, in specific conditions, the SPL in response to a calibrated stimulus is defined as the reference equivalent threshold sound pressure level (RETSPL). The calibrated stimulus is determined from measurements of a statistically significant group of otologically normal volunteers. The peak-to-peak equivalent sound pressure level of insert earphones and headphones is measured as follows:

1. The sound tube of the insert earphone is connected to an insert ear simulator and held in place by an acoustic coupling device, according to BS-EN-ISO 389-2:1997. Headphones are connected to a calibrated audiometer and connected to an acoustic coupler (Figure 8). Acoustic leakage is prevented through the use of MX41/AR cushions and a loaded spring holds the headphone in place with a nominal force of 4.5 ± 0.5N.
2. According to EN ISO 389-1:2000, the equivalent threshold at 0 dBHL is 7dBSPL, so a 1kHz pure tone signal at a level of 70dBHL is applied to the earphone at 77dBSPL (70dB + 7dBSPL).
3. The output is recorded by a microphone and displayed using an oscilloscope. The peak to peak amplitude is measured and adjusted accordingly.
4. Using the Navigator Pro system, a 100µs click stimulus is then applied at a SPL of 70dB pk-pk.
5. The pk-pk sound pressure level output and pulse amplitude is measured and adjusted to provide the desired equivalent to the 1kHz pure tone sound pressure level.



Key

a	Insert earphone
b	Airtight seal
c	Ear mould substitute for insert earphone
d	Static pressure equalization
e	Coupler cavity of 2000 mm^3 ± 70 mm^3 effective volume
f	Pressure response microphone

Figure 8 Acoustic coupler for ear insert

The click stimulus is calibrated in a similar manner with the application of a 1 kHz 70 dB sinewave. IEC 60318 states that for a click stimulus applied to an acoustic coupler, the RETSPL peak-to-peak equivalent is 31dB SPL. This value has been determined from average objective measurements conducted at a number of centres. So the click is compared to 103 dB peak SPL (70 + 31 dBSPL). At St George's Hospital, a 1.5dB tolerance is permitted between the expected and measured values.

1.3.3 ABR Test protocol

1. The equipment is mobile and in use throughout the hospital, so it must be set up and checked before use.
2. Patient details (name, hospital number and date of birth) are entered into the database.
3. The patient is seated and the test procedure explained to them, the outer ears are checked with an otoscope.
4. Using an abrasive pad or water based gel, the electrode attachment sites are prepared. The area should be dry before electrodes are placed and care must be taken to ensure the patient feels no discomfort.
5. Electrode impedance should be checked twice; firstly before attaching the vertex electrode and then again after attachment of this electrode. The impedance should be less than 6kΩ and the difference in resistance values between the electrodes should be less than 2kΩ. If values exceed these limits the electrodes should be removed and the skin preparation repeated before re-checking the impedance.
6. Insert earphones are deformed and inserted into the ear canals; once inserted they will relax back to their original shape to ensure a good seal is achieved. The colour coding system is: red wire to the right ear and the blue wire to the left ear.
7. The electrode leads are plugged into the amplifier box according to the configuration in use (refer to section 1.3.3). Once all of the electrodes are attached and the impedance is satisfactory, the test can commence.
8. The incoming EEG waveform is observed from the vertex recording, which gives an indication of the state of relaxation of the patient, this is monitored throughout. The test continues with an 80dBHTL signal and 40dBHTL contralateral mask.
9. Each measurement is repeated twice to ensure repeatability. If the traces are unclear or unrepeatable, the electrode impedances and the relaxed state of the patient is checked. Once all the tests are completed and verfied by the operator, the electrodes are removed, electrode sites are wiped and the patient is free to go.
10. The waveform features are then labelled and the absolute latency of each wave noted along with the amplitude of waves I and V. The data is then compared with the normative reference data, this deviation is highlighted in the report sent to the consultant.

The patient must not have discharging ears or abnormalities to the external ear that would cause discomfort or pain as a result of the test. The patient must have a developed CNS, understand the instructions and be able to sit still for up to one hour. To reduce myogenic artefacts, the patient is instructed to relax and remain as still as possible. The test is conducted in a soundproof booth to reduce external noise influence. The room is darkened to aid relaxation.

1.3.4 Artifacts and sources of error

The following situations may occur to cause false measurements, generate artefacts or limit data quality and repeatability:

- Discharging ears, immature CNS, patient on medication
- Incorrectly placed electrodes
- Measurement system tampered with by patient or carer
- Hardware fault – component failure or damage, break in leads or connectors
- Filters incorrectly set – signal filtered out.
- Capture window incorrectly set – signal occurring outside frame
- Incorrect stimulus type to elicit a response for that patient group
- Signal masked in myogenic artefacts, if the patient is not relaxed
- Headphones incorrectly placed
- No signal expected, patient incorrectly referred for test, unusual anatomy (path from neural generators to electrodes), subjective analysis of ABR graph

Of particular concern are:

Table 3 Key faults in electrophysiological recording

Fault	Cause	Identification	Correction
Unequal electrode impedances	Poor skin-electrode contact, either through dry and flaky skin, poor adhesion, a bent and faulty electrode or from the electrode part-falling off. This will result in differing mains coupled voltages at the skin-electrode sites, which will be amplified by the differential amplifier.	• This fault can be identified in ECG for example by a baseline drift. • An impedance check will quantify the problem.	• Greater attention to skin site preparation. • Use of a conductive gel. • Stronger electrode adhesion – tape. Use of different electrode tip type.
Signal lost in noise (low signal to noise ratio)	Noise induced in EEG leads and hardware, arising from: nearby electrical equipment, fluorescent lighting, other patient-worn devices, nearby mains circuits, current paths across other people or items in the room such as beds or diathermy equipment as in the case of interoperative monitoring.	• The signal will not appear in the data if the noise floor is too high. • Signals coupled to mains noise are characterised by a modulated data-noise signal.	• Remove sources of noise – switch off items if not in use remove to another room, or ensure good earthing of external equipment. • Reduce electrode lead lengths. Twist electrode leads to introduce voltages 180^0 out of phase with signal. • Segregate lead types (power from data) • Use an amplifier with a higher CMRR or a filter with a tighter bandwidth • Introduce active filtering, cable shielding, equipment isolation.

1.4 PROJECT - Late latency response for neonatal hearing screening

Objective: I was asked to look at how to elicit a late latency cortical response through a hearing aid, for neonatal screening. I was given five days and provided with a Siemens Prisma Pro hearing aid.

Background: A need arose from the newborn hearing screening programme for an objective technique to fit and evaluate hearing aids for young paediatric patients. Since babies are not mature enough to respond to the pure tone audiogram protocol the ABR is currently used to assess the hearing of this patient group. ABR is an early latency response that presents short duration stimuli clicks and tonebursts to the patient; this is not the stimulus type that hearing aids are designed to manage. High amplitude click stimuli artefacts emerge from the microphone electromagnetic pickup during testing. One alternative currently in use is to elicit an ABR response from a free field, however this is subject to the inaccuracies of potentially uncooperative patients moving within the free field. The stimulus required to elicit a late cortical response is longer than the ABR stimulus, thus would reduce unwanted transient effects in the DSP circuitry, improve the signal-to-noise ratio and eliminate the associated free field errors from the test. Note: A typical ABR stimulus is a 100µs click with 49.1 Hz pulse repetition rate. A typical cortical stimulus is a tone burst with a 10 ms rise time, 60 ms plateau and 0.25 – 0.8 Hz pulse repetition rate.

Method: I proposed that during clinical operation, the cortical stimulus be presented to the digital hearing aid through a direct input shoe, and then to the patient through a calibrated ear insert coupled to the hearing aid. To achieve this, a connection is required between the Biologic Navigator Pro stimuli output to the hearing aid. Modern digital hearing aids can be fitted with a direct input shoe (DI shoe), which is a plastic interface that permits direct audio connection from the user's media player or for communications with a contra-lateral hearing aid. I contacted Siemens UK Hearing Aid Division and was directed to a commercial company called called Connevans Ltd, which produce leads for consumers. At the same time I made enquiries with an online hobby group known as "hearing aid hacks", devoted to hearing aid modifications. The DI shoe receives analogue voltage data using two of three pins (Pin 1 DC - 0.9 Volt signal, Pin 2 N/C, Pin 3 Ground, large diameter pin).

The connection out of the NavPro stimulator is a standard PS2 mouse connector with 3 pins in use (stereo signal and ground). I measured the full scale output from the stimulator with a digital oscilloscope and found it to be a DC voltage level varying from 0 – 0.8 volts, which was suitable for a direct connection to the hearing aid, with the inclusion of a 330 kΩ current limiting resistor as suggested by Siemens. I produced a cable for use with two hearing aids (as per the schematic below) and mechanically turned the connector housing to suite the NavPro case.

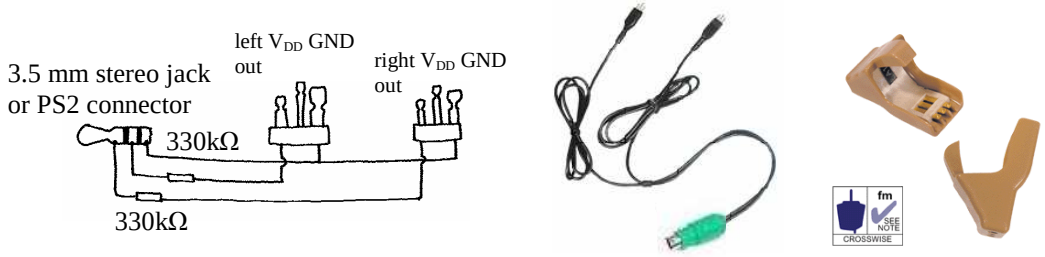


Figure 9 Direct input cable, left to right: wiring diagram, completed cable, direct input shoe

Calibration: Calibration is required to ensure that the stimulus presented to the ear conforms to the standards required by the test protocol being enacted. (For cortical evoked response audiometry stimuli standards applied to neonates see Appendix A).

The block diagram shows the complete setup I suggested for development and calibration. Non-standard connections that required additional work are shown in red and described in further detail. The stimulus is generated by the NavPro and presented through the hearing aid. The electrical output is measured with an oscilloscope and the acoustic output is measured through an artificial ear using a Bruel and Kjaer 2260 investigator sound level meter. The stimulus shape can then be modified with either the NavPro, or by the hearing aid DSP, which can be programmed using an Aurical.

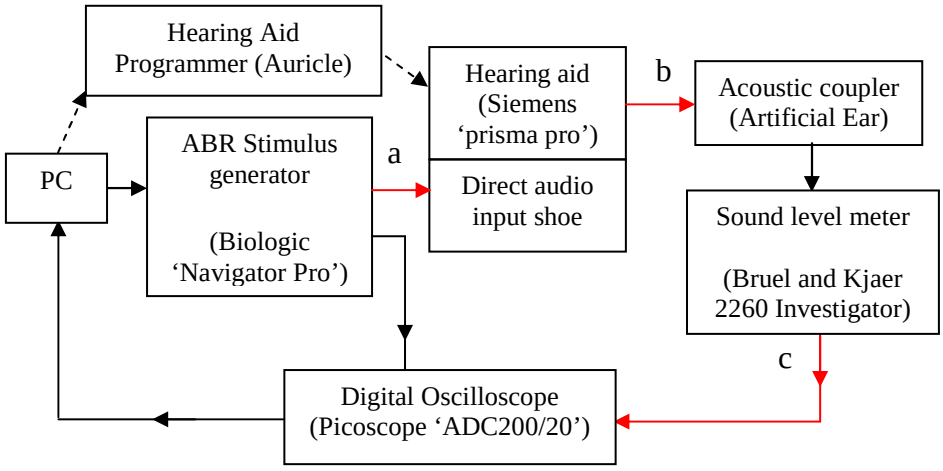
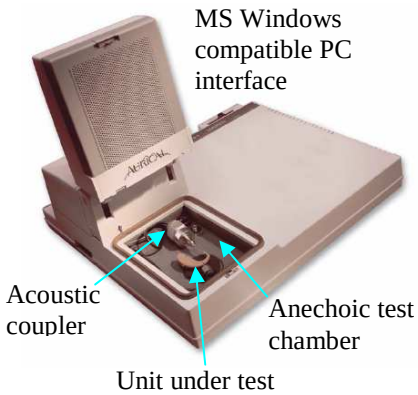


Figure 10 Proposed hardware setup for development and calibration

Hardware connections:

a) Custom direct input cable (described above)
b) A section of non-compliant tube connects the output of the hearing aid to the acoustic coupler
c) I contacted Bruel and Kjaer and proposed three methods to extract data from the sound level meter:
   1. The B&K propriety RS232 interface, which could be used with a terminal emulator such as *hyperterminal* to transfer block data to a PC
   2. An analogue output primarily intended for a DAT tape, which could be read in real-time with an oscilloscope
   3. A custom RS232 interface, which would require writing a program in NI LabVIEW for example, to read data in real-time

The Aurical (pictured right) is a hearing aid programmer, used clinically to adjust the frequency response and hearing aid characteristics to suite the patient. An electrical connection is made to the hearing aid DSP circuitry and the loop is closed with an acoustic coupler at the output. A small anechoic chamber is provided for testing. To ensure a linear response in preparation for calibration, I used an Aurical to flatten the hearing aid frequency response and switch off compression and speech processing algorithms.



Discussion: I presented a method for eliciting a cortical response through a hearing aid and produced a direct input cable. But given the tight time constraints of the project, a number of tasks are required to bring the method into clinical practice. These include completing the calibration, carrying out a risk assessment and conducting a validation trial of normal subjects.

1.5 PROJECT - Normal ABR Database Update

Objective and Background*:* I was asked to collect auditory brainstem response data from a population of people with no reported hearing problems, in order to update the database currently used by the Audiology Department at Queen Mary's Hospital. An ABR database provides a normal reference with which to compare the patient caseload. The database used at Queen Mary's Hospital was produced in 1993 and reports mean values for the absolute latency of peaks I, III and V, and the interlatencies for I-III, III-V and I-V, for the local population (Table 4).

Table 4 Current normative ABR database

	Absolute peak latency			**Inter-peak latency**		
	I	**III**	**V**	**I-III**	**III-V**	**I-V**
Mean values (ms)	1.59	3.67	5.87	2.22	1.94	4.44

Subjects*:* I recruited 11 adult subjects with no reported hearing problems, in the age range 25 – 49 years, from colleagues within St George's Hospital and Queen Mary's Hospital. See Appendix B for the subject information sheet. The test is an audit of equipment so no ethical approval was required by Wandsworth Primary Care Trust.

Contraindications*:* No history of neurological hearing disorders, no tinnitus, no noises in the ear, symmetrical hearing, no history of ear infections, perforations or middle ear effusion.

Method*:* I produced the following protocol and carried it out with each subject. I was observed by the Senior Audiologist for the first subject tested, in order to ensure the data quality conformed to the existing database.

1. Seat subject and explain test procedure, check state of outer ear with otoscope. Instruct subject to relax and remain as still as possible.

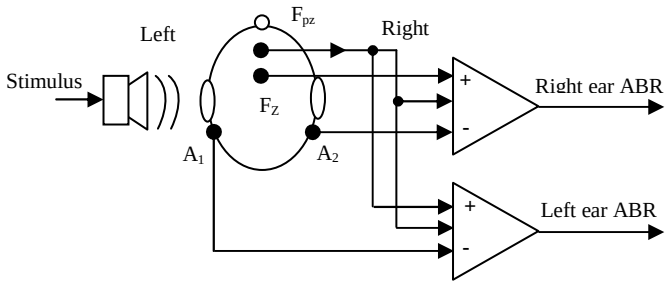
Otoscope safety: An otoscope (pictured right) is a device used to examine the outer ear and tympanic membrane, consisting of a lamp and detachable speculum. It is important to ensure a sterilised speculum is used for each patient ear. Insert slowly, avoid compacting cerumen and contact with the eardrum. Consider the possibility of abnormal canal geometry.

2. Enter subject details (name and date of birth) into the database

3. Clean electrode sites using *nuprep* water based gel and an abrasive pad if required. Allow to dry. Attach *Ambu 720* disposable electrodes to the following

recording sites: Channel 1 F_z – A_1, Channel 2 F_z – A_2, Common ground F_{pz}. F_z was chosen in favour of the vertex site (C_z) to ensure a low impedance across the sample population.

4. Plug electrode leads into the amplifier box accordingly:



5. Check electrode impedance using the Bio-logic AEP software. The impedance should be <5kΩ and the difference in resistance values between the electrodes should be <2kΩ. If values exceed these limits repeat electrode preparation and re-check the impedance
6. Individually place the TDH39A headphones over the pinna of each ear and tighten headband to rest of vertex. With red wire to the right ear and the blue wire to the left ear. Check with the subject that they are sitting comfortably. Re-check electrode impedance
7. Set stimulator and capture parameters:

Stimulator (headphones):	
Auditory stimulus:	Click 100 μs (10 kHz)
Intensity:	80 dB nHL
Polarity:	Alternating
Stimulation rate:	27.70 Hz
Masking:	Contralateral white noise @ 80 dB SPL
Averager:	
Input structure:	Ch1&2, direct
Analysis (epoch) time:	10.66 ms
Stimulus delay:	0%
Artefact rejection:	15.8 μV
Sweep limit:	2048 (more if necessary to enhance trace)
Low filter (high pass):	100 Hz
High filter (low pass):	1.5 kHz
Amplifier Gain:	150,000 (adjust if required to max 300,000)
Display gain:	Adjust as necessary to maximise waveform definition, although all traces should be at same display gain for reasonable comparisons to be made.

8. Commence test. The incoming waveform is observed from the high forehead recording, giving an indication of the state of relaxation of the patient. Observe the number of artefacts. If artefact number increases, stop test and re-check electrode and headphone placement and state of patient. Observe the shape of the averaged signal as it forms to assist in determining the location of the peaks
9. Repeat test a minimum of three times for each ear for repeatability, alternating the ear receiving stimulus
10. Identify waves I, III and V and record the best three trials from each set of repetitions

***Results*:** Figure 11 shows a typical example of four trials of unprocessed ABR data taken from one subject. Note the increase in amplitude at 10 ms represents noise originating from the posterior auricular muscle. See Appendix C for individual subject data.

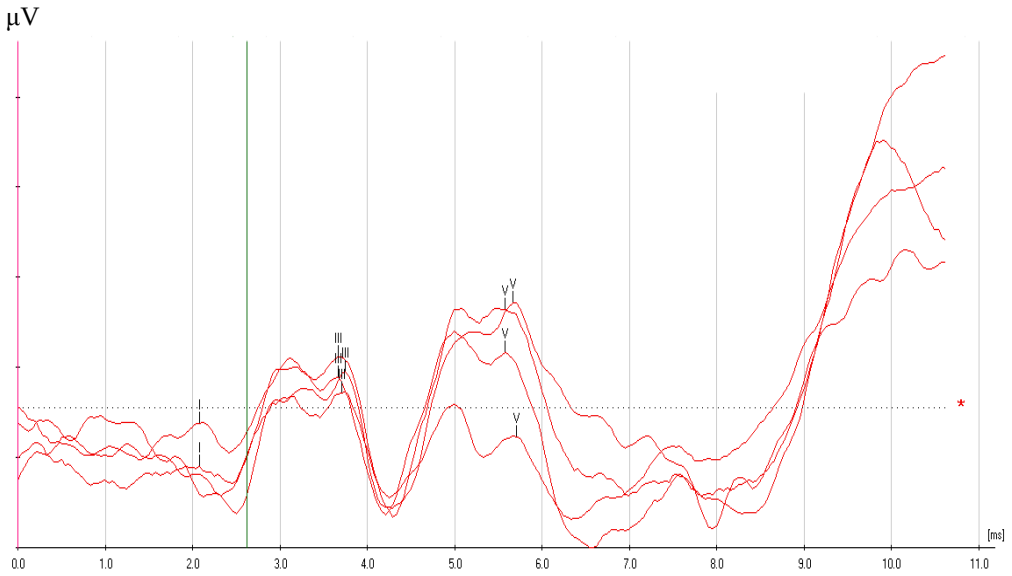


Figure 11 Representative sample of unprocessed data

The following peak and inter-peak latencies were determined from the sample population (Table 5).

Table 5 Summary of group ABR results (n=11, 3 data points of smoothing)

	Absolute Latency			**Inter-peak latency**		
	I	**III**	**V**	**I-III**	**III-V**	**I-V**
Mean	1.36	3.69	5.94	2.38	2.26	4.58
Min	0.17	2.08	5.58	1.17	1.75	3.50
Max	2.33	4.29	6.45	4.00	4.25	6.04
SD	0.67	0.41	0.20	0.78	0.44	0.73

Discussion: In comparison to the existing database, the greatest difference is an increase in peak III-V interlatency of 0.32ms, all other variations are fractions of a millisecond (a decrease of 0.23ms in peak I latency, +0.02 III latency, +0.07ms V latency, +0.16ms I-III interlatency and -0.14 ms in I-V inter-peak latency).

Despite being capable of high resolution data recording, the Biologic NavPro software has a graphical resolution of 0.04 ms, but reports to two decimal places. This manifested as number preference during the reporting process, for example: A sample of peak v latency data appeared as: 5.87, 5.87, 5.91, 5.91. This was problematic when identifying sharp peaks, but not an issue during clinical use where discrete thresholds are observed. I smoothed the data with a moving average algorithm using 3 data points, to see if peak identification with smoother peaks affected the mean latency of peak v. Smoothing reduced the group mean by 0.04 ms, the data are not interpreted to this precision.

1.6 CASE STUDIES

1.6.1 Case Study WM – Acoustic Neuroma (ABR)

WM is a 56 year old female with a history of progressive bilateral hearing loss, tinnitus and vertigo. Pure tone audiometry demonstrated an air-bone gap and loss of 55 – 70 dB throughout the speech range. She was referred for auditory brainstem response testing for assessment of her otoneurological pathway. A_1-F_z, A_2-F_z were assessed using a dual-channel amplifier with the following parameters: 150000 gain, 100 Hz HP filter, 3000 Hz LP filter, 10.24 ms window, 2048 averages with approximately 30% rejected as artefacts.

Results: The following sample is representative of the response obtained at 80 dBnHL (Figure 12 left). Absolute latencies are: 1.98 (wave I), 3.58 (III), 6.32 (V). Interpeak latencies are: 2.14 (I-III), 1.69 (III-V), 4.08 (I-V).

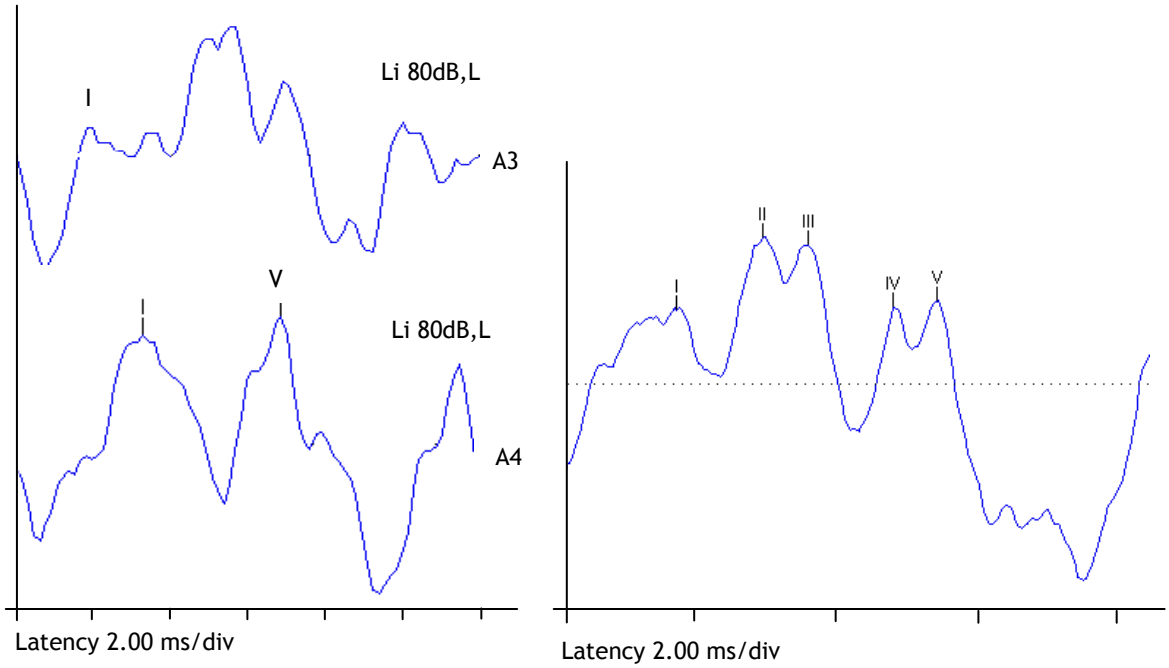
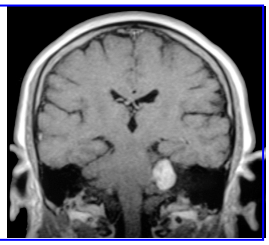


Figure 12 ABR test result (left ear ipsi-lateral). Right: Typical normal ABR waveshape

Conclusion: The data are indicative of an acoustic neuroma. The ABR waveshape lacks the definitive peaks that can be seen in the normal population, a normal response is shown for comparison (taken from the new database). Notably, there is an absence of peaks II-IV bilaterally. Peak V latency is much slower than normal. Queen Mary's Hospital use an absolute peak V latency of 6.12 ms as a threshold for further investigation.

An acoustic neuroma (or vestibular schwannoma, shown as a white mass in the MRI scan) is a benign slow-forming intracranial tumour. They are formed by the schwann cells that line the eighth cranial nerve and compress against the nerve causing vertigo, hearing loss and sometimes pain. Current treatment options include observation, microsurgery and radiotherapy.



1.6.2 Case Study JP – Otosclerosis (adult hearing assessment)

History: JP is a 35 year old female suffering from generally poor hearing levels was referred to the Audiology Department by her GP for hearing tests. The patient's case was listened to by an audiologist. The procedures were explained to her as the process developed.

Pure Tone Audiometry (PTA): A GSI Clinical Audiometer was used with a tone stimulus to test the air-conduction response from both ears. This was followed by a bone–conduction pure tone test. The test started with the better hearing ear (according to the patient) at a frequency of 1000 Hz, followed by 2000, 4000, 8000, 500 and 250 Hz. The first ear was retest at 1000 Hz, if the retest value was more than 5 dB more acute than the original value, the next frequency is retested and so on. The more sensitive threshold is taken as the final value. The opposite ear is then tested in the same order. A poor left ear threshold can be seen from the audiogram (Figure 13). The right ear was then masked with a narrow band noise signal through a headphone, whilst a left ear bone conduction test was repeated. This was done to mask any signal conducted across the skull and isolate the ear under test.

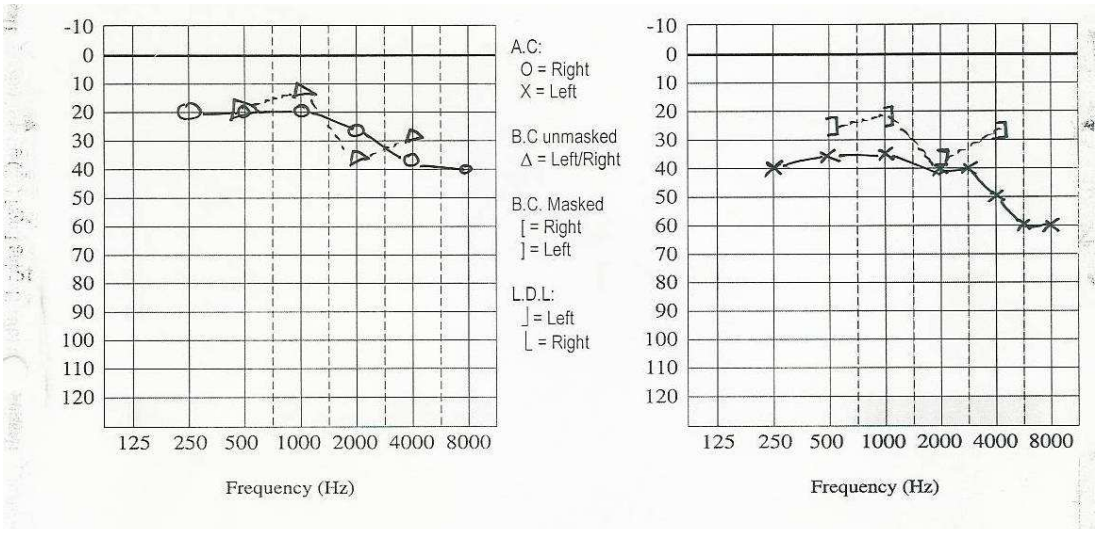


Figure 13 Air conduction (left) and bone conduction audiogram

Tympanometry: A GSI33 Middle ear analyser was used to assess membrane compliance. 46% reduction in compliance can be seen on the LHS compared to RHS, indicating a stiffer mechanism somewhere between along the ossicle chain from the oval window to the tympanic membrane. This is supported by a slight reduction in middle ear pressure.

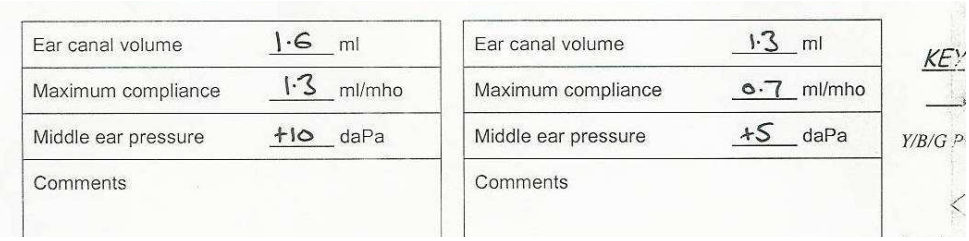
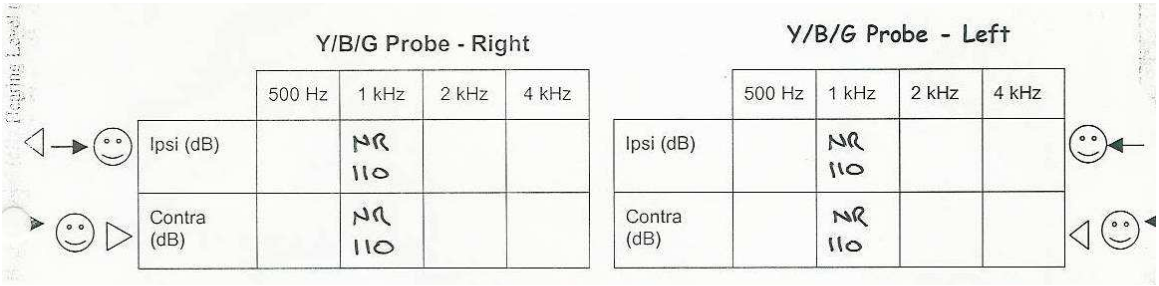


Figure 14 Tympanometry results, right ear (right)

Acoustic reflex thresholds: The acoustic reflex thresholds were measured and found not to be present at the test threshold of 110dB.



Legend: NR - No Response

Figure 15 Acoustic reflex results, right ear (right)

Diagnosis: The notes and reports from all tests were sent with the patient to the Consultant for a diagnosis. A suggested diagnosis is otosclerosis. Otosclerosis is a sclerotic plaque part-fusing the ossicle chain generally between the stapes and the oval window. From the audiogram: a 2kHz characteristic notch present on the bone conduction trace is indicative of a middle ear conduction loss, this is called the Carhart notch [6]. There is also a bone-air gap that suggests a conduction problem. This is supported by the tympanometry results and the absence of an acoustic reflex.

It is likely JP will receive a computed tomography scan to determine the density of bone growth around the oval window. If otosclerosis is confirmed the surgical treatment involves the replacement of the stapes in a stepedectomy. This is a 45 minute procedure were entry to the middle ear is gained from behind the pinna.

Discussion: This case is a good example of how a conclusion can be drawn from complementary test data. Individually the tests would not be as informative. The test results are collected on, and presented to the Audiological Physician as, a single sheet.

1.6.3 Case Study CT – Neonatal hearing screening

A neonatal screening program has been in place at St Georges Hospital for two years now. Newborns are screened on the wards by a technician, using an automated pass-fail handheld ABR device. Any baby who fails this test is invited to the Audiology Department for a more in-depth analysis. The presenting patient failed the test and came to us, accompanied by both parents, six weeks following the initial screening. I assisted an audiologist to measure the baby's oto-acoustic emissions (OAEs) and ABR.

The procedures were explained to both parents. The mother held the baby and encouraged him to sleep. Electrodes were placed on the mastoids (A_1 and A_2) and vertex (C_z). A Bio-Logic Evoked Potential System was used to capture the conditioned data, as shown below. Data for the right ear ($A_2 - C_z$) is not shown because it is almost identical.

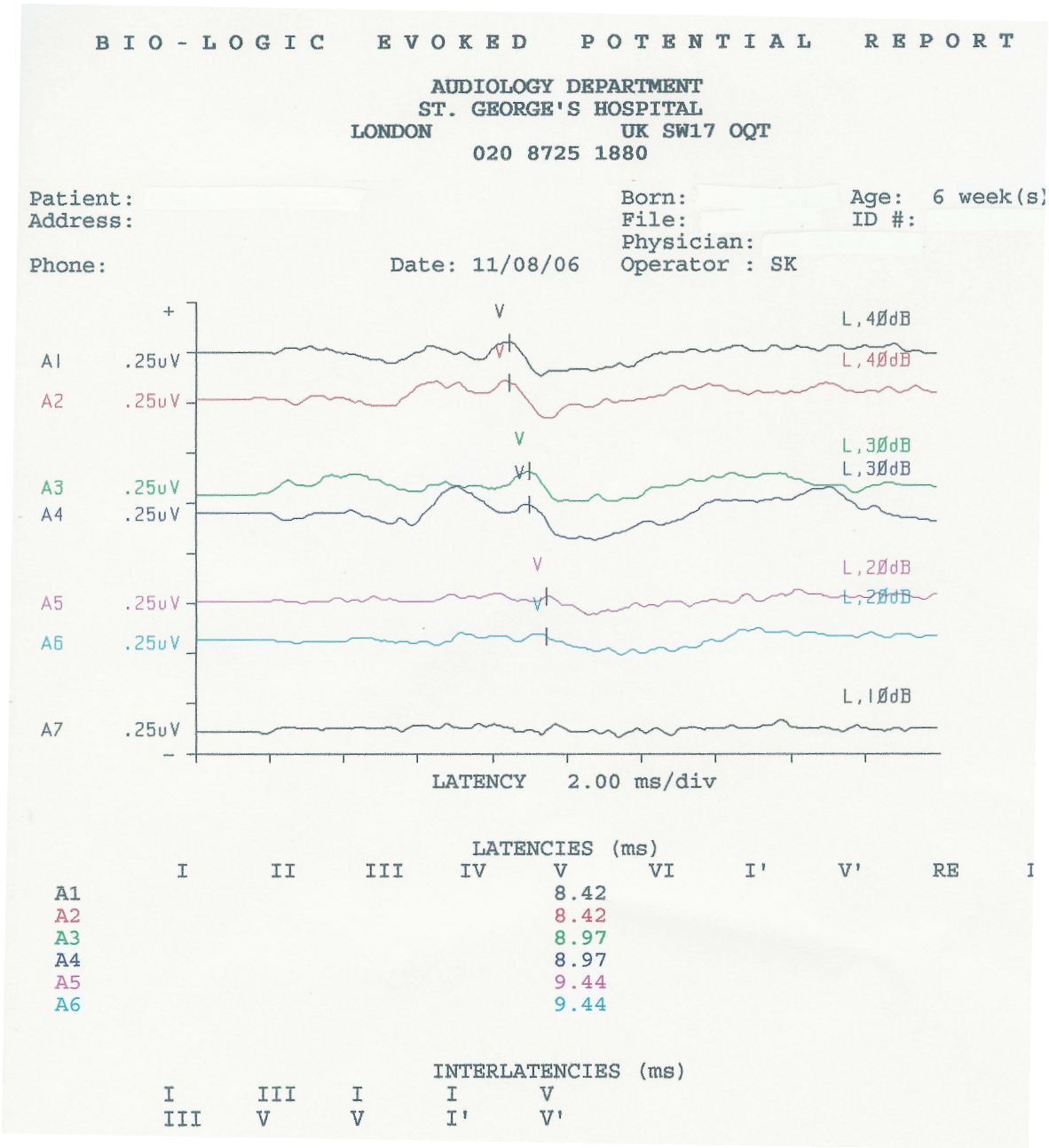


Figure 16 ABR results for left ear ($A_1 - C_z$)

According to NHS Newborn Hearing Screening Programme protocol, the test aims to identify the presence of a response, not a threshold [7]. The response in this case is confirmed by the presence of wave V. The slow latency period of approx. 8ms is typical for a developing ear, since at 6 weeks the 8th nerve mylination is still underway. This figure will reduce by 2-3ms as neural propagation improves. An OAE ear insert was then placed carefully inside both ear canals and the fully automated "Otodynamics EZ-Screen" OAE system demonstrasted the presence of OAEs at 1.5 - 4kHz. Finally, the parents were questioned to ascertain if there were any conduction problems. Along with the presence of OAEs and an ABR wave V the baby passed his first hearing test. The actual measurements took a few minutes to complete but the assessment took approximately two hours because the baby frequently woke and removed the ear inserts and electrodes.

1.6.4 Case Study GW – Adult hearing assessment

History: GW is a 31 year old male referred to the Audiology Department by his GP, complaining of hearing loss and a sense of fullness bilaterally, which was greater in the right ear. He was seen by the Consultant Audiological Physician, I attended the consultation. A history was taken. As a result of seasonal flu and a poor ability to equalise pressure GW has a history of otitis media and frequent build-up of fluid in the middle ear, which has led to perforation of both tympanic membranes. The recent perforation had healed and he was taking *Solfradex* (a sterile solution) to clean the outer ear of cerumen. Examination by otoscopy showed a reddened eardrum and scarring where the perforation had been. The Physician referred him for a tympanogram and a standard adult pure-tone audiometry test which were conducted within the same day. I attended both hearing tests and conducted the tympanometry.

Pure Tone Audiometry Results: A PTA test was conducted with a tone stimulus to test the air-conduction response from both ears. Since the patient presented with an unresolved problem which has recurred for a number of years, I accessed the notes and produced the following summary of audiometry results (Figure 17). 13 samples were collected over 24 years (from 1982 to 2006).

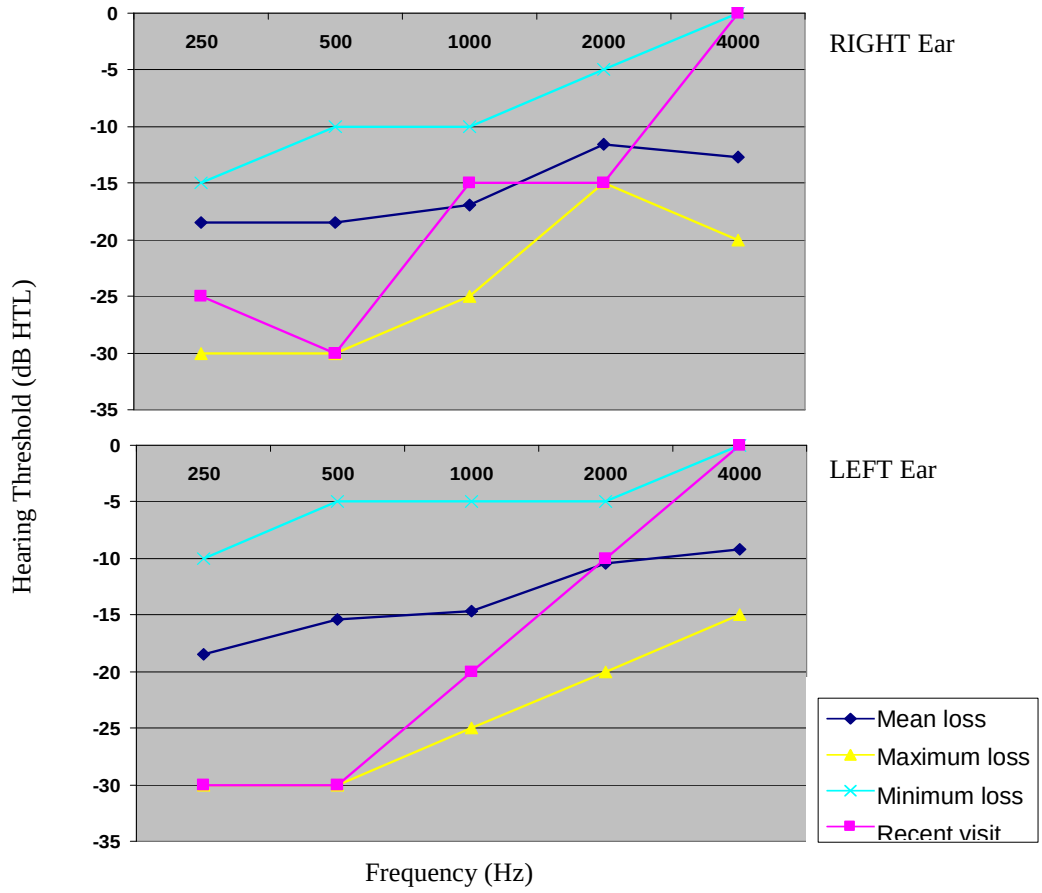


Figure 17 Summary of pure tone audiograms from 24 years

Table 6 Standard deviation across test frequencies

	250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz
Right Ear	5.16	6.25	4.80	3.76	5.36
Left Ear	6.58	7.20	5.76	3.80	4.94

Discussion: There is a bilateral loss of approximately 15 dB HTL, with a poorer threshold in the low frequency range. This recent visit presented the patient's greatest loss of -30 dB HTL at 500 Hz bilaterally and the best response at 4000 Hz bilaterally. A loss of 25-39 dB is regarded as a mild hearing loss. The mean values suggest GW only dipped into a mild, and possibly temporary loss recently. The greatest variation in measurement has also occurred bilaterally, in the 500 Hz range and the lowest variation in the 2000 Hz range. Curiously these peaks in sensitivity correspond with the peaks of the equal loudness contour (see Appendix D). There may simply be a pathological cause or it could suggest that the A-weighting filters used by the audiometer to conform to the equal loudness contour, are not fulfilling their function. It would be interesting to compare audiometry variability data retrospectively, over a large population group. GW was advised to practice middle ear equalisation techniques to reduce the incidence of fluid build-up.

1.7 Safety

Two major patient safety concerns exist in terms of audiological activities: outer/middle ear damage and electric shock risk. The delicate tympanic membrane (0.1mm thick) can easily be perforated if a foreign object impinges on it. This could be a cotton bud, an otoscope tip, an ear insert or anything else placed inside the ear canal. To reduce the risk of a perforated eardrum, a normal anatomy should not be assumed. For example: a short external meatus could deceive a cotton bud wielder. The ear should be approached with care and patient cooperation.

High pressure exposure for an extended duration or from a short loud burst can perforate the membrane and/or cause damage to the stereocillia (noise induced hearing loss). To prevent this occurring, all stimuli presented to the ear are calibrated according to British and International Standards. For clarity, that range of standards is not included here. The reader is referred to the excellent online guide to audiometric calibration and standards produced by The National Physics Laboratory [9].

Electrodes used to record a response from evoked tests complete an electrical path from the patient to the amplifier and filter box, then to the PC and the 50Hz ring mains circuit. Risk of a macro-shock will increase if a fault condition occurs. AEP equipment is therefore classified as patient connected equipment and given the designation "Type B, Class I". A type B device is one with an external or internal connection to the patient, except direct cardiac connection. A class I device is equipment that is earthed and has a fuse in the live wire. To satisfy designation criteria, the device must pass the following safety tests, which quantify the means of potential device failure and current paths from mains to patient, or patient to device. See Chapter 5 for a deeper coverage of electrical safety.

More than 60,000 NHS staff were physically assaulted by patients and relatives of patients in 2005, one assault for every 22 NHS staff [10]. St George's Healthcare NHS Trust has a policy in place entitled "Prevention and Management of Intimidation and Violence (Including exclusion/withholding of treatment)" [11]. This aims to reduce incidence of assault with a series of guidelines and progressive courses of action, leading to the exclusion of the perpetrator (unless admitted on emergency grounds). The key here is good communication and cooperation with the patient; however psycho-social background is not always clear to the clinician.

2. RESPIRATORY FUNCTION

2.1 Introduction

The placement proceeded to six weeks within the Lung Function / Sleep Studies Clinic of St George's Hospital Chest Clinic, during which time I carried out assessment of respiratory function for in-patients and out-patients. Five Clinical Physiologists conduct lung function tests on approximately twelve patients per day. The respiratory tests include: spirometry, body plethysmography and single breath carbon monoxide diffusion testing. The unit also provide a home sleep study service for patients suffering obstructive apnoea. Out-patients are typically referred to the unit by their GP, suffering from shortness of breath. In-patient cases may include pre-operative assessment for the validity of anaesthesia administration and repeat referrals for drug impact assessment, such as chemotherapy drugs. This chapter describes the experiences met within the clinic. The measurement types covered lung volumes and gas flow.

To appreciate the role the department plays in the Community, attention is now paid to the anatomy and physiology of the patient groups who attend the clinics.

2.2 Background

The respiratory system facilitates the intake of oxygen and emission of carbon dioxide for the purpose of metabolic energy conversion [2]. The conducting portion (nose, pharynx, larynx, trachea, bronchi and bronchioles) facilitates the mechanical flow of gas (78.6% nitrogen, 20.9% oxygen, 0.04% carbon dioxide and water vapour) to the areas where diffusion occurs [3]. The respiratory portion consists of the tissues at the blood-gas interface, where external respiration takes place. The cycle is completed at systemic capillaries during internal respiration. Perfusion is the supply of nutritive arterial blood to capillary beds.

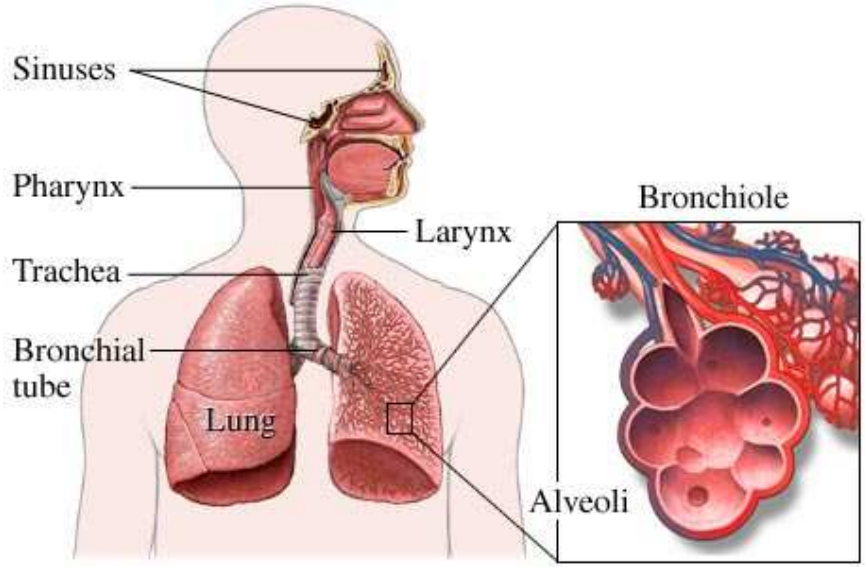


Figure 18 Respiratory system with detail of respiratory portion.

The forces driving gas around the system include the diaphragm and intercostal muscle activity, varying the relative volume (and hence pressure) of the lungs in

relation to ambient pressures; the relative partial pressures of the gases at the blood-gas interfaces and the mechanical action of the circulatory system. Accessory muscles recruited during forced ventilation, include the sterrnocleidomastoid, scalene, pectoralis minor and abdominal muscles. The rate of airflow and the amount of effort required for breathing is influenced by alveolar surface tension, compliance of the lungs and airway resistance [12].

The following common parameters are recorded from patients attending the clinic; they describe lung capacities and volumes [13].

Table 7: Nomenclature of lung capacities and volumes (capacities are combinations of volumes)

Variable	Description	Typical average values (ml BTPS)	
		Male	Female
Inspiratory reserve volume (IRV)	Maximum volume of gas inhaled. From tidal volume to end-inspiratory level.	3100	1900
Tidal volume (V_T)	Volume of gas moved during normal ventilation.	500	500
Expiratory reserve volume (ERV)	Maximum volume of gas that can be expelled following a normal tidal exhalation.	1200	700
Residual volume (RV)	Anatomical volume of gas remaining following maximum expiration.	1200	1100
Inspiratory capacity (IC)	Maximum gas volume that can be inhaled = V_T+IRV	3600	2400
Functional residual capacity (FRC)	Volume not employed during normal tidal breathing = ERV+RV	2400	1800
Vital capacity (VC)	Useful volume, total lung capacity minus residual volume.	4800	3100
Total lung capacity (TLC)	Sum of vital capacity and residual volume	6000	4200

Reference normal indices: To aid the objective assessment of lung function, reference values are used in comparison with patient data. The reference data is internationally recognised and has been collected over a number of years from studies carried out on subjects who were non-smokers, without previous disease that would have affected their respiratory function. The tests and equipment used was similar to that employed today. The data is distilled in regression equations with a mean and standard deviation, incorporating factors such as age, ethnicity, height, weight and gender. A range of these indices can be found in Appendix E.

Note: - Volume measurements are quoted at Body Temperature, Pressure and Saturated with water vapour (BTPS).

The range of lung function variables demonstrated during defined breathing manoeuvres, such as tidal breathing, forced inspiration and forced expiration; can be seen on a spirograph (or spirogram).

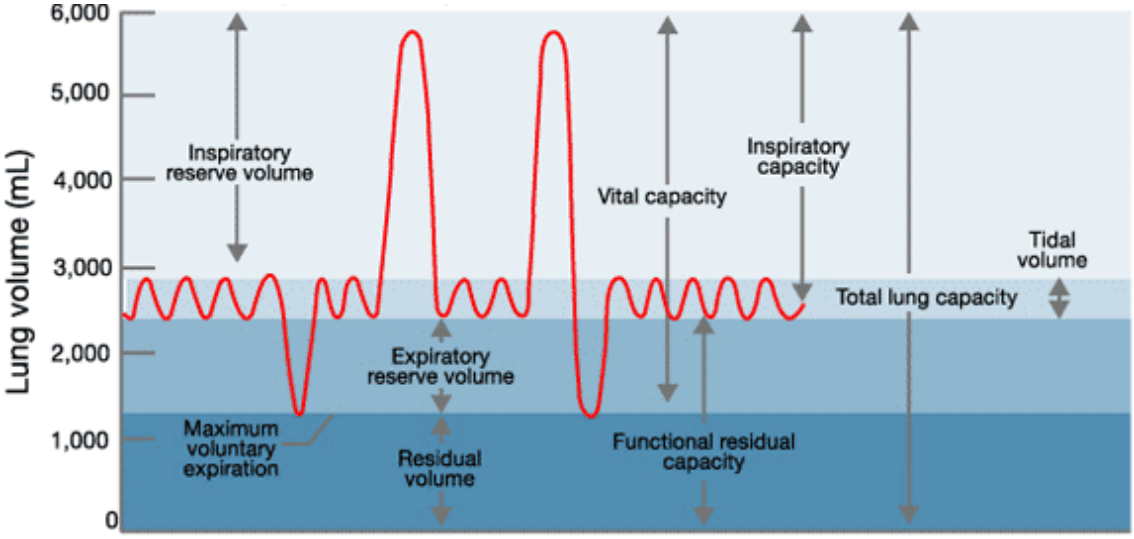


Figure 19 Spirogram showing lung capacity and volume ranges

2.2.2 Respiratory disorders

Respiratory disorders generally present as modifications to the depth and frequency of breathing manoeuvres [14] and can be placed into two categories: restrictive and obstructive.

Restrictive cases limit expansion of the lung volumes, lung compliance is reduced and stiffness is increased. Greater pressure is required to utilise a given volume increase [15]. Examples include: inspiratory restrictive lung disease such as: pulmonary fibrosis, pleural fibrosis or effusion and diaphragmatic weakness. In these cases inspiratory reserve and tidal volumes are reduced (representing shallow breathing). Expiratory restrictive lung indications include obesity, pregnancy, weakness of abdominal musculature, during which expiratory reserve volume is reduced [14]. Skeletal deformities such as kyphosis and scoliosis can restrict lung movement.

Obstructive cases present obstructions to the flow of gas through the conduction pathways. This causes increased resistance and hence more work required to ventilate. An obstruction may not be evident in normal or slow deep breathing since it may have the characteristics of a frequency dependant fluid inertance. This will become evident by raising the frequency during forced ventilation tests. Residual volume is greatly increased and compensated for by an increased inspiratory reserve volume. Examples include: Tumours, chronic bronchitis (mucus plugging), chronic obstructive lung disease (combined chronic bronchitis and emphysema), asthmatic or allergic reaction, damage from pneumonia, pneumothorax, pulmonary oedema, pleural effusion or tuberculosis [16]. The breakdown of alveoli from emphysema is considered to be obstructive.

Disorders of the circulatory system reduce the efficiency of perfusion and internal respiration cycles. For example: heart failure, pulmonary embolism, pulmonary hypertension and anaemia [16].

The Lung Function / Sleep Studies Clinic have these tests at their disposal:

- Spirometry (mechanical function)
- Total Body Plethysmography (mechanical function)
- Single Breath CO Diffusion (gas exchange function)
- Bronchodilator Response (airway function)
- Actigraphy (sleep studies)
- Pulse Oximetry (sleep studies)
- Semi/polysomnography (sleep studies)

2.3 Spirometry

Pathologies alter a combination of lung volumes, capacities or gas flow parameters and as such a range of physiological measurements are carried out on patients referred to lung function clinics. Spirometry is the most commonly used test [17]. The following clinically relevant variables are of interest during spirometry testing [13].

2.3.1 Physiological measurement variables

Table 8 Summary of variables used in spirometry testing

Variable	Unit	Definition	Clinical Significance
FEV_1 - Forced expiratory volume in one second	Litres (BTPS)	The volume of gas expired as quickly as possible in 1 second following maximum inspiration.	Low FEV_1 indicates obstructive disease. Normal or High FEV_1 indicates occurs in restrictive disease.
FVC - Forced vital capacity	Litres (BTPS)	The maximum volume of gas that can be expired as rapidly as possible following a maximum inspiration.	FVC can be reduced in both obstructive and restricted cases. For some cases of obstructive disease a normal FVC can be recorded but will take a longer time to be achieved. If FVC is low but FEV_1 and FET are normal, restrictive disease may be suspected.
FEV_1/FVC	%	Percentage of vital capacity expelled in 1 second.	Normal in normal or restrictive cases. Lower in obstructive cases.
FET - Forced expiratory time	Seconds	Time taken to achieve a full forced expiratory volume.	Longer if obstructed. Shorter if restricted.
PEF - Peak expiratory flow, or peak flow rate	Litres/sec (BTPS)	Peak flow rate during a forced expiratory manoeuvre.	Decreased in obstructive cases. Normal in restrictive cases. Subject dependant.
PIF - Peak inspiratory flow	Litres/sec (BTPS)	Peak flow rate during a forced inspiratory manoeuvre.	Lower in both obstructive and restrictive cases.

Flow volume loops: Volume and flow can be plotted against each other and since one is a derivative of the other, the curves join to form a loop, the volume-flow loop. The following is a presentation of a typical normal vital capacity manoeuvre, from a rapid inspiration to maximum inspiratory capacity, followed by a maximal effort expiration to maximum expiratory level.

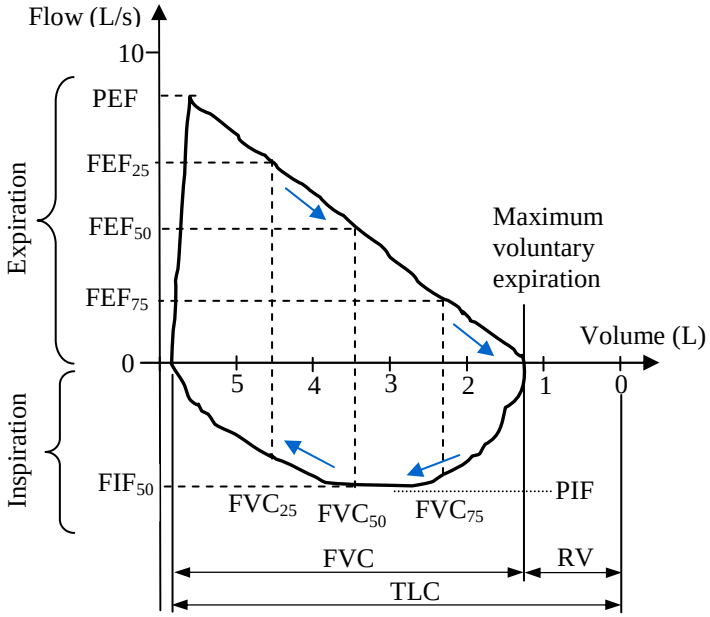
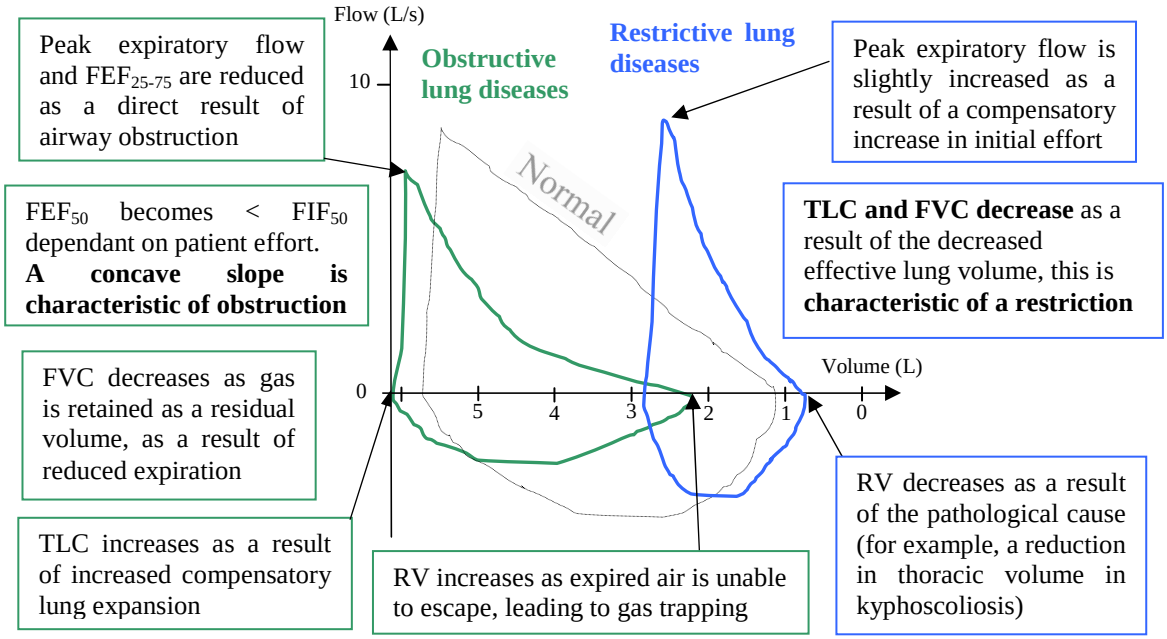
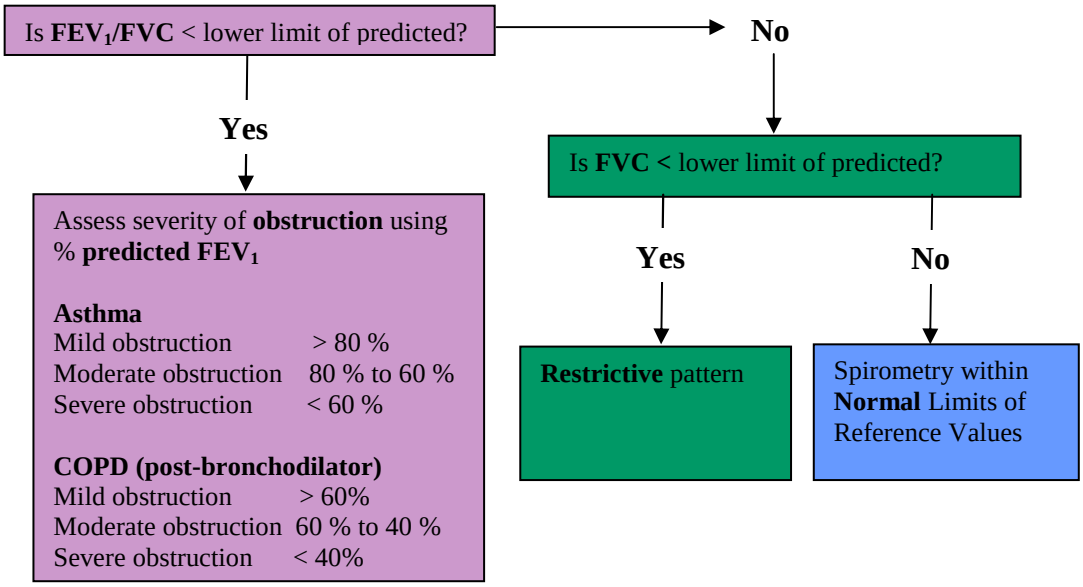


Figure 20 Flow-Volume loop for forced vital capacity manoeuvre (typical values)

A number of parameters are used in conjunction with volume-flow loops, for example: Peak Expiratory and Inspiratory Flows (PEF, PIF) and subdivisions of those at specific volume percentages (FEF_{25}, FEF_{50}, FIF_{25} etc), Forced Vital Capacity (FVC) and volume fractions of FVC at specific time intervals (FEV_1, FEV_3 etc). The shape of the loop can immediately provide a visual indicator to pathology [18], for example:



From the percentage of gas flow forced out during a one second expiratory manoeuvre, it is possible to devise a simple diagnostic heuristic [18]:



If FEV_1/FVC is below 2 standard deviations (SD) of the reference normal an obstruction is suggested because an obstruction is impeding the flow rate of the expelled vital capacity. The severity of the obstruction is assessed as a percentage of that flow. Assuming FEV_1/FVC is within normal limits and the forced vital capacity is below 2SD of reference normal values, a restriction can be assumed since the lungs are not utilising the full reserve volumes.

2.3.2 Spirometry measuring equipment

A number of methods have been employed to measure physiological gas flow, for example:

- ***Rotating vane***: A simple and low cost method. But the dynamic properties (vane inertia and bearing friction) limit the operation to unidirectional gas flow and impede the time response to fast changing flow rates.
- ***Ultrasonic*** transmitter and receiver: Placed obliquely to the gas flow path and determines flow rate from the changes in acoustic transmission. The devices are expensive and assess unidirectional flow
- ***Thermal convection*** measurement: Can be achieved with a hot wire anemometer, where the current required to maintain a constant temperature is measured to determine the rate of cooling produced by a flow of gas, against a reference wire. This is a unidirectional method unless multiple wires are used. However this doesn't take into account the different thermal properties of different expired gasses, this disadvantage is notable when traces gases are expired during diffusion testing.
- ***Differential pressure*** transducers operate on the venturi effect by measuring the pressure drop across a flow restriction. The restriction is created by a group of tubes in parallel, producing a laminar flow. The pressure either side of that flow is measured with a diaphragm capacitive or resistive pressure transducer. From Bernoulli's principle, the volumetric flow rate V' can be found from ΔP, which is then integrated to find volume. The mesh is heated to prevent condensation forming during expiration.

The spirometers used at St George's Hospital Chest Clinic use differential pressure pneumotachometers, because they are capable of measuring bi-directional flow and the transducer is more robust to changes in ambient conditions.

The schematic for a typical pneumotachometer is shown below (Figure 21). The mouthpiece is a cardboard or plastic tube disposed of after each patient use. Gas flows through a filter to remove airborne bacterial and viral pathogens before flowing the laminar flow passes through a series of two or three parallel mounted mesh screens.

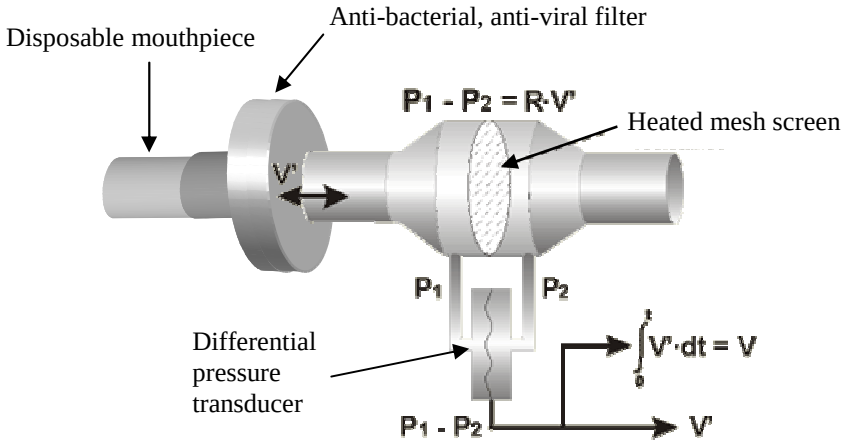


Figure 21 Representation of typical pneumotachometer.

St George's Hospital Chest Clinic use a "Master Screen" produced by Jaeger Tonnies, a brief technical specification can be found in Appendix F.

2.3.3 Calibration of pneumotachometer

The pneumotachometer is calibrated once a day. A two litre syringe is used to deliver a fixed volume of air into the meter with an accuracy of ±25ml. The Physiologist delivers differing flow rate samples of air, ensuring that each sample has a constant flow rate. The procedure is ended automatically by the calibration software.

2.3.4 Spirometry test procedure

1. Patient details are recorded: Hospital number, age, weight, height, sex, and race. Factors that could influence the measurement outcome, such as medications taken and smoking history are also taken into account.
2. The patient is asked to sit with an erect posture, in the body plethysmography box or in a chair at a pneumotachometer. A new mouthpiece (with new anti-bacterial/viral filter for each patient) is fitted to the pneumotachometer, the height of the meter is adjusted to within comfortable reach of the patient. Any abnormal trunk posture could negatively influence the outcome.
3. The patient is asked to place their mouth around the mouthpiece and create an airtight seal. A nose peg is available to occlude airflow through the nose.
4. The patient is asked to relax and breath normally, this provides a normal baseline of tidal flow. Instructions for the following breathing manoeuvre are given, the technologist demonstrates the manoeuvre away from the machine and asks the patient to enact that process.

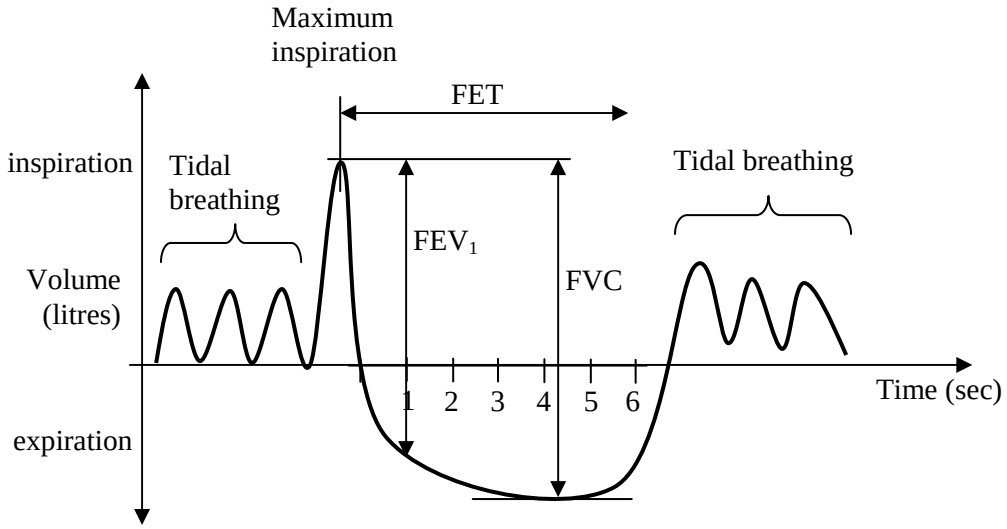


Figure 22 Breathing manoeuvre (spirogram) used during spirometry

5. The manoeuvre is as follows: the patient breaths normally to establish tidal flow, then following an exhalation, a full inspiration with maximal effort is made to followed by a full exhalation with maximal effort to cover the vital capacity range. FEV_1 and FVC are taken from the resulting spirogram.

6. Three trials are carried out to ensure data consistency. Performing more than eight trials is considered to produce inadequate data since the patient begins to tire at this stage. The data should have a maximum variation of 10% across trials, to be clinically useful.

Infection Control: Patients with TB or MRSA are assessed at the end of a working shift, to minimise the spread of nosocomial infections. The filters and tubing are sterilised at the end of each day.

2.3.5 Limitations of spirometry

Spirometry measures relative lung volume changes and as such a major limitation is the inability to measure intrathoracic gas volumes. A pneumotachometer measures flow from a pressure drop, transduction is therefore affected by the condensation content of expired gas and requires temperature control. The pneumotachometer can be placed closer to the mouth which reduces the calculated dead space length of additional tubing required by volume displacement devices. Older volume displacement spirometers are simpler, thermally stable and robust, but are larger immobile devices using a lower resolution chart recorder and often lack the statistical and database facilities of modern computers, all of which make them less practical in departments with a busy clinical caseload.

Forced capacity recordings rely on patient best effort and as such are sensitive to the patient state at the time of testing. The manoeuvres can be difficult to remember and enact. I found that many patients had trouble gripping the mouthpiece and suppressing the gag reflex, which appeared particularly true for elderly ladies.

2.4 Body Plethysmography

The volume of air remaining in the lungs after expiration (functional residual capacity) or after a forced expiration (residual volume) are intrathoracic gas volumes (ITGV) used to determine total lung capacity. Since the spirometry test measures relative differences in an open volume, it is limited in that it cannot determine RV, FRC and the TLC. Information about intrathoracic gas volumes is provided by a body plethysmography (or 'body box') test. The patient is placed within a closed box of known volume (a constant volume plethysmograph). A shutter mechanism operates to briefly occlude flow of gas between the box and lungs, providing a measurable box volume change. Any changes in box volume are related to the pressure changes due to the patients breathing, this is used to determine ITGV.

2.4.1 Physiological measurement variables

The following table summarises the physiological measurement variables of interest during body plethysmography testing (Table 9).

Table 9 Summary of variables used during body plethysmography [13]

Variable	Unit	Definition	Clinical Significance
FRC - Functional Residual Capacity	Litres (BTPS)	Volume not employed during normal tidal breathing = ERV+RV	Increased in obstructive cases. Decreased in restrictive cases.
VC - Vital Capacity	Litres (BTPS)	Useful volume, total lung capacity minus residual volume.	Normal in obstructive cases. Significantly reduced in restrictive cases.
RV - Residual Volume	Litres (BTPS)	Anatomical volume of gas remaining following maximum expiration.	Increased in obstructive cases. Significantly reduced in restrictive cases.
TLC - Total Lung Capacity	Litres (BTPS)	Sum of vital capacity and residual volume	Normal in obstructive cases. Significantly reduced if a restriction is present.
RV%TLC	%	Ratio of RV to TLC expressed as a percentage	Increases in the presence of an obstruction. Remains constant in presence of a restriction.

2.4.2 Body plethysmography measuring equipment

The body plethysmograph (Figure 23) is essentially a box of known volume containing a pneumotachograph. Some additional instrumentation is included for calibration and measurement purposes.

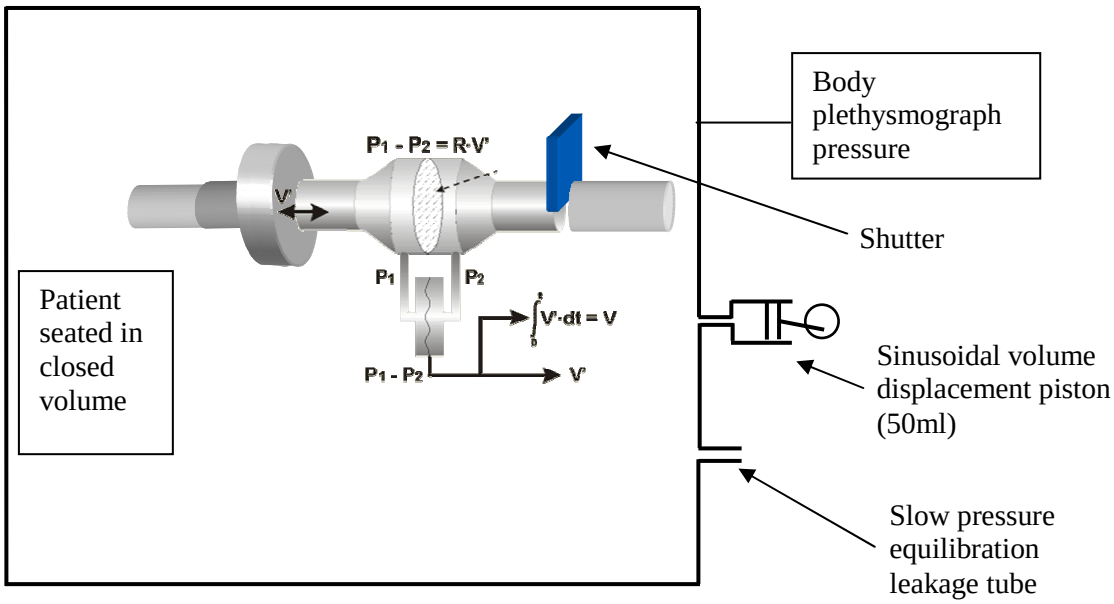


Figure 23 Representation of a Body Plethysmograph

The box is transparent for patient comfort and solid to ensure no unaccounted volume changes during measurement. Internal box pressure is measured and recorded. A shutter is placed in the breathing path after the mouth pressure transducer. The shutter is open at the start of the test and the patient breaths through the pneumotachometer in the same manner as during a spirometry test.

Once a regular tidal breathing pattern is observed, the shutter activates at the next FRC to occlude flow of gas between the box and the lungs. The patient is asked to breath rapidly against the shutter. Since the glottis is open and no gas is being transferred between the box and the lungs, alveoli pressure is equal to the mouth pressure.

The mouth pressure and box volume are plotted against each other, a line of best fit is produced and the gradient $\Delta P/\Delta V$ is taken. Figure 24 shows typical samples obtained from this manoeuvre.

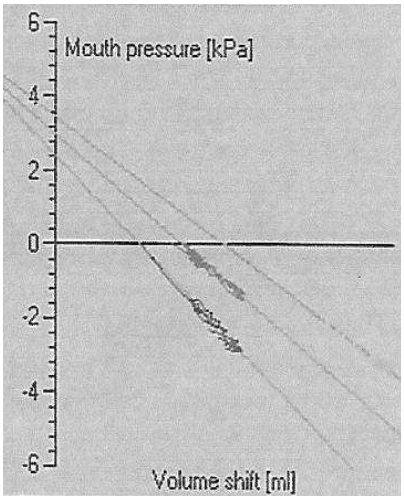


Figure 24 Change in mouth pressure against change in volume

The intrathoracic gas volume at FRC is given by: $V_L(FRC) = P_{AP}\left(\frac{\Delta V_{BP}}{\Delta P_{mouth}}\right)$

Where: P_{AP} is atmospheric pressure
ΔV_{BP} is the change in volume within the body plethysmograph
ΔP_{mouth} is the change in pressure at the mouthpiece.

The derivation can be found in Appendix G.

When the shutter opens, the patient is required to perform a maximum inspiration to fill the lungs (to V_{IN} in Figure 25), followed by a maximum expiration and a further maximum inspiration. This provides a measure of vital capacity (VC) and hence residual volume (RV), since RV = FRC + V_{IN} – VC.

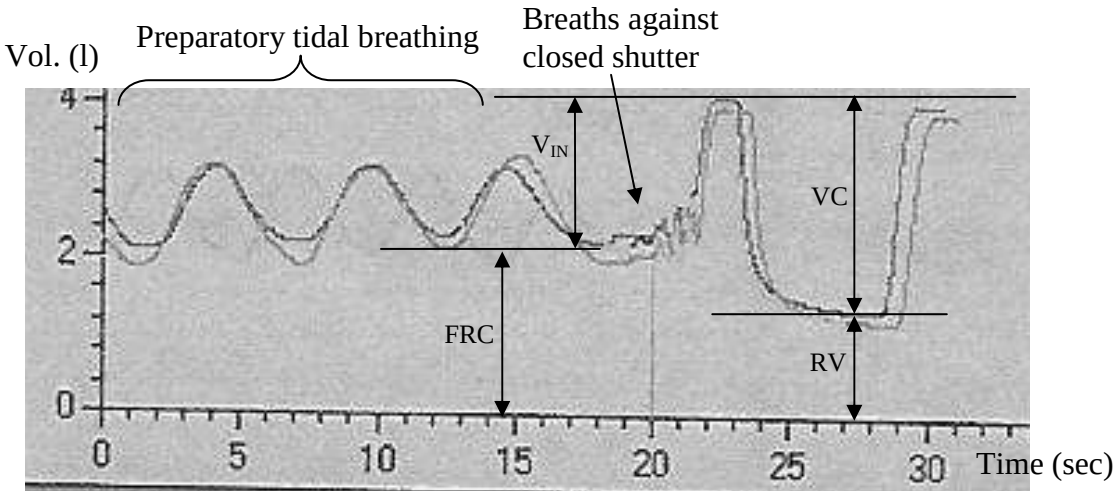


Figure 25 Screenshot of breathing manoeuvre during body plethysmography

2.4.3 Equipment calibration

I carried out three calibration protocols to ensure the quality of body plethysmography data at St George's Hospital; these are conducted once a day at the start of the clinic.

- ***Box pressure-volume calibration****:* The door is closed, the system is switched on and allowed to settle for a few minutes to allow pressures and temperatures to stabilise. An automated pump then delivers a sinusoidal volume of air into the closed box, with a peak volume of 25ml. The corresponding pressure changes are monitored and a correction factor is automatically applied to the subsequent measurements.

Note: the volume of the box is corrected for the volume of the patient, using the approximation: Patient volume (litres) = patient weight (kg)/1.07, where 1.07 kgm^{-3} represents the average density of the human body. For this reason 'puffer jackets' and other such low density, high volume clothing is removed before the test.

- ***Air leakage – halve value test:*** A small leakage hole and plastic tube is in place in the box wall to slowly equalise the pressure between the inside and outside of the box when the door is closed. The leak does not have an impact on the box pressure

measurement because the leakage time constant is higher than the breathing manoeuvre sample duration. The manufacturer calculates that the time constant should be calibrated to fall within 4 and 7 seconds, this can be done so by altering the tube length. The calibration process involves changing the volume of the box and measuring the time taken for the pressure to fall to half its initial value, this is achieved with the calibration pump.

- ***Pneumotachometer calibration:*** Is carried out with a two litre syringe as per the protocol used during spirometry testing (see section 3.4.4).

2.4.4 Plethysmography test procedure

The following procedure is observed with patients undergoing body plethysmography testing at St Georges. The hardware functions of the test are automated.

1. The procedures are explained to the patient prior to carrying out the tests, they are instructed as to what is required of them.
2. The patient is seated inside the body plethysmograph and the position of the pneumotachometer is adjusted to reach the patients mouth. As with spirometry, any slouching could restrict the expiratory capacity of the lungs and so the patient sits with an erect posture.
3. A nose clip is provided to occlude the nose, the patient asked to grip the disposable mouthpiece and make an air tight seal with the lips. Some frail patients or people with a very sensitive gag reflex find this difficult due to the size and dryness of the tube. A new anti-bacterial/viral filter is used with each patient.
4. The patient is then asked to breath normally until a relaxed tidal flow ensues, during which time the volume-flow loops are plotted.
5. When the Physiologist is confident that a repeatable relaxed breathing pattern is occurring, a command is sent to close the shutter at the next FRC.
6. The patient is instructed to breath in and out twice, making rapid panting breathes against the shutter. At which point the mouth and box pressures are recorded.
7. The shutter opens immediately after the panting breaths have been carried out and the patient continues breathing manoeuvres for spirometry testing: maximum inspiration, maximum expiration and a further maximum inspiration.

Some patients find the instructions a little daunting and struggle to remember them. The manoeuvres require maximum effort, so the Clinical Physiologist stands by the patient, encouraging and coaching them through the procedure, by shouting relevant instructions.

2.4.5 Limitations of body plethysmography

The test suffers the limitations of using a pneumotachometer (as described in §2.3.5). In addition, the results of whole body plethysmography assume all volume changes within the box to be intrathoracic gas volume changes in origin. The test is therefore sensitive to space filling items such as colostomy bags and low density high volume clothing. The test assumes isothermal conditions which requires the system to acclimatise to the seated patient, but is sensitive to changes in ambient conditions. The Lung Function Unit at St George's Hospital has recently moved from a *portacabin* in direct sunlight into an air conditioned brick building, staff have noticed a great improvement in the stability of measurements, particularly in summer months.

2.5 Single breath carbon monoxide (CO) gas diffusion

Understanding gross lung properties forms only part of the picture of pulmonary pathophysiology. The effectiveness of blood-gas diffusion can be assessed using one of a number of gas diffusion tests [14]. The method employed at St George's Hospital (and generally within healthcare) is the modified Krogh technique, to determine the capacity for carbon monoxide to travel from alveoli to the blood. Assessment of the resulting transfer factor D_{LCO} (or T_{LCO} in Europe) provides information on pulmonary microcirculation. In cases of airflow obstruction, T_{LCO} helps to distinguish asthma from bronchiectasis and to distinguish both from emphysema. In volume loss without airflow obstruction, the T_{LCO} will tend to be normal in extrapulmonary restriction, but it will reduce in intrapulmonary alveolar and vascular disease [18]. The following variables are of interest during single breath CO testing, they will be referred to in the following discussion.

2.5.1 Physiological measurement variables

Table 10 Summary of variables of interest during CO diffusion testing

Variable	Unit	Definition	Clinical Significance
T_{LCO}SB – Transfer factor of CO during single breath test	mmol/min/kPa (STPD)	Measure of the diffusing capacity of CO across the alveoli capillary membrane	Reduced in both restrictive and obstructive lung disease
T_{LCOc}SB – Haemoglobin corrected	mmol/min/kPa (STPD)	As above – corrected for haemoglobin count	As above – corrected for haemoglobin count
V_A – Alveolar volume	Litres (STPD)	Alveolar volume V_A = RV + $V_{inspired}$ – $V_{equipment\ deadspace}$ – $V_{anatomical\ deadpsace}$	T_{LCO}SB is proportional to V_A, so a reduction in one results in a reduction in the other variable.
K_{CO} – Transfer coefficient	mmol/min/kPa/Litre	The ratio T_{LCO}SB / V_A	In normal subjects: 4-5ml of CO is transferred per minute per litre of lung volume. This is reduced in both restrictive and obstructive cases.
H_b - Haemoglobin count	g/100ml	Haemoglobin count	Affects the amount of CO that is taken up by the blood. Normally for each gram of Haemoglobin, CO uptake is 7%
K_{COc} - Haemoglobin corrected	mmol/min/kPa/Litre	As above – corrected for haemoglobin count	As above – corrected for haemoglobin count

2.5.2 Theory of operation

The test involves the patient breathing a gas mixture (0.28% CO, 9% H_e, 19% O_2 and 71.72% N_2) and holding the breath for eight seconds. Gas analysers measure the concentrations of the inspired and expired CO and He gases. The transfer factor is then calculated. Carbon monoxide is used over oxygen because it diffuses in a single direction (oxygen is bi-directional), also CO combines 210 times more readily with haemoglobin than oxygen does. The transfer factor ($T_{LCOc}SB$) corrected for haemoglobin levels is given by:

$$T_{LCOc}SB = \left(\left(\frac{V_A \times 60}{(P_B - P_{H_2O}) \times T}\right) \times \left(\frac{1}{22.4}\right) \times \ln\left(\frac{F_{ACO}\,|_{t=0}}{F_{ACO}\,|_{t=T}}\right)\right) \times Hb_{correctionfactor}$$

$$= KCO \times V_A \qquad \text{(expressed in mmol/min/kPa at STPD)}$$

Where:
- V_A = alveolar volume (ml STPD)
- P_B = barometric Pressure (kPa)
- P_{H_20} = water vapour pressure at 37^0C (kPa)
- T = breath hold time (seconds)
- 60 gives the conversion from seconds to minutes
- 1/22.4 gives the conversion from ml to mmol
- F_{ACO} at time t is the inspiratory alveolar CO concentration, given by:

$$F_{ACO}(t) = FICO \times \left(\frac{F_{EH_e}}{F_{IH_e}}\right)$$

Where: FICO is the CO concentration in the inspired gas
F_{EHe} and F_{IHe} are the expiratory and inspiratory helium concentrations.

Also, the $Hb_{correctionfactor} = \dfrac{10.22 + Hb}{1.7 \times Hb}$

The alveoli volume V_A is given by:

$$V_A = \frac{F_{IH_e}}{F_{EH_e}} \times (V_1 - V_D) \times STPD_{correctionfactor}$$

Where:
- V_1 = the inspired volume
- V_D = dead-space volume
  - Equipment dead-space = 280ml
  - Anatomic dead-space = 2.2 ml/kg of body weight.

All inspired volumes must be corrected to standard temperature, pressure and depth (STPD).

2.5.3 CO diffusion measuring equipment

Figure 26 below shows the additions to the spirometry equipment used at St George's Hospital, to implement CO diffusion testing. Inspired and expired gas volumes are measured from the integration of flow across the heated pneumotachometer. Test gases are delivered from the reservoir, under the control of a demand valve. An electrochemical fuel cell analyses expired CO, a katherometer measures thermal conductivity to assess helium concentrations. The shutter ensures test gases are being inspired during the sampling period.

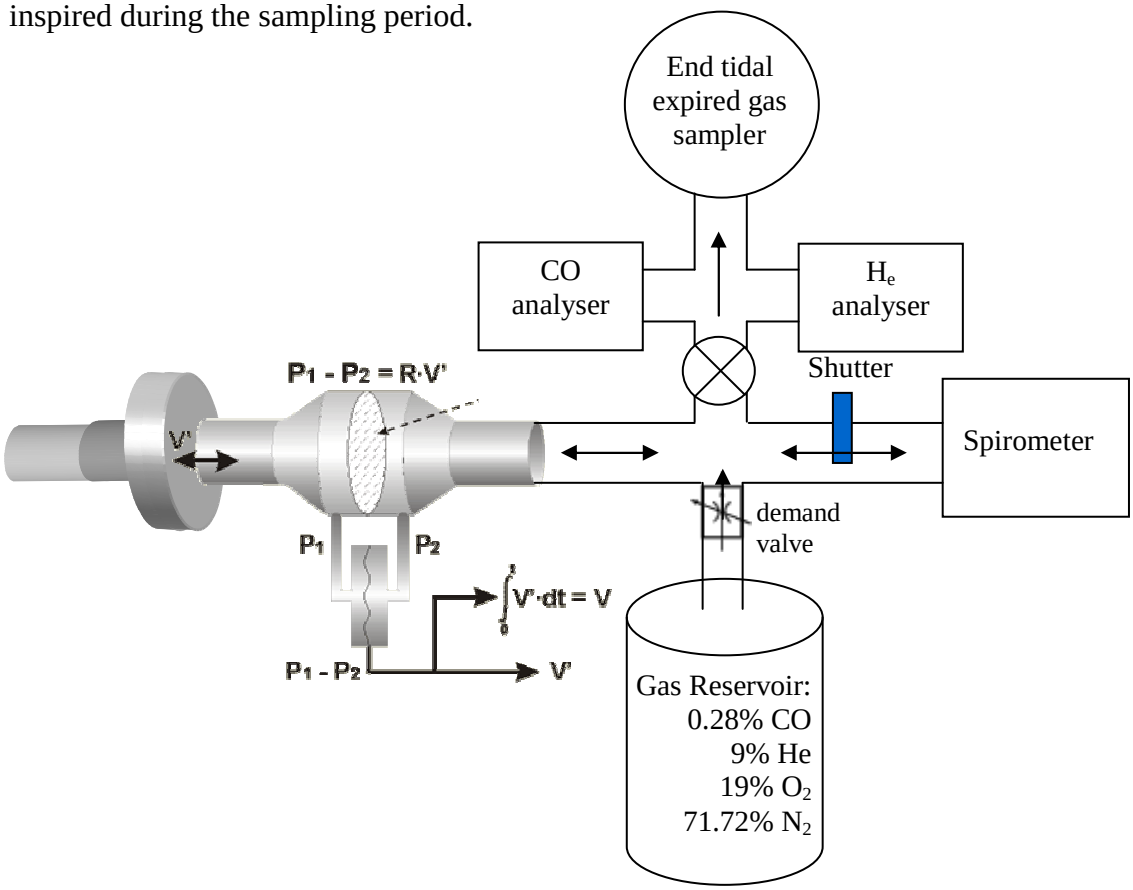


Figure 26 Functional diagram of the single breath CO diffusion measurement equipment 'Master Screen Jaeger Toennies'.

2.5.4 Equipment calibration

1. The pneumotachometer is calibrated with a two litre syringe as discussed in section 2.3.3.
2. The gas analysers are calibrated to room air and to a peak gas concentration level using the test gases in the reservoir. The reservoir cylinder is opened to deliver test gas to the analysers. A delay is made for the transducer to respond, then the system automatically takes peak values when a sufficient plateau is reached. Due to the nature of the different operating principles of the transducers, the response times vary. The CO analyser has a slower response.

Test gas (BOC MA 0735 / 0011R) is supplied by BOC Special Products Division, which hold a Department of Health medical product licence for the production of medical grade gases. For information regarding BOCs traceability chain see [19]

2.5.5 Single-breath CO diffusion test procedure

1. The patient is asked to breath normally through the disposable mouthpiece, the shutter is open and the demand valve closed.

2. The patient is asked to gently inspire and then expire as much as possible. At the end of maximum expiration the shutter is closed and the patient inspires rapidly to maximum lung capacity. At the start of inspiration the demand valve opens and the patient inhales the test gas.

3. After an eight second breath hold, the patient is asked to exhale. The equipment discards the first 750ml to account for anatomical and equipment dead space. The second 750ml is sampled as it represents alveolar expired gas. The concentrations of helium and carbon monoxide are measured; the plateaus reached in the corresponding graphs determine the concentration of gases.

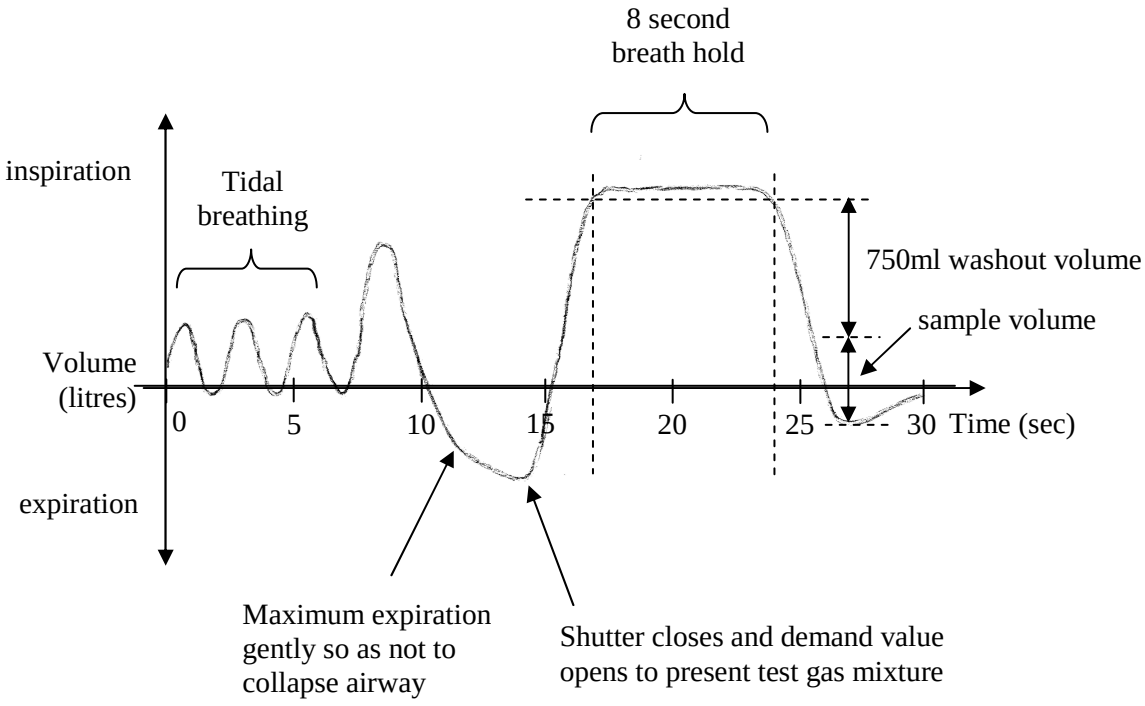


Figure 27 Breathing manoeuvre for gas diffusion

4. The test is repeated after a four minute break, to allow the gases to be completely washed out of the lungs. The test is repeated until reproducible results are obtained with a variation of no more than 10% from each other.

5. When the operator is satisfied with the results, the transfer factor and alveolar volume is calculated by the software. The carbon monoxide transfer factor is approximately 13.5 for women and 14.5 for men.

6. A small blood sample is taken from the finger and a ‘Hemocue’ device measures the haemoglobin in the blood. The factor is entered into the computer and corrects the $T_{LCO}SB$ values to those for normal haemoglobin levels ($T_{LCO}SB$).

2.5.6 Data Quality

Reproducibility of results is checked and can be added as a comment for the Physician. This is important to mention, to improve the standardisation and performance of the test. Published guidelines are available from the American Thoracic Society (ATS). One important factor in the success of such tests is the encouragement given by the operator throughout the tests.

The criteria that should be met during the tests are:

1. The volume-time graph should show a smooth, rapid inspiration from RV to TLC.

2. The inspired vital capacity should be at least 90% of the vital capacity that was measured in the spirometry test performed earlier.

3. The breath-hold line should be flat.

4. The alveolar sample volume should be 05-1 litres and must be collected in 3 seconds or less.

5. The expiration of the dead space gas should be rapid and smooth.

6. The volume of the discarded dead space gas should be between 0.75-1 litres, if the subjects VC is less than 2 litres, the dead space gas volume may be reduced to 0.5 litres.

7. If the spirometry volume exceeds the best previously determined VC measured from the spirometry test, then the VC should be questioned and may need to be repeated.

2.6 CASE STUDIES

2.6.1 Case Study – Chronic obstructive pulmonary disease (COPD)

The patient is a 59 year old male outpatient of a Consultant Cardiothoracic Physician at St George's Hospital. He is an ex-smoker of 12 years, 10-20 cigarettes per day, unable to walk for more than a few metres without experiencing severe breathlessness. He was referred for lung function tests and seen by a locum Clinical Physiologist, I observed the tests. Blood gases (pH, $PaCO_2$, PaO_2, SaO_2, ctHb) were also taken by the Chest Clinic Sister and shown to be normal.

The patient conducted spirometry and gas diffusion tests. The plethysmograph was faulty at the time of this visit (09/01/07), so I have included plethysmography results from a previous referral (24/01/06).

	PredL	Pred	PredU	Act1	Act2	Act3	Act4	Act5	%Pred
Date				10/05/05	10/05/05	24/01/06	09/01/07	09/01/07	
FEV 1	2.62	3.45	4.29	0.57	0.62	0.67	0.48	0.73	21
FVC	3.38	4.38	5.38	2.71	4.11	3.35	3.03	3.40	78
VC MAX	3.64	4.56	5.47	2.71	4.11	3.54	3.18	3.46	76
FEV1%M	64.83	76.59	88.35	21.03	15.09	18.92	15.23	21.06	28
PEF	6.56	8.54	10.53	3.00	3.20	3.24	3.49	2.92	34
PIF						6.65	6.03	6.39	
FET						20.13	17.49	17.09	
TLCOSB	7.53	9.85	12.17	2.74		2.77	2.53		
TLCOc	7.53	9.85	12.17	2.61		2.59	2.47		
VA	6.99	6.99	6.99	5.74		5.59	4.53		
KCO	-1.21	1.38	3.97	0.48		0.50	0.56		
KCOc	-1.21	1.38	3.97	0.45		0.46	0.55		
VIN	3.64	4.56	5.47	4.16		3.81	3.53		
Hb				15.40		17.20	15.00		
ITGV	2.62	3.61	4.59			7.65			
RV	1.73	2.40	3.07			6.12			
TLC	5.99	7.14	8.29			9.76			
RV%TLC	28.02	36.97	45.92			62.70			
VC	3.64	4.56	5.47			3.64			
ERV	1.21	1.21	1.21			1.53			

Figure 28 Results for Case 1: Spirometry (upper set), gas diffusion (middle set) and total body plethysmography (lower set)

Note: the results are not corrected for race.

The patient also conducted spirometry pre- and post-ventilation through a nebuliser, containing 20mg of the bronchodilator *ventolin*. The clinic also use *salbutamol*. The bronchodilator results are unremarkable and not included here.

The best representative spirometry manoeuvre is shown below (Figure 29). From the reduction in peak flow and the flattened response, the graph immediately suggested a severe distal airway obstruction. This is confirmed by spirometry variables: FEV_1 is very low (21% of predicted), FVC is low (78%), the ratio FEV_1/FVC is therefore very low (28% of predicted). Peak flow is also very low (34% of predicted), which indicate an obstruction. The shape of the expiratory curve suggests that the airway obstruction is distal.

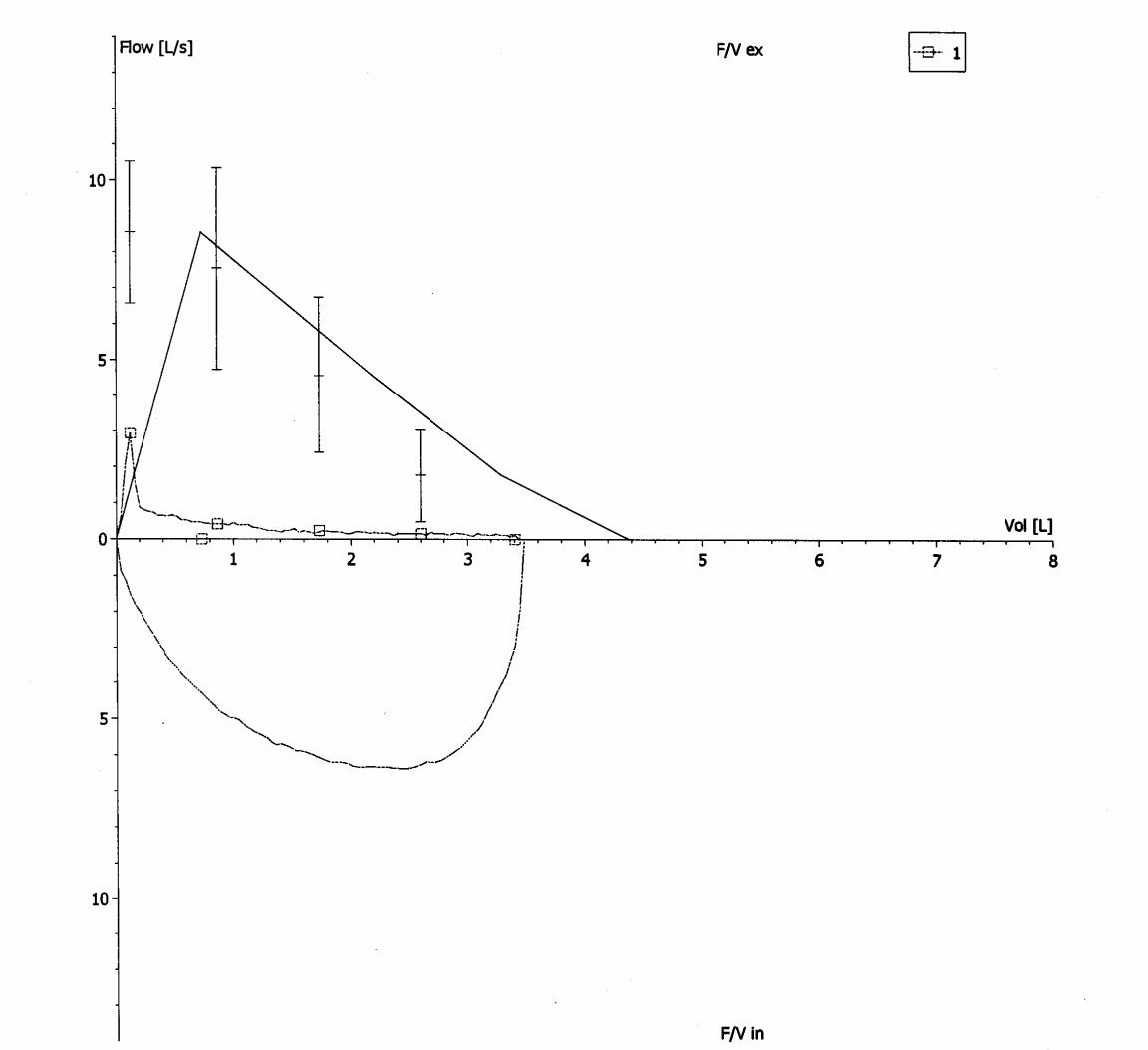


Figure 29 Spirometry results for case 1

$T_{LCOc}SB$ and K_{COc} are much lower than predicted (25% and 40% respectively), indicating the alveoli to be the site of obstruction. The plethysmography results indicate high intrathoracic and residual volumes. These data are indicative of emphysema. In some individuals there is a genetic deficiency of the protein $alpha_1$-antitrypsin, which inhibits the production of proteases released by neutrophils during the inflammatory response. Unchecked, these destructive enzymes digest the alveoli tissue, causing the sacs to coalesce, reducing the effective surface area for gas transfer. I tracked the patient's notes and found that he does have an $alpha_1$-antitrypsin deficiency. There is no treatment for emphysema. The patient was prescribed anti-inflammatory drugs and advised increase use of a bronchodilator through a nebuliser.

2.6.2 Case Study – Restrictive lung disease

The patient is a 64 year old black male and a cardiothoracic inpatient at St George's Hospital. He uses a wheelchair. He was referred for lung function testing and seen by the Chief Clinical Physiologist. The patient showed diminished cognition and high levels of fatigue, there were some communication difficulties.

The patient undertook spirometry in the morning and returned to complete one measurement of gas transfer. The best spirometry trace is shown below (Figure 30).

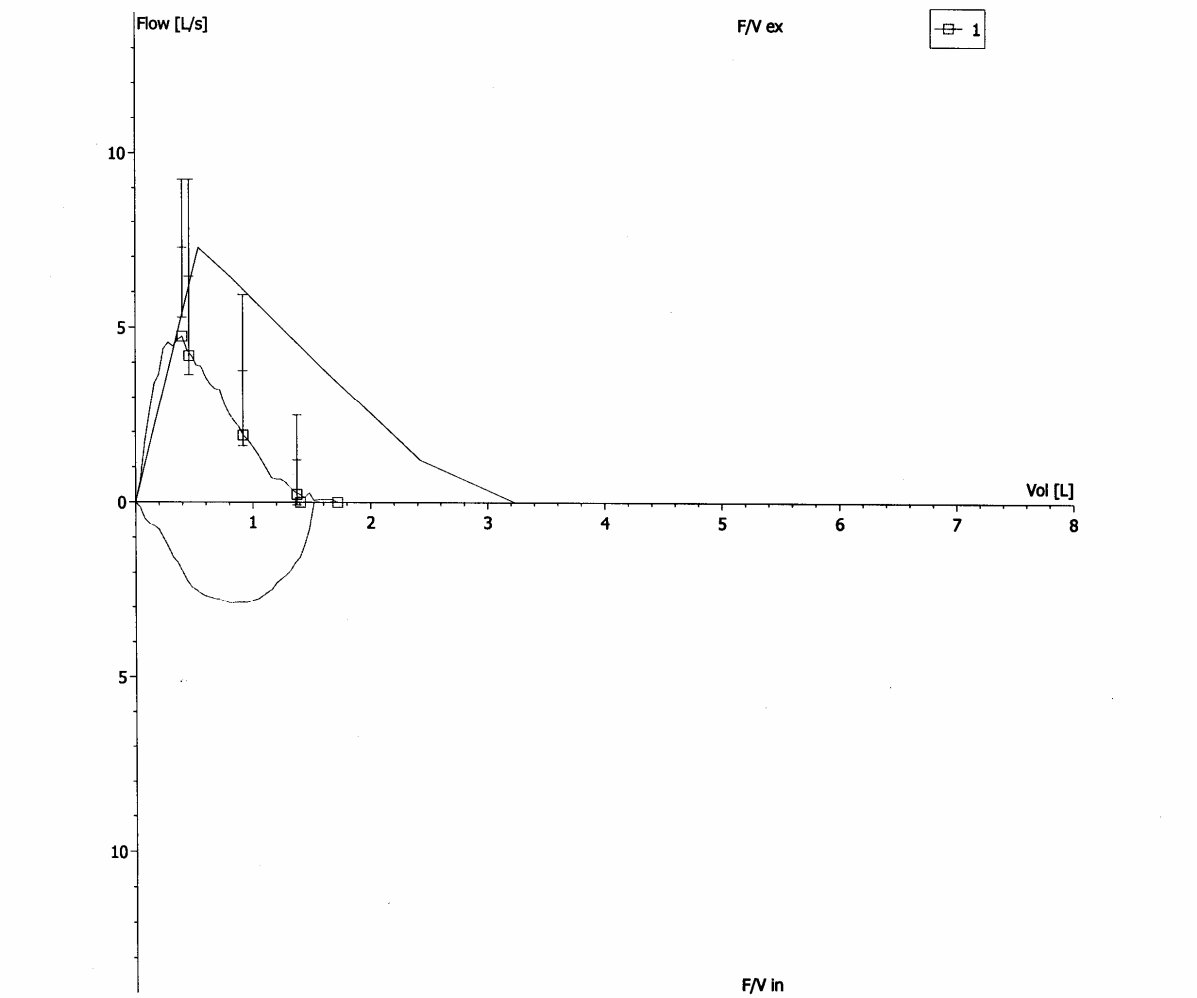


Figure 30 Spirometry results for case 2

The spirogram immediately suggests a restriction, from the reduction in forced vital capacity. This is confirmed numerically as a 53% reduction from predicted. All other lung volume and capacity variables are below predicted (see overleaf), other than the FEV_1%M and KCO_c. However FEV_1%M is a ratio of two low values and KCO_c is a corrected variable using a very low haemoglobin count. The spirometry is indicative of a restrictive cause, whilst the gas transfer results demonstrate a reduction in diffusion efficiency.

	PredLL	Pred	PredUL	Act1	%Pred	SR Value
Date				08/01/07		
FEV 1	1.73	2.56	3.40	1.40	55	-2.27
FVC	2.24	3.24	4.24	1.72	53	-2.49
VC MAX	2.43	3.35	4.26	1.83	55	-2.70
FEV1%M	64.11	75.87	87.63	76.56	101	0.10
PEF	5.28	7.26	9.25	4.75	65	-2.08
PIF				2.88		
FET				5.51		

Date	PredLL	Pred	PredUL	Act1 09/01/07	%Pred	SR Value
DLCOSB	5.27	7.59	9.91	2.62	35	-3.51
DLCOc	5.27	7.59	9.91	4.15	55	-2.43
VA	5.55	5.55	5.55	2.26	41	
KCO	-1.26	1.33	3.92	1.16	87	-0.11
KCOc	-1.26	1.33	3.92	1.84	138	0.32
VIN	2.43	3.35	4.26	1.43	43	-3.42
Hb				8.90		

Figure 31 Numerical results for case 2

This case demonstrates that a number of factors can prevent the presentation of high quality data: the body plethysmography calibration facility was faulty and under a commercial service contract with maintenance engineers situated hundreds of miles from the hospital. The patient's fatigue and lack of communication with the clinician prevented trial replication. Many patients, especially elderly people with dry mouths, find it physically demanding to grip and form a seal around the pneumotachometer. Some find enactment of the breathing manoeuvre mentally challenging.

In such instances other assessment measures must be found, for example use of the Medical Research Council's Dyspnoea Scale, as shown below.

Table 11 Medical Research Council dyspnoea scale

Grade	Degree of breathlessness related to activities
1	Not troubled by breathlessness except on strenuous exercise
2	Short of breath when hurrying or walking up a slight hill
3	Walks slower than contemporaries on level ground because of breathlessness, or has to stop for breath when walking at own pace
4	Stops for breath after walking about 100m or after a few minutes on level ground
5	Too breathless to leave the house, or breathless when dressing or undressing

2.6.3 Case Study – Chronic obstructive pulmonary disease (COPD)

TD is a 56 year old male referred by his GP for respiratory function testing, following concerns about breathlessness and fatigue. The patient was a heavy smoker for approximately 35 years prior to stopping last year. TD undertook spirometry, body plethysmography and gas diffusion testing.

Results:

Table 12 Lung function test results - TD

	PredH	Pred	PredUL	Act1	%Pred	ΔValue
FEV_1	2.82	3.66	4.49	**2.27**	62	-2.72
FVC	3.56	4.56	5.56	**5.83**	128	2.09
VC Max	3.83	4.75	5.67	**5.83**	123	1.93
FEV_1%M	66.09	77.8	89.61	**38.89**	50	-5.43
PEF	6.86	8.84	10.83	**6.23**	70	-2.16
PIF				**5.08**		
FET						
D_LCOSB	7.99	10.31	12.63	**5.61**	54	-3.33
D_LCOc	7.99	10.31	12.63	**6.13**	59	-2.96
VA	6.99	6.99	6.99	**8.45**	121	
KCO	1.04	1.44	1.84	**0.66**	46	-3.21
KCOc	1.04	1.44	1.84	**0.73**	50	-2.95
VIN	3.83	4.75	5.67	**5.76**	121	1.80
Hb				**11.90**		
ITGV	2.56	3.54	4.53	**7.56**	213	6.70
RV	1.57	2.25	2.92	**5.05**	225	6.83
TLC	5.99	7.14	8.29	**11.01**	154	5.53
RV%TLC	25.29	32.24	43.19	**45.83**	135	2.12
VC	3.83	4.75	5.67	**5.97**	126	2.17
ERV	1.30	1.30	1.30	**2.52**	194	

Note: TD is Caucasian with height 178.5 cm and weight 63.3kg. See Appendix H for volume-flow loop

Interpretation:

- Spirometry demonstrates a 50% reduction in FEV_1 as a percentage of FVC and a 30% reduction in peak flow, which are indicative of an airway obstruction. According to the guidance (shown in §2.3.1) this is a severe obstruction. FVC has decreased as gas is retained as a residual volume, as a result of reduced expiration.
- Total body plethysmography shows that residual volume has more than doubled (225% predicted) as expired air is unable to escape, leading to gas trapping. TLC has also increased (by 154%), as a result of increased compensatory lung expansion.
- The diffusing capacity of the lungs (corrected for Haemoglobin level) has reduced by 41%, however an increase in alveolar volume places the figure at a 50% reduction per unit volume. This suggests that the obstruction has resulted from poor perfusion.

TD also conducted spirometry pre- and post- ventilation through a nebuliser, containing 2.5 mg of salbutamol. Spirometry data showed less than 10% difference, suggesting a chronic condition was present. These data, with the patient's smoking history point towards emphysema. An option for managing emphysema us the use of steroids.

2.6.4 Case Study – Restrictive lung disease - asbestosis

FD is a 77 year old male undergoing repeat assessment following treatment for a myocardial infarction. He has never smoked, but has had a 20 year occupational exposure to asbestos. FE conducted spirometry, body plethysmography and gas diffusion testing.

Results:

Table 13 Lung function test results - FD

	PredH	Pred	PredUL	Act1	%PredSR	Value
FEV_1	1.54	2.37	2.21	1.90	80	-0.93
FVC	2.16	3.16	4.16	2.32	73	-1.39
VC Max	2.34	3.26	4.18	2.32	71	-1.69
FEV_1%M	61.59	73.35	85.11	81.93	112	1.20
PEF	4.99	6.97	8.95	7.99	115	0.84
PIF				2.85		
FET				7.31		
DLCOSB	4.90	7.22	9.54	2.92	40	-3.04
DLCOc	4.90	7.22	9.54	2.97	41	-3.01
VA	5.95	5.95	5.95	2.91	49	
KCO	0.74	1.18	1.62	1.00	85	-0.66
KCOc	0.74	1.18	1.62	1.02	86	-0.61
VIN	2.34	3.26	4.18	2.11	65	-2.05
Hb				14.10		
ITGV	2.48	3.46	4.45	2.75	79	-1.19
RV	1.95	2.63	3.30	1.60	61	-2.50
TLC	4.96	6.10	7.25	4.05	66	-2.93
RV%TLC	35.04	43.99	52.94	39.45	90	-0.83
VC	2.34	3.26	4.18	2.45	75	-1.44
ERV	0.84	0.84	0.84	1.15	138	
R tot	0.30	0.30	0.30	0.17	56	
SG tot	0.85	0.85	0.85	1.99	234	

Note: FD is a non-Caucasian with height 165 cm and weight 69kg. See Appendix H for volume-flow loop

Interpretation:

- Spyrometery shows a decrease in vital capacities (27% decrease in FVC, 29% in VC_{max}, 20% in FEV_1); peak expiratory flow has increased by 15%, all of which are indicative of an airflow restriction.
- Plethysmography demonstrates a 49% decrease in residual volume, which may be a compensatory volume to account for the 34% decrease in total lung capacity.
- Overall gas diffusion capacity has reduced by 49% (corrected for Haemoglobin level). However, taking a reduced alveoli surface area into account, the carbon monoxide transfer coefficient shows only a slight decrease of 14% (Hb corrected), suggesting the restriction is not occurring due to effective alveoli volume.

These data are suggestive of asbestosis, which is a fibrous thickening in the terminal bronchioles following an inflammatory response to asbestos inhalation. Current management of asbestosis includes the use of bronchodilators, postural drainage and pharmacological erosion of the fibrous material.

2.7 Risk Management and Safety

The first step in improving safety is to identify and quantify the risks associated with an activity. The International Standards Organisation have produced ISO 14971:2000 “Medical Devices – Application of risk management to medical devices”, which sets out standards for manufacturers placing devices into medical markets. Of interest is a questionnaire set out in Annex A, which presents a series of questions to be asked of a specific device, in order to aid the identification of risks. I have answered these questions with attention to the Jaeger Tonnies Masterscreen equipment used by the Lung Function Clinic, the answers can be found in Appendix I. A summary of the salient points raised through this process is highlighted below.

Table 14 Summary of salient risks associated with lung function clinic activities

Nature of Risk	Action
Infection or spread of hospital acquired infection (HAI)	• Use a new anti-bacterial, anti-viral filter with the pneumotachometer for each patient. • Use gloves when taking blood samples • Use gloves and apron when assessing infected patients, assess at end of day session • Close clinic if assessed patient later found to have TB, if same day • Sterilise tubing and mouthpieces at end of day • Disinfect general aspects of equipment regularly
Electrical injury	• Jaegger tonnies masterscreen equipment designed to comply with BS-EN-60601-1 and appropriate collateral standard • Jaegger tonnies classified as a Class I, Type BF medical device • Installation of equipment supervised by Medical Physics department • Planned maintenance of equipment managed under commercial contract and audited by Medical Physics department
Misdiagnosis	• Calibration of gas sensors and pneumotachometer • Trial repetition • Measurement practiced under recognised protocol • Maximum effort samples taken • Training of staff • Effective communication amongst clinical team

For additional notes on electrical safety see Chapter 5 and for additional notes on risk assessment see Appendix J.

3. NEUROLOGY

3.1 Introduction

The placement proceeded to five weeks within the neurophysiology department. The tests experienced are electrophysiological in nature. I used electroencephalography (EEG) for the assessment of seizures, and electroneurograghy (ENG) and electromyography (EMG) for the assessment of peripheral nervous system disorders, notably carpal tunnel syndrome.

3.2 Electroencephalography

EEG is the measurement of the electrical activity of the brain. It is used in the Neurology Department at St George's Hospital to diagnose seizures. This section of the report begins by describing the functional anatomy of the brain followed by a brief description of seizures. It then describes the measurement technique used, followed by case studies.

3.2.1 Functional Anatomy of the Brain

The central nervous system consists of the spinal cord and brain. The brain is divided into 3 main functional parts: cerebrum, brainstem and cerebellum.

- The cerebrum is associated with the conscious functions of the nervous system. It is anatomically divided into both the left and right frontal, parietal, temporal and occipital lobes. Regions of the cerebrum are known to be dedicated to motor and sensory functions, e.g. hearing is associated with the upper part of the temporal lobes and vision is located within the occipital lobes. The thalamus sorts the vast array of incoming information, whilst the hypothalamus regulates autonomic and homeostatic functions (such as sleep-wake cycles, temperature and fluid balance).

- The cerebellum (located posteriorly and inferiorly to the cortex) is a coordinator in the voluntary muscle system and acts in conjunction with the brainstem and cerebral cortex to maintain balance.

- The brainstem links the brain with the spinal cord. The m edulla regulates respiratory and cardiovascular functions and the pyramidal corticospinal tracts coordinate muscle function. The cord extends to the cauda equina, propagating peripheral nerves to the musculature and receiving efferent signal from the sense receptors.

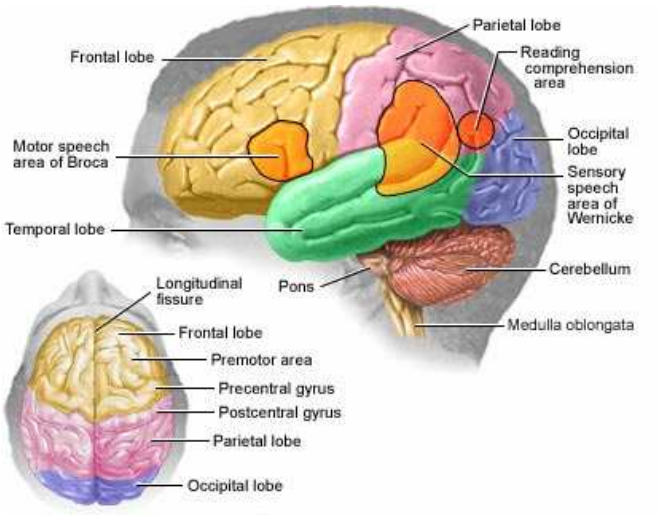


Figure 32 Major functional components of the brain

3.2.2 Normal EEG

EEG signals range from approximately 10µV – 500µV, with a frequency of 0.5 – 40 Hz and originate from inhibitory post-synaptic potentials (located in neuron cell bodies) and excitatory post-synaptic potentials (located in the dendrites) [20]. Signals picked up on the surface are vector sums of a large number of equivalent dipoles per unit volume. Currents from widely scattered generators travel through the inhomogenous media to detection electrodes. An infinite number of combinations can give rise to the same pattern of surface potentials; this is a great problem in localising the source of an event. Fortunately patterns can be found in the signals by evoking responses. Therefore much of EEG interpretation revolves around comparison with known data sets [21]. Normal patterns include alpha, beta, theta, delta, mu and lambda rhythms [22], as summarised in Table 15 below.

Table 15 Summary of major brainwave features. *Note: There is no international agreement on the bandwidth of different brainwave activities.

Wave	Bandwidth*	Characteristic	Location	Significance
Alpha	8Hz – 13Hz	Background activity arising from normal cognisant behaviour Attenuated with eyes open	Symmetrical Max. amplitude in occipital region	Asymmetry suggests disorder such as infarction, or hemiatrophy
α 20-200µV Approx. scale  1s				
Beta	14Hz +	Variable amplitude. Does not respond to state of eyes. Amplitude increases with drowsiness and drug inducement.	Fronto-central regions	Hemispheric localisation.
β µV  1s				
Theta	4Hz – 7Hz	Lower frequency, variable amplitude	Symmetrical Midline and temporal regions	Focal activity indicates site of local lesion
θ µV  1s				
Delta	<4Hz	Not present in normal waking adult	Cortex	Presence implies cerebral dysfunction or localised brain disease
δ 50µV  1s				

Mu and Lambda waves are measured to indicate normal function, but are less familiar to electroencephalographers [23]. EEG frequency increases with cerebral activity and amplitude increases when the eyes are closed.

3.2.3 Seizures

A seizure (previously known as a convulsion or epilepsy) is a sudden and uncontrolled excessive neuronal discharge within the brain [24]. Many seizures are idiopathic (of unknown cause), others have been classified, such as:

- *Localised (or focal) partial seizures* originate in areas of the cerebral cortex. Simple partial seizures occur for 30 - 60 seconds and end without loss of consciousness. Complex partial seizures involve a loss of consciousness, occur for approximately 1 minute and originate from the temporal lobe. Partial seizures may develop across brain regions and become tonic-clonic

- *Tonic-clonic seizure* (or grand mal) is so-called after the exhibited tonic and clonus behaviour and is a more dangerous generalised seizure originating from both cerebral hemispheres, it looks like a random signal as seen in Figure 33

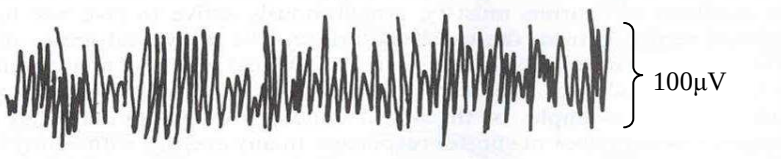


Figure 33 Characteristic tonic-clonic seizure [25]

- *Absence seizure* (or petit mal) is characterised by generalised, bilaterally synchronous, predominantly frontal spike-and-wave discharges, with a frequency of 2.5 Hz – 3 Hz, lasting less than 30 seconds [35]. Post-ictal EEG is normal. An absence seizure may cause a loss and immediate resumption of consciousness for a few seconds. The patient is unaware of the event and it may occur many times per day. I recorded the following absence seizure from an in-patient at St George's Hospital. Note: The peak amplitude is 10 times greater than resting alpha amplitude

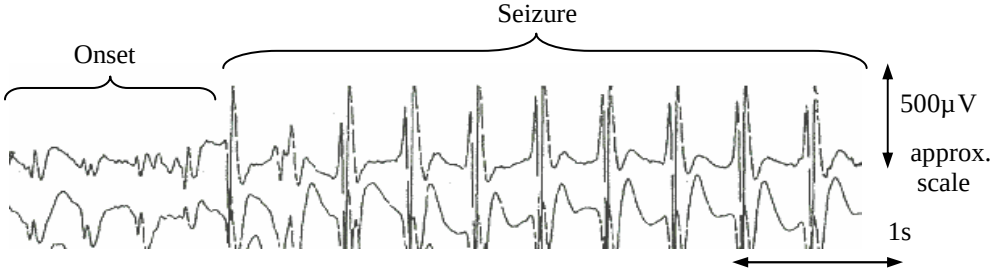


Figure 34 Characteristic spike and wave discharge of an absence seizure.

- *Myoclonic seizures* are sudden brief repetitive jerks of limbs without warning

- *Atonic (akinetic) seizures* have an even quicker onset loss of muscle tone, resulting in a sudden fall which may cause more serious subsequent injuries

A non-clinical seizure is one that presents no outward clinical signs. This demands intense concentration from the investigating staff to spot pre-ictal and ictal signs, such as muscle tremors. For this reason the bedside EEG is a valuable diagnostic tool for the Consultant Neurophysiologist. A recurrent or continuous seizure without recovery of consciousness is termed *status epilepticus* and requires intensive care.

Incidence is higher in the young and old [24], with childhood seizures following a typical pattern:

- *Febrile seizures* occur from 3 months to 5 years of age. CT imaging is the primary screening method
- *Childhood absence seizures* occur from 4 to 10 years and present as normal background activity interrupted by hyperventilation induced 3 Hz spike-and-wave activity
- *Tonic-clonic seizures* develop into puberty and present as spike-and-wave activity
- *Juvenile myoclonic seizures* occur from 10 - 20 years and present as poly-spike-and-wave activity induced by a strobe light (photic stimulation)

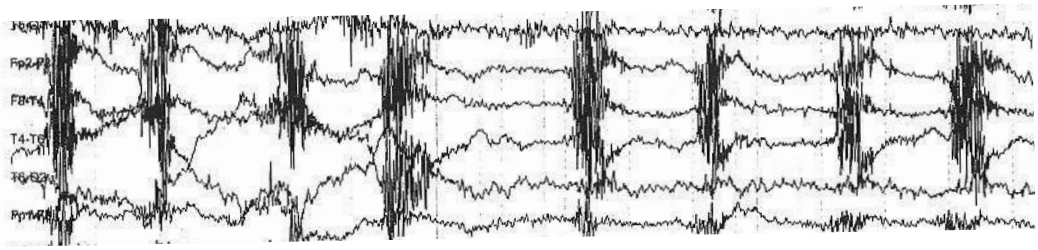
Acquired adult seizures are generally focal as a result of a neuropathology or trauma

3.2.4 Noise and Artefacts

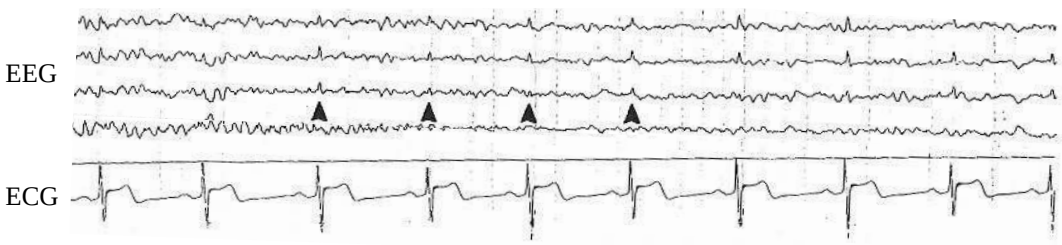
The signal-to-noise ratio for a 10µV signal is approximately 5:1 (for purely biological noise). This highlights the major difficulty in obtaining EEG before external noise is even applied [26]. External noise comes from various sources such as the 50 Hz mains system, which introduce harmonics at 100 Hz, which is within range of signals under research investigation [27]. This source can be attenuated with a notch filter, but this may also filter out a small fraction of relevant biological data.

The following is a selection of artefacts and noise sources found in EEG traces:

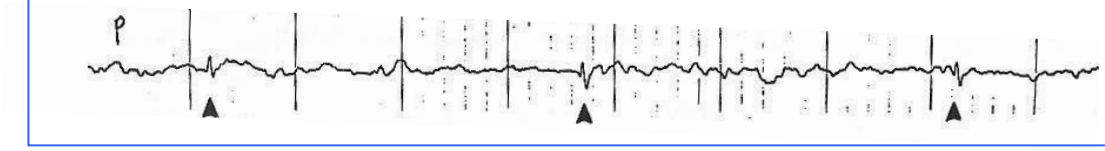
EMG artefact: A rhythmic chewing artefact originates from the patients masticatory muscles. Since EMG occupies approximately 10 Hz – 5 kHz, this can be reduced with a low pass filter. This example demonstrates the importance of having knowledge of resting muscle activity - information which can be found by touching the patient's skin which is of particular value in the case of non-clinical seizures.



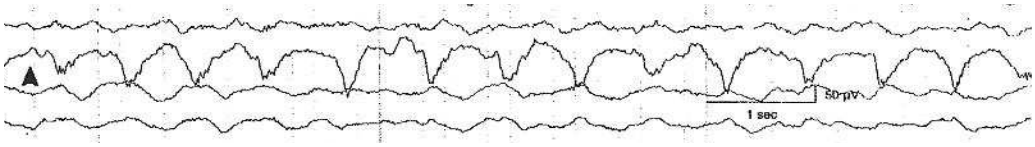
ECG artefact: ECG is also recorded from the clavicles to account for cardiac spikes.



IV drip artefact: A fascinating example of EEG sensitivity is shown in this IV drip artefact. The electrostatic charge surrounding the single drop of fluid falling to the drip chamber produces a periodic noise spike on the trace. This type of artefact is accounted for with experience and a methodical approach to data interpretation. It also demonstrates a difficulty of working outside the controlled EEG clinic.



Feeding pump artefact: On intensive care units, the EEG headbox is often positioned close to feeding pumps, which induce an artefact that closely resembles epileptiform behaviour.



The reporting Physician may not be present at the EEG recording, so these examples demonstrate how thorough a technical report must be.

3.2.5 Clinical Data Collection

The challenge of capturing the small signal and reducing unwanted data requires a methodical data collection protocol which takes into account the following:

Clinical Setting:

- An EEG clinic is ideally situated away from any source of electromagnetic noise, such as a radiography department, lift switch gear, an air-conditioning plant, or the air-call transmitter system.
- Electrical mains distribution is such that conductor paths around EEG rooms are separated and kept to a minimum, some artefacts are characteristic of faulty electrical equipment in adjacent buildings on the same mains line.
- No unnecessary electrical devices (such as radios) are used in the clinic.
- Temperature is kept as constant as possible to reduce variable perspiration artefact at the skin-electrode interface.
- The test elements within the room are physically separated, including: the computer, headbox (which contains pre-amps. and filters), the patient and the clinician.
- EEG amplifiers typically have a high common mode rejection ratio (see below).
- Leads lengths are as short as possible.
- Detachable tip electrodes are not in use in St George's Hospital.
- The patient is asked to be as still as possible.

Electrode Placement: The 10-20 system is an internationally recognised system of electrode placement. It provides clinics with data collection uniformity around the world and is the same system I used in the Audiology Department. The system is so-called because the surface of the head is divided into 10 and 20 degree divisions against a reference line, which is drawn circumferentially from the nasion to inion, (see Appendix L for the full 10-20 system).

An electrode pair is called a *montage*. Different montages are used to highlight different physiological features, for example: A bipolar montage is one were two neighbouring electrodes on the 10-20 map are connected for simple differential amplification; this is useful in detecting local events, such as focal seizures. An average reference montage is one were each electrode is applied to one input of each amplifier, thereby contributing a fraction to the total output; this is useful when recording global activity, such as a tonic-clonic seizure. For a good discussion on the types, limitations and uses of different montages see [28]. The system in use at St George's Hospital records all individual electrode voltages with respect to reference zero so that it is possible to generate all possible montages for later viewing. It is also possible to view different montage types during the investigation.

***Electrode-skin contact*:** To ensure a consistent signal-to-noise ratio over the duration of the test and reduce differential noise coupled to electrodes, a number of factors are required of the electrode-skin contact:

- Electrode impedance and the difference between electrode impedances should be kept low and constant. At St George's Hospital electrode impedance should be less than 6 kΩ and the difference between electrodes less than 2 kΩ. The leads are also aligned. These methods ensure that 50 Hz coupled currents for example, are induced in equal proportion between electrodes and hence rejected as common mode noise
- Non-polarisable Ag/AgCl cup electrodes are used to reduce local voltages at the electrode-skin interface
- Cup electrodes are used with a conductive adhesive gel called *collodion* in the Neurophysiology Department to ensure an even contact surface area and reduce local capacitances, this is particularly important where hair obscures the surface
- Secure contact surfaces are required for the duration of the test (up to 20 to 30 minutes). Cup electrodes can be held in place with surgical tape
- A smooth and clean contact area is ideal, but hair, oils and skin preparations are the reality. The skin is prepared with the application of a saline or aluminium oxide solution called *nuprep* and if required, a slight abrasion using clinical emery paper
- Minimum movement of electrodes is required to reduce variations in electrode local impedance and prevent movement artefact
- No discomfort or skin irritation to the patient
- Care is taken when placing the electrodes over the fontanel of neonates
- Some leads and cups can withstand sterilisation, whilst others are interchangeable and disposable

Amplification: Electrode pairs are amplified using double differential amplification, using what is commonly referred to as an *instrumentation amplifier* which is referenced to a separate ground electrode. This is placed in close proximity to the

measurement site on an electrically neutral surface. Bony surfaces such as the back of the hand provide good low impedance reference points.

Stimuli: Once a background EEG is taken to determine the baseline resting state, a range of stimuli can be used to evoke responses. Verbal commands such as "open and close eyes" simply serve to demonstrate variation from normal physiological spikes. Others are *activation procedures* that attempt to induce a state of seizure. Activation procedures include: Photic stimulation using a strobe light, drug induction, hyperventilation and sleep deprivation among others. It is important to note that during an activation procedure great care and attention is paid by the Clinician to the EEG traces. The aim of stimulation is to determine what frequencies and stimuli cause a seizure and where they originate, it is not to evoke a seizure. As soon as pre-onset signs have been noted, the stimulus is removed. For an introduction to this topic see [27].

Equipment used at St George's Hospital*:* The Neurophysiology Department use two 'NicoletOne' examination systems (VIASYS Healthcare inc., Pensylvania, US) as shown below. One is portable and used with a laptop for use outside the ward, the other is set up with a desktop PC in the clinic. Both have the same software functionality.

Figure 35 NicoleteOne system in use at St George's Hospital.

The system has a resolution of 256 16-bit samples per channel per second. It is capable of bedside and long term recording of EMG and EEG, with photic stimulation. Facilities exist for data telemetry, network support, data analysis and video synchronisation. Video is captured digitally with a simple webcam style camera. Amplifier and filter settings used in the department include:

Parameter:	**Typical value:**
• High pass cut off frequency	5 Hz
• Low pass cut off frequency	70 Hz
• Bandwidth	65 Hz
• Common mode rejection ratio (CMRR)	120 dB
• Mains band stop filter	50 Hz
• Gain	100 dB
• Noise amplitude	2 µV pk-pk

The Paediatric Intensive Care Unit (PICU) sees a number of patients with seizures, warranting the purchase of a 4-channel EEG headbox. Currently only T_3-T_5 and F_Z-C_Z were being recorded due to underutilisation of the patient monitor. After witnessing the efforts made by the Consultant Paediatric Neurophysiologist to obtain physiological data from a patient (in Case PB discussed at the end of this Chapter), I instigated the use of all four channels on the ward.

The Siemens Infinity SC7000 patient monitoring system (Figure 36) which was being used to monitor PB, is capable of displaying five full screen traces and a number of numerical parameters. The traces being monitored for PB included: NIBP, heart rate and two channels of EEG, with SPO_2 displayed numerically. After discussion with the Consultant and the Senior House Officer, I withdrew a separate SPO_2 monitor from the equipment library, connected it to PB and reprogrammed the Siemens SC7000 to display NIPB, heart rate and all four channels of EEG data. I later demonstrated the correct operation of the monitor to the nurses on the ward.

Figure 36 Siemens Infinity SC7000 patient monitoring system

Reporting data: EEG data supports differential diagnosis, it does not solely provide a diagnosis. Because increasingly recruited limb movements provide clues to help localise the seizure, video is captured to record prodromol signs (onset behaviour) of the patient directly in clinic or via the telemetry system. The case history and neuropsychological profile also form major aspects to assessment [21].

An important distinction is made between a technical report and a diagnostic report produced by EEG Physiologists. The former being an accurate account of recording parameters and any deviations from a normal recording session (including photic stimulation frequency, filter settings, commands given and those not carried out and so on). The later is an interpretation of the results. Neurophysiology clinics across the NHS and across the world partition tasks differently, some specialist EEG Physiologists perform analysis of the results and present a diagnostic report. Local arrangements must be understood by all staff working within the department.

At St George's Hospital the data collection process is conducted by a Physiologist, who then presents a technical report to the Physician. It is useful for the Physician to observe which muscles are stimulated during seizure onset and how that stimulation progresses around the body, so video data is also collected and the team meet once a week to discuss the caseload. This teamwork approach is a valuable quality check particularly when less experienced staff join the group.

3.2.6 EEG Test Protocol

The following protocol is observed during EEG collection at St George's Hospital:

1. The patient removes their outdoor clothes and is comfortably seated in an armchair. In the case of bed-bound patients, the area surrounding the head is cleared

2. The clinician introduces his or herself and asks for confirmation of name and date of birth, a brief history of the neurological problems, detail of the current presenting problem and if there is a seizure history or known cause for seizure onset. The procedure is explained to the patient

3. The scalp is prepared and electrodes are individually placed:

    a. The area should be dry before electrodes are placed and care must be taken to ensure the patient feels no discomfort
    b. An abrasive pad or water based gel can be used to help reduce skin-electrode impedance
    c. 23 Ag/AgCl cup-electrodes are used in the routine montage and attached with collodion according to the 10-20 system. These include ground and reference electrodes and two additional electrodes placed on the clavicles to monitor ECG. To avoid confusion each free end is plugged into the appropriate headbox connection as each electrode is placed.

4. Impedance is checked automatically by the software package 'NicoleteOne'.

5. The recordings begin with a lead-in period of eyes closed relaxation. If a length of consistent background EEG is apparent, the clinician proceeds to a range of commands according to the clinical request. Each command and any observed event is labelled on the trace for quick retrieval during analysis

6. Video data is also recorded of the patient and time-synchronised to the EEG trace

7. The examination lasts approximately thirty minutes. After which time the electrodes are removed, the patient's hair is cleaned and the patient leaves

8. At St George's Hospital a technical report is submitted to the Physician with the results. An EEG review meeting is then held with the Physician and the Clinical Physiologist in attendance.

There are no contraindications for EEG, but some physiological aspects can alter the recording, such as: pharmacological history and pregnancy.

3.3 Peripheral Nerve Conduction Assessment using ENG and EMG

3.3.1 Peripheral Neuropathies

The peripheral nervous system comprises: sensory nerve axons, which extend from distal sense receptors to the cell bodies of the dorsal root ganglia and motor nerve axons, which extend from neuromuscular junctions to the anterior horns [3] (Figure 37). The system is a widely bifurcating network that innervates dermatomes with apparent irregularity, for example: the median nerve innervates 2 ½ fingers of the hand [2].

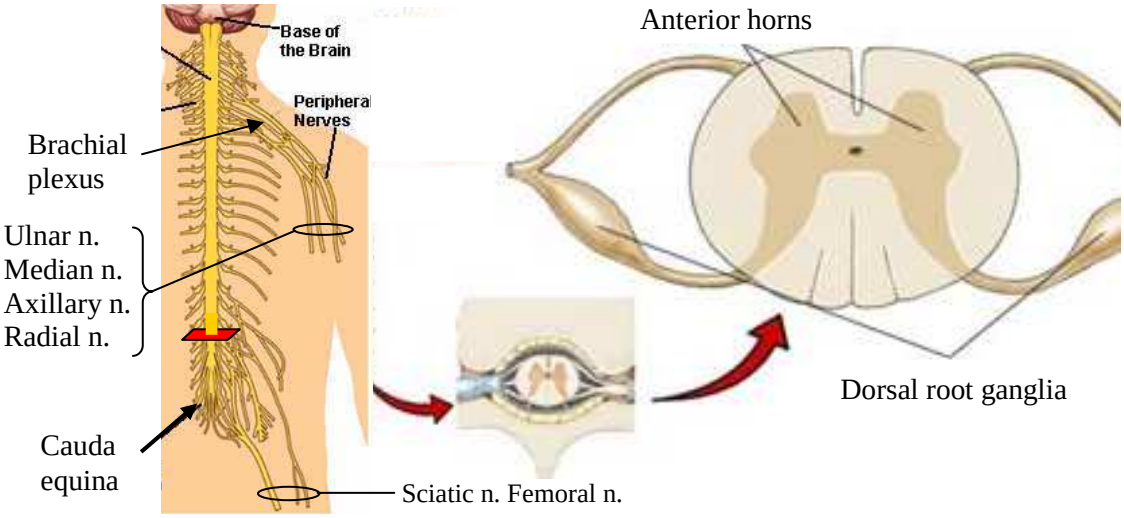


Figure 37 System overview with detail of nerve origins and major branches

Neurophysiology patients can present with numbness or weakness in the extremities, which translates to sensory or motor nerve fibre dysfunction. The extent of dysfunction or rate of rehabilitation is assessed through measurement of nerve conduction velocity (NCV) using electroneurography (ENG). Fibres mylinated by Schwann cells conduct action potentials at approximately 40 – 70 m/sec, unmylinated fibres conduct at 1-5 m/sec (generally too slow to be of interest in NCV studies), demylinated fibres do not conduct (conduction block of a pathophysiological cause) [17]. Nerve conduction measurement at the neuromuscular junction is of interest to anaesthetists, who monitor the action of nerve blocks during general anaesthesia.

3.3.2 Normal nerve conduction variables

Nerve conduction signals are evoked by an electric or magnetic stimulus and can be measured *orthodromically* (in the physiological direction of the nerve conduction) or *antidromically* (opposite to the physiological direction). Antidromically evoked signals have a more distinct AP waveshape, with a greater amplitude than orthodromically evoked signals and as such are the signals recorded [31].

With reference to Figure 38, the following variables are of interest when recording electroneurographic signals [30]:

- ***Latency*** (milliseconds): time from stimulus to peak onset of the fastest fibre. Motor nerve latencies include the additional delay the action potential experiences passing through the neuromuscular junction. Normal indices must take into account a standard distance (such as 80 mm for median nerve). Increased latency indicates a pathophysiological cause, for example: increased motor median nerve latency is found in carpal tunnel syndrome

- ***Amplitude*** (µ-mV): sum of local action potential amplitudes, measured from baseline to negative peak (Note: recording electrode polarity convention dictates negative peaks point upwards). Since the waveform is a summation, the peak shape includes range of speeds and number of axons recruited. Area under curve is reduced under pathophysiological conditions, indicating compression or lesion

- ***Velocity*** (metres/second): directly recorded action potential velocity in sensory fibres. The differential from two recording points is used for motor fibres, to ensure invariance to the neuromuscular junction delay

- ***F-wave response***: F-waves are low amplitude late responses due to antidromic activation of motor fibres, which then cause orthodromic impulses to pass back along the axon. Absence or delay may indicate a proximal pathology

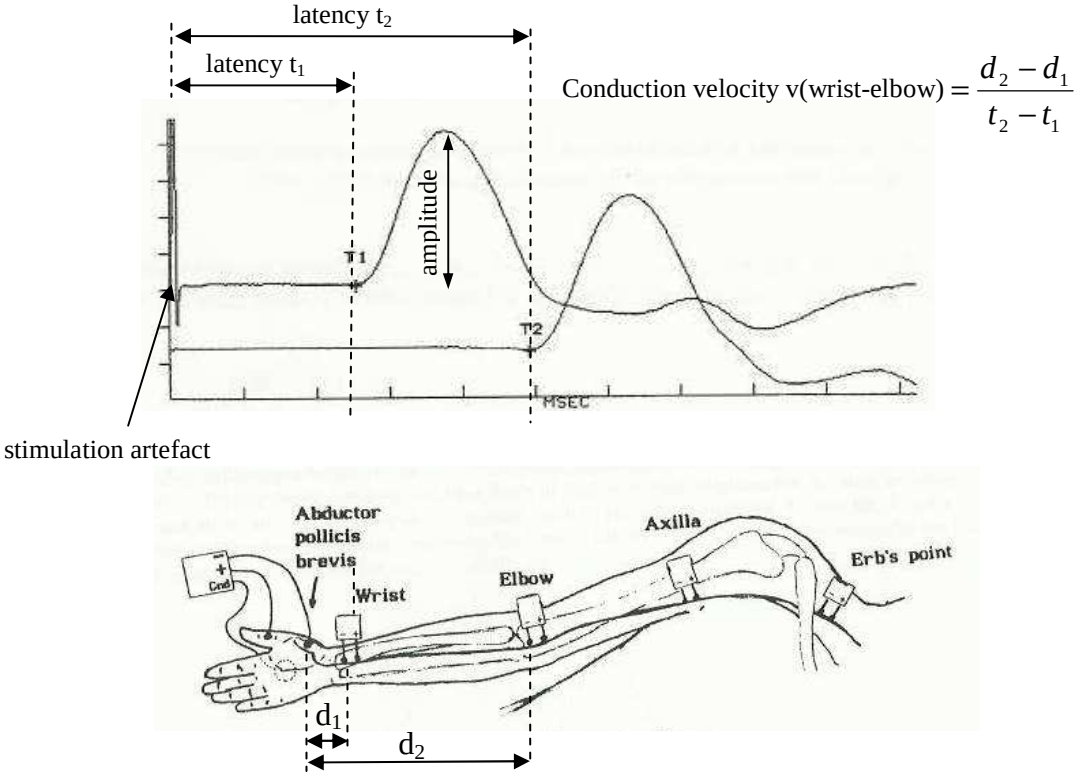


Figure 38 Recording of median motor nerve conduction velocity

3.3.3 Carpal Tunnel Syndrome

The incidence of Carpal Tunnel Syndrome (CTS) presented to the department warrants a weekly CTS clinic. The median nerve runs through the carpal tunnel with the flexor tendons (see detail overleaf). The nerve can become compressed when this space reduces, resulting in parasthesias and numbness in the thumb (I), fingers II & III and the lateral aspect of finger IV. A carpal tunnel release is an outpatient procedure to cut the carpal ligament. ENG is the primary method of pre- and post-surgical assessment, which aims to compare deviation of NCV from a statistically normal sample. Carpal tunnel syndrome presents with:

- Slowing of median nerve sensory conduction velocity across the carpal tunnel (> 45.0 m/s in normal cases)
- Prolonged distal latency of the median motor nerve (<4.2 ms in normal cases)
- Low amplitude of the median sensorineural action potential, (>20.0 µV in normal)
- Low amplitude of the median compound motor action potential, (>4.0 mV in normal)
- Spontaneous potentials in the ABP muscle (fibrillations or PSWs)
- F-wave response is not significant in CTS

Severity is represented by the scale:

Mild – median sensory nerve conduction slowing and/or median sensory amplitude decreased by more than 50% of reference value (no motor involvement)
Moderate – median sensory and motor slowing, and/or SNAP amplitude less than 50% of the reference value
Severe – absence of median SNAP with motor slowing or median motor slowing with decreased median motor amplitude or CMAP abnormalities with evidence of axonal injury from EMG testing of the thenar muscles

3.3.4 Conduction velocity testing for carpal tunnel syndrome

General nerve conduction testing is carried out at St George's Hospital by Consultant Neurophysiologists and forms a small part of a wider electrodiagnosis, during which assessment can be made for CTS. Specialist Practitioner Physiologists also hold a regular CTS clinic. The test protocol is as follow:

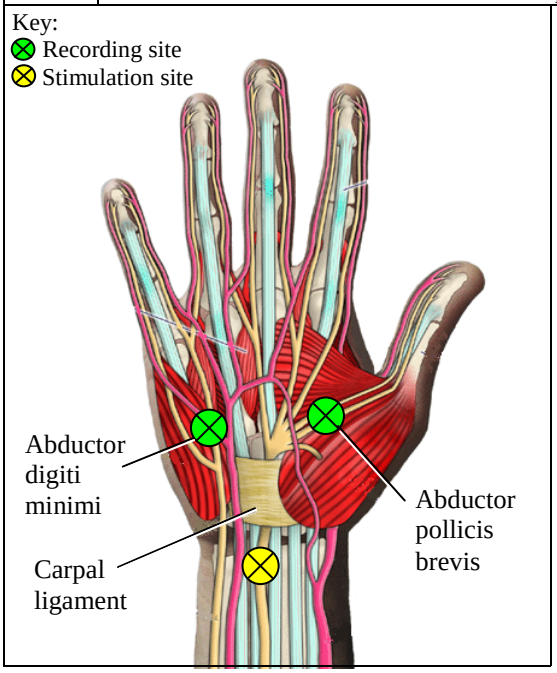
- Document current patient history, diabetes, muscle wasting and current medication
- Check patient skin temperature. If cold, place hands (or feet) in a warm water bath to elevate temperature above 32^{o}C.
- Collect data from both sides of the body, comparing results with the following department normative tables.

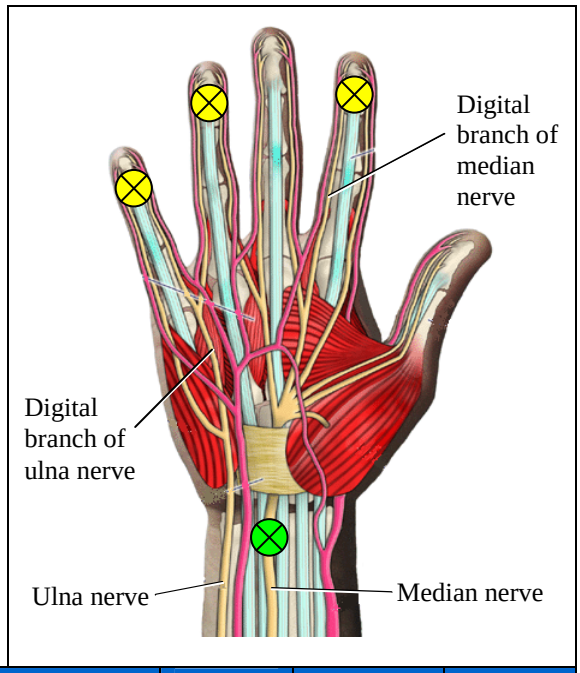
Since the ulnar nerve enters the distal region of the hand through a different canal (Guyon's canal), comparison between median and ulnar nerve conduction properties is a useful technique CTS evaluation.

The recording and stimulation sites and normative values are summarised below.

Table 16 Sensory and motor nerve conduction parameters

Test	Extremity	Nerve	Active Electrode	Stimulation sites	Onset latency (ms)	Amplitude (mV)	Velocity (m/s)
1	Upper right (motor)	Median	Abductor pollicis brevis	Wrist	< 4.2	> 4.0	> 50.0
2	Upper right (motor)	Ulnar	Abductor digiti minimi	Wrist	< 3.4	> 4.0	> 50.0
3	Upper right (motor)	Ulnar	Abductor digiti minimi	Above Elbow	< 3.4	> 4.0	> 50.0
				F-latency (ms)			
4	Upper right (motor)	Median	Abductor pollicis brevis	Wrist 29.1± 2.3 Elbow 24.8 ± 2.0 Axilla 21.7 ± 2.8			
5	Upper right (motor)	Ulnar	Abductor digiti minimi	Wrist 30.5 ± 3.0 BE 26.0 ± 2.0 AE 23.5 ± 2.0 Axilla 11.2 ± 1.0			
6	Repeat for left side						





Test	Extremity	Nerve	Active Electrode	Stimulation sites	Onset latency (ms)	Amplitude (µV)	Velocity (m/s)
7	Upper right (sensory)	Median digit 2	Wrist	2nd digit	< 3.5	> 20.0	> 45.0
8	Upper right (sensory)	Median digit 3	Wrist	3rd digit	< 3.5	> 20.0	> 45.0
9	Upper right (sensory)	Ulnar digit 5	Wrist	5th digit	< 3.1	> 18.0	> 44.0
10	Repeat for left side						
11	Upper right (sensory)	Median	Wrist	Palmer	< 1.9	> 20.0	> 45.0
12	Upper right (sensory)	Ulnar	Wrist	Palmer	< 3.1	> 18.0	> 44.0
13	Repeat for left side						

3.3.5 Limitations of ENG

The following are limiting factors in nerve conduction studies:

- Fastest fibre first:– since the latency of the earliest response to a stimulus is used in the calculation of velocity, this measurement reflects the conduction velocity of the fastest motor fibres only. This indicates that motor latencies and conduction velocities are not indicators of motor fibre loss. The fastest fibres have the largest diameters which produce the largest electrical fields compared to smaller fibres. A complete loss of small fibres will result in a false positive recording since one healthy fast fibre will provide the acceptable data

- Temperature:- as mentioned previously, the temperature should be kept at or below 32^0C. Latency is increased by 0.2 ms / deg.C., amplitude is increased with cooling and conduction velocity is decreased 1.8 - 2.4 ms / deg.C. Temperature is not currently corrected for at St George's Hospital. Correction factors have been published [32]

- Measurement errors:- measuring subcutaneous landmarks from surface features is a notoriously difficult task [33]. The problem in ENG is further broken down into:

    - Identifying the location and course of a nerve:- it is impossible without imaging techniques to manually identify nerve pathways. The task relies on experience and is assisted by observing changes to evoked response waveforms.
    - Accounting for abnormal anatomy:- the above is particularly true for abnormal anatomy. However there are a range of anomalies that neurophysiologists are aware of, such as the Martin-Gruber anastomosis in which fibres from the median nerve cross the forearm and join with the ulnar nerve.
    - Measuring surface distances:– tape measures are used to provide distance measurements for the velocity calculation. Smaller distances have increased overall error (for example: a fixed 5mm measurement error introduces a 5% overall error on a 100mm measured length, but 10% error if the measured length is 50mm). This is particularly relevant to mobile nerves: the ulnar nerve across the elbow is taught when the arm is flexed and slack with extended, measurements must reflect this. Some new automated NCV systems incorporate fixed acrylic electrode mounting templates, designed for specific investigations, which account for distance calibration.
    - Identifying stimulation sites:- the above location uncertainties are equally applicable to identifying stimulation sites and intensity of stimulus.
    - Subcutaneous tissue depth:- the evoked response amplitude does not vary significantly in patients with normal adipose tissue layer thickness. But for obese patients, the amplitude varies with applied pressure [30]. There are no protocols or system in place at St George's Hospital, to take this variation into account.
    - Inter-observer variability:- due to the reliance on experience and personal approach NCV studies are susceptible to measurer variability [34].
    - Low signal-to-noise ratio: Due to noise induced in hardware.

3.4 CASE STUDIES

3.4.1 Case Study JK – Carpal Tunnel Syndrome

JK is a 69 year old male with numbness and weakness in both hands. This suggested sensory and motor involvement and concentrated the area of investigation to the media and ulnar nerves of both hands. ENG was carried out to assess sensory conduction velocities between: median F(finger)2 and wrist, median F3 and wrist, ulnar F5 and wrist, median palm and wrist and ulnar palm and wrist, left and right hands. Motor conduction latencies were also assessed at the abductor pollicis brevis (ABP) and abductor digiti minimi (ADM) muscles, with stimulation at the wrist and elbow, for median and ulnar nerves, both hands. EMG was conducted at the thenar eminence.

Results: Table 18 shows the results for the motor nerve, Table 18 shows results for the sensory nerve bundle. The values highlighted in red demonstrate the characteristic features of carpal tunnel syndrome. EMG demonstrated very slight positive sharp wave muscle activity. JK has moderate carpal tunnel syndrome in both hands.

Table 17 Summary of upper motor results

Nerve	Side	Stim	Record	ΔDist (mm)	LatOn (ms)	Normal LatOn	CV (m/s)	Normal CV	P-PAmp (mV)	Normal P-PAmp
Median	Right	Wrist Elbow	ABP	80 265	4.83 10.08	< 4.2	 50.5	 > 50.0	6.48 5.48	> 4.0 > 4.0
Ulnar	Right	Wrist B. Elbow	ADM	 310	2.25 8.33	< 4.2	 51.0	 > 50.0	11.90 11.37	> 4.0 > 4.0
Median	Left	Wrist B. Elbow	ABP	 265	4.33 9.50	< 4.2	 51.3	 > 50.0	5.96 5.92	> 4.0 > 4.0
Ulnar	Left	Wrist B. Elbow	ABP	 295	2.58 8.33	< 4.2	 51.3	 > 50.0	11.75 11.91	> 4.0 > 4.0
Median	Left	Wrist B. Elbow	ABP	 280	4.33 9.83	< 4.2	 50.9	 > 50.0	5.53 4.13	> 4.0 > 4.0
Median	Right	Wrist B. Elbow	ABP	 295	4.58 10.00	< 4.2	 54.5	 > 50.0	7.38 6.03	> 4.0 > 4.0
Median	Right	Wrist B. Elbow	ABP		4.50 4.50	< 4.2			7.06 8.21	> 4.0 > 4.0

Table 18 Summary of upper sensory results

Side	Stim	Dist (mm)	LatOn (ms)	Normal LatOn	CV (m/s)	Normal CV	P-PAmp (μV)	Normal P-PAmp
Right	Med F2 - Wr	130	3.58	< 3.5	36.3	> 45.0	5.50	> 20.0
Right	Med F3 - Wr	135	3.48	< 3.5	38.8	> 45.0	5.20	> 20.0
Right	Uln F5 - Wr	120	2.42	< 3.1	49.7	> 44.0	8.52	> 18.0
Right	Med Plm - Wr	90	2.33	< 3.5	38.6	> 45.0	37.01	> 20.0
Right	Uln Plm - Wr	85	1.42	< 3.1	60.0	> 44.0	20.38	> 18.0
Left	Med F2 - Wr	135	3.40	< 3.5	39.7	> 45.0	0.28	> 20.0
Left	Med F3 - Wr	140	3.42	< 3.5	41.0	> 45.0	0.45	> 20.0
Left	Uln F5 - Wr	115	2.27	< 3.1	50.7	> 44.0	7.53	> 18.0
Left	Med Plm - Wr	90	2.37	< 3.5	38.0	> 45.0	32.34	> 20.0
Left	Uln Plm - Wr	85	1.40	< 3.1	60.7	> 44.0	20.19	> 18.0
Right	Med F2 - Wr	135	2.82	< 3.5	47.9	> 45.0	10.67	> 20.0
Right	Med F3 - Wr	140	3.22	< 3.5	43.5	> 45.0	3.85	> 20.0
Right	Uln F5 - Wr	105	1.83	< 3.1	57.3	> 44.0	6.09	> 18.0
Right	Med Plm - Wr	90	1.90	< 3.5	47.4	> 45.0	36.75	> 20.0
Right	Uln Plm - Wr	85	1.48	< 3.1	57.0	> 44.0	14.98	> 18.0

3.4.2 Case Study IM – Structural Lesion / Partial Seizure (EEG)

IM is a 45 year old man who has been observed by his work colleagues to have had a number of episodic events at work. The events are affecting his work and he has been advised by his local hospital not to drive. He was referred to the Neurophysiology Department from his local hospital Neurology Department, for a sleep deprived EEG. He has no known history and has also been referred for MRI and a neuropsychological assessment. The referral queries temporal lobe seizures as the cause of the events. EEG Assessment: IM was advised not to sleep the night before the test and have a friend bring him to St George's Hospital. He was alert, cooperative and sleep deprived. The standard montage was used. The waking record over the temporal regions is masked by muscle artefact. The EEG is of low amplitude but responsive, symmetrical alpha waves can be seen at 9-11Hz. Hyperventilation was performed well for 3 minutes and produced an increase in the amount of alpha rhythm. There are frequent single and runs of sharp and slow waves in the right anterior to mid temporal regions during drowsiness (shown below). The findings are consistent with a structural lesion and partial epilepsy. See Appendix M for a longer representative sample of this activity.

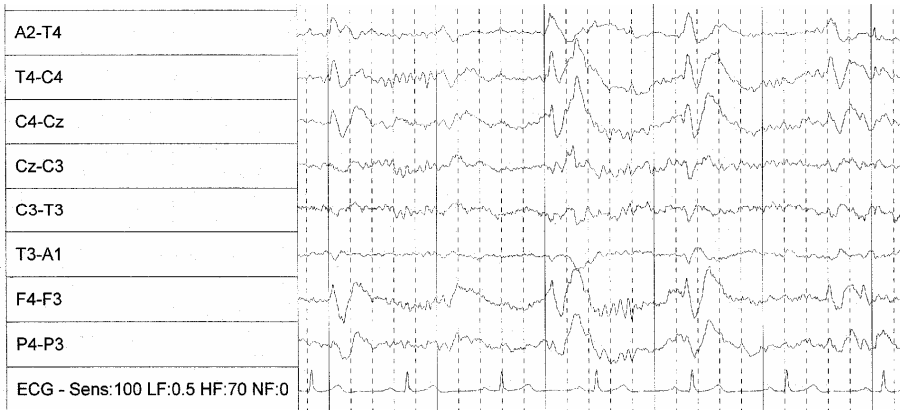


Figure 39 Section of EEG trace from IM showing abnormal sharp and slow waves in right anterior and mid temporal regions. (Note: Even electrode numbers relate to right hemisphere)

3.4.3 Case Study PB – Absence seizure (EEG)

PB is a six year old boy admitted to St George's Hospital Paediatric Intensive Care Unit (PICU) after experiencing ongoing absence seizures. The Neurophysiology Department were asked to provide an EEG record at his bedside to provide data for his seizure management. The seizures were being managed by clonazepam. Each time PB was brought into a state of consciousness, under observation, a non-clinical seizure followed. This was managed by returning PB to a pharmacologically controlled state. EEG was collected and two electrodes were placed on his clavicles to record ECG. Recordings normally last approximately 20 minutes. PB experienced non-clinical absence seizures when the investigation began, they came and went almost continuously until the investigation was ended by the Consultant by returning PB to a medicated state. A section of the EEG trace is shown overleaf (Figure 37). This portion follows on from a seizure, so what appears to be myogenic at the start of the trace is focal activity around T4. The main section shows characteristic generalised absence seizure discharges. These data confirmed the presence and frequency of the seizures. It was expected that if PB did not respond to medication, he would be diagnosed as being in status epilepticus.

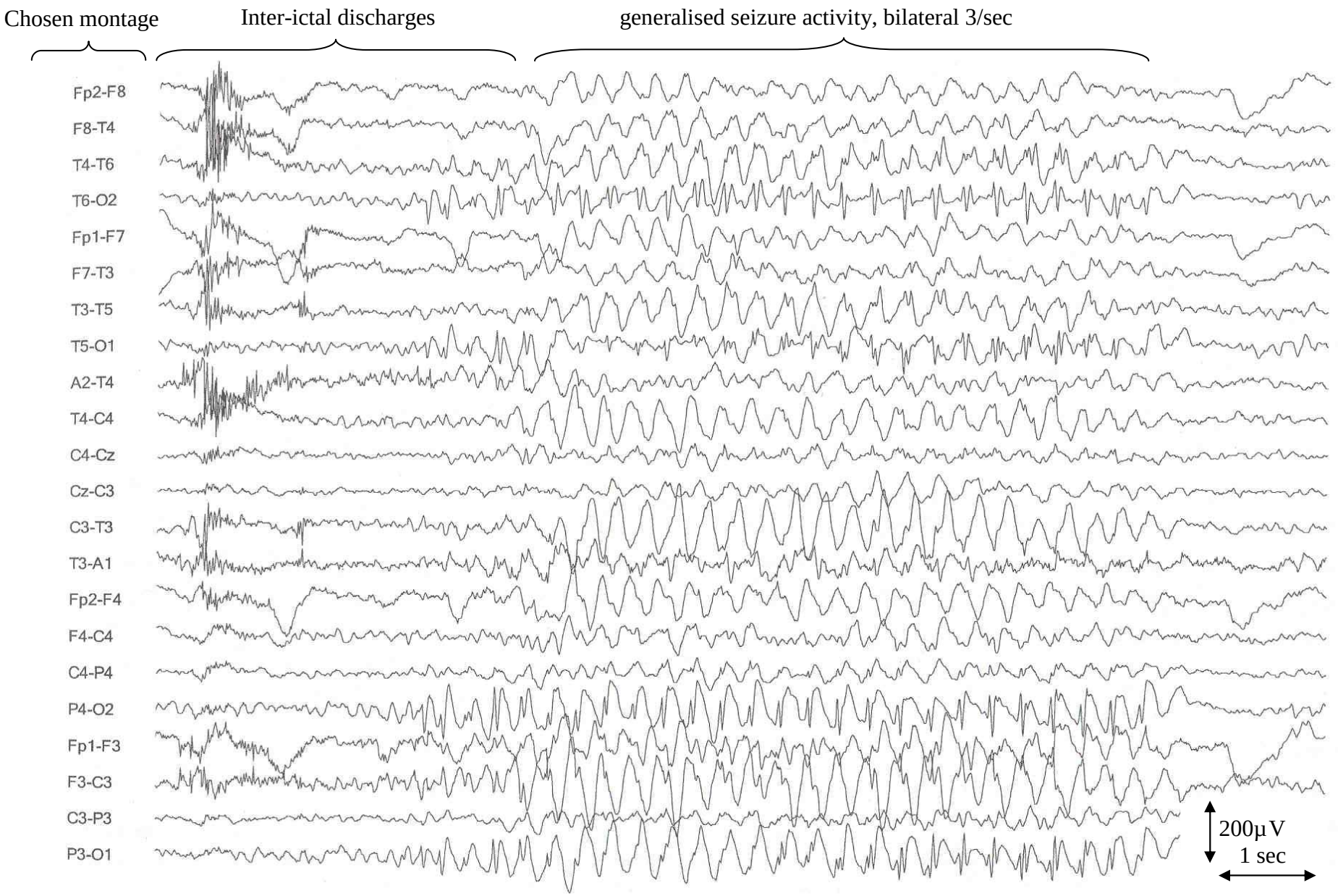


Figure 40 Case 1 – EEG trace indicating absence seizure

4. BLOOD PRESSURE MEASUREMENT

4.1 Introduction

This section of the portfolio discusses my work on the measurement of arterial blood pressure, carried out in the last two weeks of the placement. I reviewed the protocols used in the Neonatal Unit and carried out a practical investigation of catheter line dynamics.

4.2 Arterial Blood Pressure

For the purposes of physiological measurement, blood pressure is measured as a gauge pressure, in millimetres of mercury. Note: absolute pressure = gauge pressure + atmospheric pressure (atmospheric pressure is 760mmHg = 1 atmosphere = 101kPa). The following are physiological parameters of interest:

- Systolic pressure (SP) is the maximum aortic pressure following left ventricular ejection, typically 90-120mmHg.
- Diastolic pressure (DP) is the lowest pressure in the aorta just before the ventricle ejects blood, typically 60-90mmHg.
- Pulse pressure (PP), is the difference between systolic and diastolic pressures, typically 0-60mmHg.
- Mean arterial pressure $MAP = \frac{SP + (2 \cdot DP)}{3}$ is the average pressure in the arterial system during complete cardiac cycle, typically, 70-100mmHg.

As a rule-of-thumb, healthy SP/DP and PP are represented by the ratios 3:2:1. For example: 120/80 and 40mmHg, giving an MAP of 93. Referring to Figure 41, MAP is regulated by systemic vascular resistance (excluding pulmonary vasculature) and cardiac output. Cardiac output = heart rate x stroke volume. Stroke volume is influenced by inotropy (ventricular contractility) and preload (which refers to initial stretching of myocytes prior to contraction).

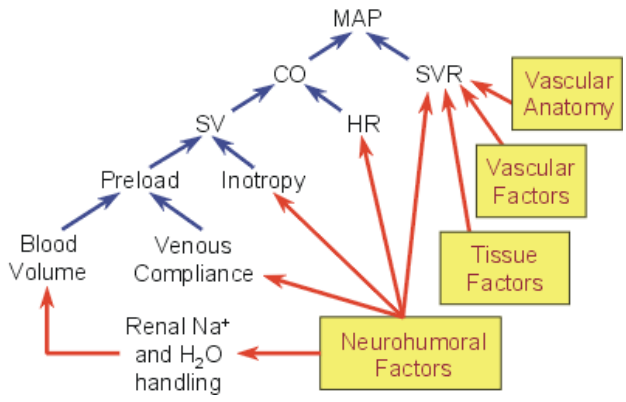


Figure 41 Factors influencing mean arterial blood pressure

The governing relationship:

$\frac{\Delta P}{\Delta V} = R$, where:

ΔP = the driving pressure $(P_A - P_V)$

ΔV = volumetric flow rate

R = systemic vascular resistance, which increases with blood viscosity (η), vessel length (L) and is inversely proportional to vessel radius[4].

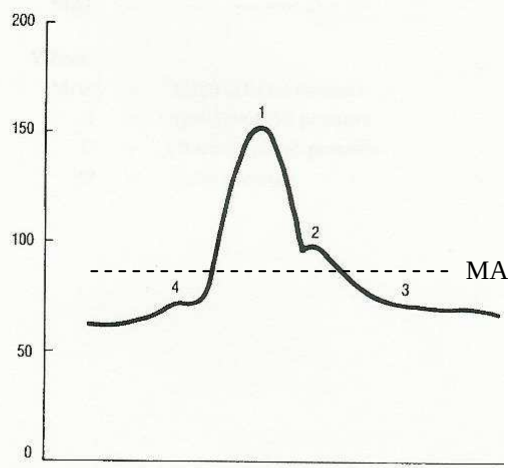
Gives **Poiseuille's equation:**

$$Flow \propto \frac{\Delta P \cdot r^4}{\eta L}$$

According to Poiseullie's equation, a reduction in vasculature radii (as blood travels distally), increases resistance to flow. However the systolic pressure is higher in peripheral arteries than in the aorta, due to reflections and constructive interference of the pressure wave. The diastolic pressure reduces with distance from the heart and the MAP remains fairly constant and is representative of perfusion pressure.

Changes to the pulse shape also occur under pathophysiological conditions. For example: a stenotic aortic valve is presented as a reduction in systolic pressure, when held against ECG the peak is delayed and the dicrotic notch is sometimes ill-defined. A regurgitating valve is shown as a high peak (to account for the increased volume of backflow ejected) and a wide pulse pressure. Continuous ABP monitoring also provides information about cardiovascular status. For example: atrial fibrillation can be recognised in the ABP waveform by a shortened diastolic filling time and a decrease in systolic amplitude.

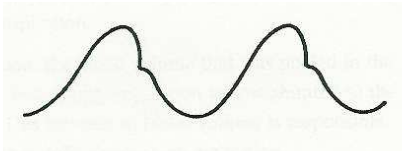
The normal ABP waveform:



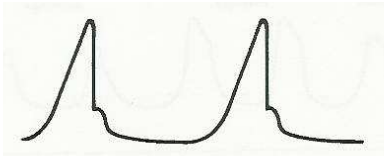
Features:

1. Peak systolic pressure
2. Dicrotic notch (aortic valve closure)
3. Diastolic pressure
4. Anacrotic notch (during isovolumetric contraction, a presystolic rise in pressure may be seen)

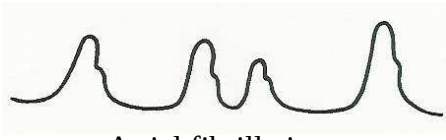
Pathological ABP waveforms:



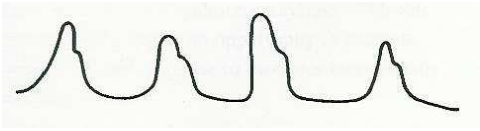
Aortic stenosis



Aortic insufficiency



Atrial fibrillation



Pulses Alterans

Measurement methods are divided into non-invasive and invasive types. Non-invasive methods tend to be less accurate, provide global pressures, but are quicker, require less training and are more acceptable to the patient. Invasive methods access blood vessels, providing accurate local recordings of wave shapes. Continuous invasive monitoring requires specialised training and equipment and is of great value in intensive care units.

4.2.1 Non-invasive blood pressure measurement

Sphygmomanometer:- (from the Greek *sphygmus* - pulse, *manometer* - pressure meter). The device was simplified by Riva-Rocci and has been the gold standard for non-invasive measurement since introduced in 1896. A cuff is connected to a mercury (or aneroid) manometer and inflated around the upper arm, occluding brachial artery blood flow. A valve on the inflation bulb is slowly released to allow blood to resume flow into the artery. As the lumen expands, turbulent flow causes vibration of the compliant vessel walls, generating audible sounds known as Korotkoff sounds. Five distinct sounds can be heard through a stethoscope placed against the artery, in a process called auscultation. Pressures are read from the height of mercury as the sounds are heard. The first sound corresponds with systolic pressure, the last sound (quietness) corresponds with diastolic pressure. Under conditions of high systemic vascular resistance, increased wall tension can diminish the amplitude of Korotkoff sounds, leading to false measurements.

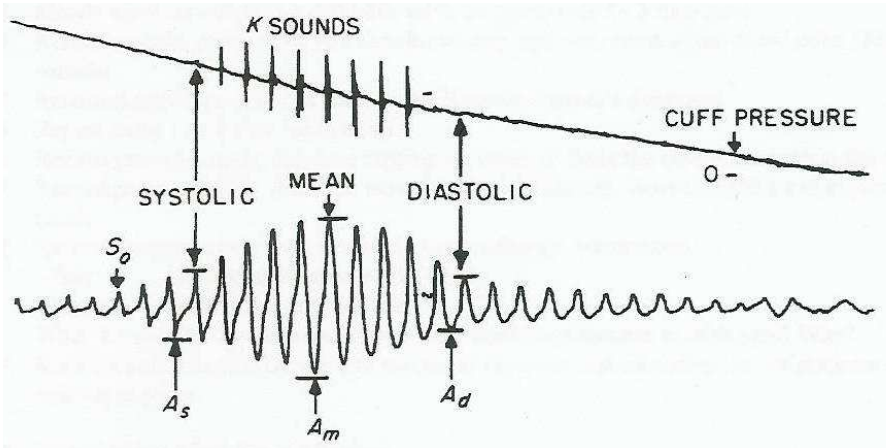


Figure 42 Korotkoff sounds superimposed onto cuff pressure (above), cuff oscillations (below)

Oscillometric method:- An automated digital device that operates on a similar principle to the sphygmomanometer. During vessel occlusion, the pulsatile flow of blood alters the volume of the inflated cuff surrounding the arm. The corresponding mean pressure variation is measured and diastolic and systolic pressures are calculated using an algorithm (which may vary between manufacturers). Oscillometric devices generally inflate to a higher pressure beyond systolic than sphygmomanometers, this can cause discomfort for some patients. Oscillometric devices can be purchased and used without training and provide systolic / diastolic pressure and heart rate. CE marking is not a guarantee of the accuracy of consumer devices. The Blood Pressure Association is currently working with the Department of Health and the British Standards Institute to introduce accuracy testing standards [36].

4.2.2 Invasive blood pressure measurement

The most common invasive method employs non-compliant tubing to form a fluid conduction path from the blood volume to a transducer pressure gauge (Figure 43). A flush solution of saline provides the transmission medium within the tubing and is kept at a higher pressure than arterial pressure. This ensures no blood enters the fluid circuit and establishes a flush flow to help maintain a clean catheter tip. A 0.45% heparinised flush solution is commonly used to prevent clots at the tip.

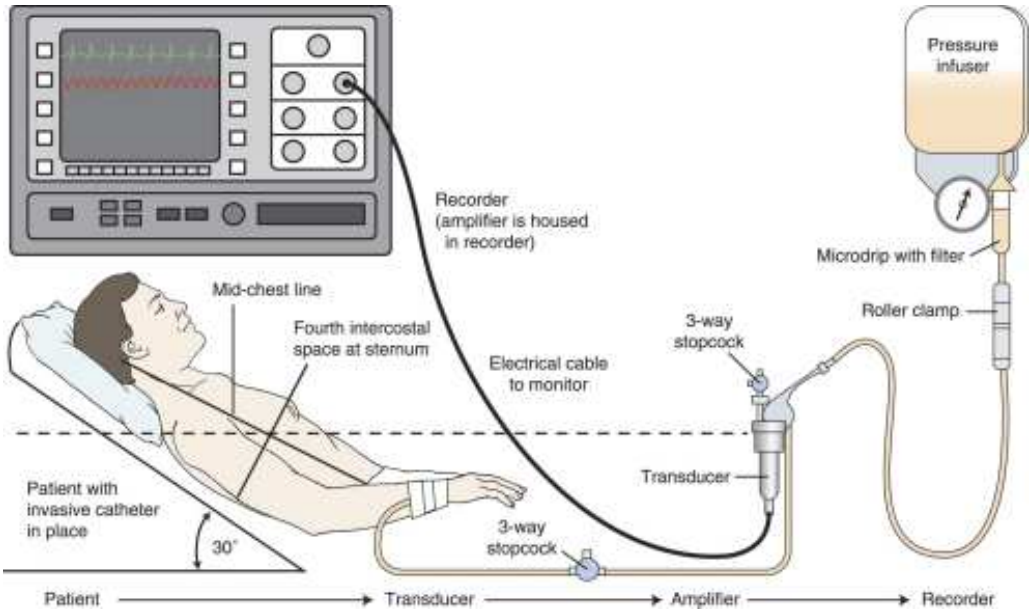


Figure 43 Complete arterial blood pressure monitoring system

The pulse pressure-wave deflects a compliant membrane within the transducer, which separates the sterile chamber from a strain gauge. The resistance change is measured with a Wheatstone bridge circuit, amplified and sent to a data recorder. A more expensive method places the transducer in the catheter tip, but the sterilisation process shortens this catheter lifetime. St George's Hospital favour fluid-filled disposable systems. Note:-

1. The flush solution bag is held on a pole 600mm above the transducer to provide a positive static pressure of 45mmHg (=1000 x 9.18 x 0.6 x 7.5). A pressurised bag can also be used to create the flushing pressure. In the neonatal ward at St George's syringe drivers are used to ensure precise control over flush volumes.
2. Non-compliant pressure tubing diameter sizes are standardised.
3. The transducer is zeroed at the *phlebostatic axis*, this is the precise point of origin of the hemodynamic pressure being measured, in most cases this is the heart [37], for an interesting discussion see [38]. Some new transducer stands incorporate laser pointers to help alignment with the axis.
4. A three-way tap is placed in series to gain access for medications, remove air bubbles and sample blood. A second tap is placed to prime and flush the system.
5. Non-vented caps are placed on unused ports to maintain a closed sterile system.
6. Colour coding is used for pressure line identification, red for ABP and blue for CVP.
7. It is vital to manually follow any tubing, to find out what is connected, before connecting or disconnecting any patient connected tubing (see section 4.6.3).
8. A system specification can be found in Appendix O.

4.2.3 Clinical Example - measurement in the Neonatal Unit

The Neonatal Unit (NNU) at St George's Hospital is the regional centre of excellence for South West London and the South East of England. Each year the Unit cares for over 500 new-born babies (up to 30 at any one time) in the intensive care, high dependency and special care nurseries. Many of the babies are there because they have been born prematurely, some mothers reaching only 24 weeks of pregnancy. I observed blood pressure measurements taken from babies on the unit, the following are pertinent observations supported by the NNU blood pressure measurement policy [39].

NNU Measurement Protocol

Clinical use:- Blood pressure is monitored in any sick baby, including those with urinary tract abnormalities, chronic lung disease, those on steroids and infants of drug addicted mothers. Cardiac babies, newborn infants suspected of having a cardiac abnormality are also monitored. Any baby on ventilatory support or FiO_2>30% has an indwelling arterial line for continuous BP measurement.

Data:- Systolic, diastolic and mean blood pressures are measured, for example:

Table 19 Example measurements taken from patients in neonatal unit

Parameter	**Baby 1**	**Baby 2**	**Baby 3**
Systolic	47.0	76.4	43.8
Diastolic	31.3	47.4	27.8
Mean	40.0	61.0	40.0
Heart rate	162.3	162.3	165.8

Good nursing practice is recognised by accessing the previous twelve-hour blood pressure trend and developing an awareness of the baby's response to handling and procedures.

NIBP:- The Unit use standalone portable ‘Neocare’ non-invasive blood pressure (NIBP) monitors and HP Merlin NIBP modules. I found mixed views regarding the use of oscillometric blood pressure monitoring in low birthweight neonates [40, 41]. The Unit only use oscillometric NIBP on very low birthweight babies when insertion of a UAC or peripheral arterial cannula has failed. Non-invasive blood pressure readings taken during activity or when the baby is active or distressed are inaccurate.

Mean arterial pressure is overestimated if the cuff width is too small and under estimated if the cuff width is too large, the width should be 75% of the distance between the axilla and the elbow or have a width 40-50% of the arm circumference. The lower leg may also be used, with similar cuff width restrictions.

Timer controlled intermittent blood pressure monitoring is performed with NIBP monitoring. However, the nurse must be vigilant in ensuring the baby is at rest during these recordings, the height of the centre of the cuff is at the same level as the heart and the cuff should not compress the limb when it is not pressurized. The cuff should be removed every three to six hours to inspect the arm for signs of pressure injury or skin irritation.

Invasive Measurement:- Invasive waveforms are given greater diagnostic merit over NIBP values. The Unit measures invasive blood pressure from the aorta, radial artery or post tibial artery. Access to the aorta is made using an umbilical artery catheter (UAC) and access to the arteries is made with a cannula. All invasive systems are fluid-filled, perfused with heparinised "0.45%"- or "half normal" saline. The setup process is as follows:

1. Gain arterial access and prepare skin site.

2. Zero transducer:
   - Elevation of the transducer from the site of measurement by 14mm will cause a pressure reduction of 1mmHg. Likewise a lower sensor will cause a false high reading. For this reason the transducer is maintained at the phlebostatic axis, (if measuring from the heart the axis is defined as the 4th intercoastal space, midway between the anterior-posterior aspects of the chest). If measuring from a different location, position the sensor at that level. Open the transducer port and zero the patient monitor. Zeroing is performed by nursing staff when the line is first setup, at the beginning of each shift and after each procedure that requires the patient or monitoring system to be moved.

3. Prime line:
   - Attach syringe driver (or flush solution bag in case of adult BP and half fill drip chamber) and set to 30ml/hr.
   - Close distal valve, open nearest proximal valve
   - Allow fluid to fill section of tube
   - Open next nearest valve and allow fluid to fill next section of tube
   - Continue to patient ensuring no air bubbles enter the tubing
   - Fit distal tip of tubing to patient's arterial cannula, lock the luer lock.

Catheter system calibration aims to ensure high data accuracy by setting the system to operate within the designed linear stable region. Transducer linearity and range is assumed by nursing staff and can be checked by ICU technicians with an aneroid manometer if required.

4.3 Safety

The major risks associated with blood pressure monitoring are:

1. Nosocomial infections - those received by the patient whilst in hospital (iatrogenic). A path for expedient cross-contamination could exist in the handling and use of invasive devices. For this reason the invasive monitoring kits are sterile and opened using an aseptic technique. The tubing is flushed and fluids not permitted to escape. Needleless systems and one-handed capping procedures reduce risk of needlestick accidents.

2. Tubing misconnections arise through lack of attention and the verisimilitude of tube connectors. The MHRA and FDA have reported these errors: an enteric feeding tube connected to an IV catheter, CV catheter, peritoneal dialysis catheter and a hemodialysis line. Injection of barium sulphate into a CV catheter. A NIBP insufflator tube connected to an IV catheter and to a needleless IV port. Oxygen

tubing to a needleless IV port [42]. Also a batch of transducers with a flush flow rate suited to neonates (30ml/hour) was used in adults. Equipment colour coding and good nursing practice should be in place to reduce incidence of these errors.

3. Inaccurate patient data could lead to incorrect changes to the patient's management programme.

4. The MHRA reviewed the use of mercury in sphygmomanometers and concluded that while aneroid sphygmomanometers are preferable (and should be calibrated with a mercury device) mercury devices can be used within a controlled and safe working environment [43].

4.4 INVESTIGATION - Arterial catheter-transducer dynamics

Background: The ABP fundamental frequency range is 1 – 3Hz. Six harmonics are considered sufficient to capture the waveform [25], (Figure 44). If the heart rate increases to 180 beats per minute during a tachycardia episode, the highest frequency component will be 18Hz. In addition, the fluid-filled catheter-transducer line behaves as a second order system, with inertial, friction and elastic properties. The natural resonant frequency and damping factor are, respectively:

$$f_n = \frac{r}{2}\left(\frac{1}{\pi\rho L}\frac{\Delta P}{\Delta V}\right)^{1/2} \quad \text{eqn.1} \qquad \zeta = \frac{4\eta}{r^3}\left(\frac{L\left(\Delta V/\Delta P\right)}{\pi\rho}\right)^{1/2} \quad \text{eqn.2}$$

A derivation of these models can be found in [12].

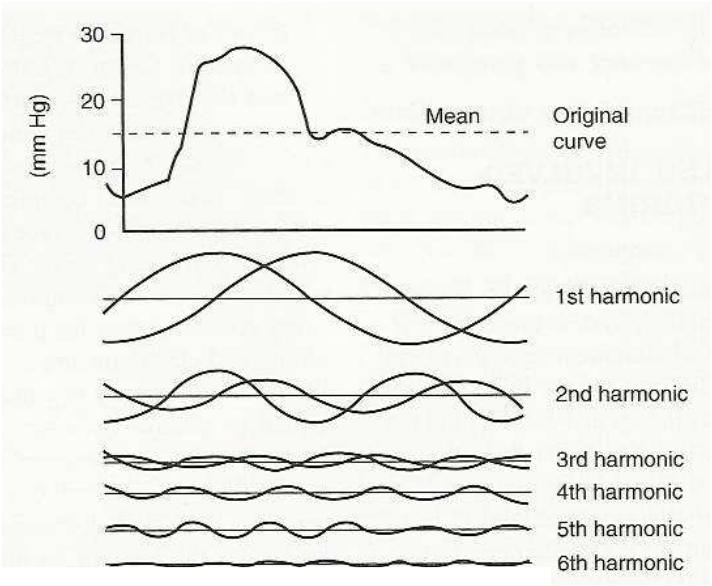


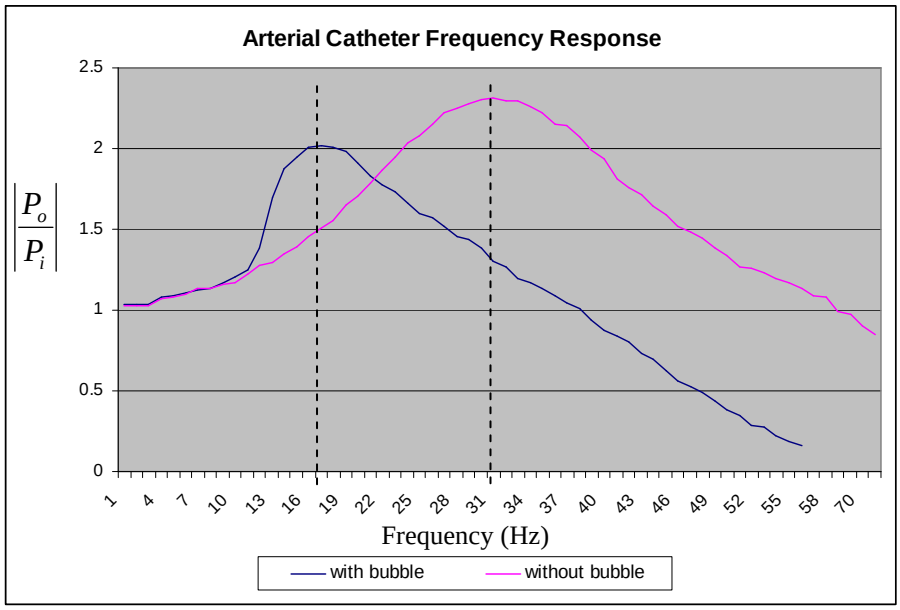
Figure 44 Arterial blood pressure waveform harmonics

To ensure accurate amplification of the pressure wave and prevent distortion, it is important to have a flat frequency response across the physiological bandwidth and below the resonant frequency of the measuring system. Most lines in clinical use are designed to be robust if a series of events occur at once, such as a hypertensive patient with tachycardia, monitored with long thin tubing. DC to 30-60Hz is a suggested maximum bandwidth [44].

I conducted the following experiment to examine the changes to resonance with the introduction of a compliant air bubble and demonstrates the importance of priming the system before use and ensuring the lines are maintained bubble-free during use.

Experimental method: I used a function generator to provide a sinewave input function, with a varying frequency from 1Hz to 60Hz, in 1Hz increments. This drove a moving coil piston (the Biotek Model 601A Blood Pressure Calibrator) to generate a pressure waveform. An Edwards Lifescience combined ABP/CVP pressure line kit was used to establish the water filled transducer circuit. French gauge 5 (1.67mm diameter) tubing was used. The gain and dc offset of a differential amplifier was calibrated with a mercury manometer and the output was displayed in millivolts on a PC, via a digital oscilloscope. The fluid circuit was setup from new, giving a true reflection of the configuration used on the wards. The fast flush input (normally connected to an IV bag) was sealed with a non-vented cap. Water was boiled and allowed to cool (to reduce bubble formation) and used to prime the system according to the standard protocol. The frequency response was plotted with and without a 5mm long air bubble. A syringe was used to inject an air bubble into the system through a 3-way stopcock.

Results: The meniscus and parallax error presented by the small diameter tubing made it very difficult to precisely measure the bubble length. For this reason, the following results present a graphical indication of the principles.



The resonant frequency was found directly from the data:

without bubble ω_n = 31Hz
with bubble ω_n = 17Hz

The damping factor (ζ) was found from the relationship: $\zeta = \frac{k}{2x10^{\left(M_{90}/20\right)}}$, where k is the DC gain, assumed to be 1 and M_{90} is the magnitude (in decibels) at 90^0.

without bubble ζ = 0.2166
with bubble ζ = 0.2475

Discussion: The resonant frequency is reduced by 14Hz, as expected from equation 1. Damping is increased by 0.0309, as expected from equation 2. The increase in damping and compliant action of the bubble was clearly observed by marking the boundaries of the bubble with a felt tipped pen on the tube, then reducing the input frequency to watch the motion of the bubble. In summary: If, through a careless technique, an air bubble enters a monitoring system, the resonant frequency will reduce and higher harmonics will be amplified nonlinearly, distorting the wave shape.

I went on to replicate the distortion an ABP waveform undergoes through overdamping and underdamping. This was achieved by varying the parameters expressed in equation 2: altering the tube length and diameter, using very compliant tubing (not designed for blood pressure measurement), by increasing resistance to flow by compressing the tube and by addition of bubbles of various number and length. The following waveforms are indicative of the results.

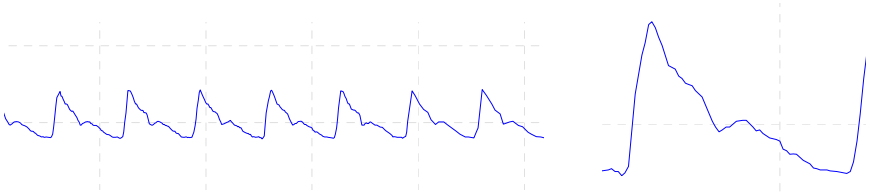


Figure 45 Optimally damped waveform, detailing enlarged section of trace. Note the clarity of the dichrotic notch.

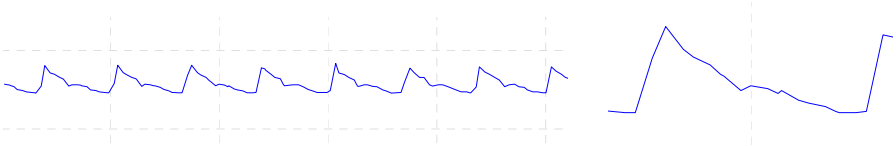


Figure 46 Over damped waveform, detailing enlarged section of trace. Note the reduction in amplitude and reduction in resolution.

Figure 47 Under damped arterial waveform, detailing enlarged section of trace, achieved by using 2m long tubing. Note the extenuation and additional features as a result of the added harmonics.

A transient response test is used to check the ability of the measuring system to respond to changing system dynamics. Traditionally the response is elicited by inputting a brief square wave from a function generator or from a burst surgical glove sealed over the top of an open chamber. I performed these tests using both methods and achieved the responses shown below.

Figure 48a Optimally damped - square wave test input

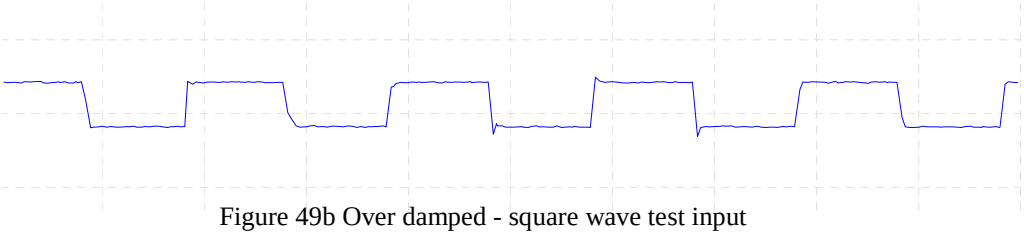
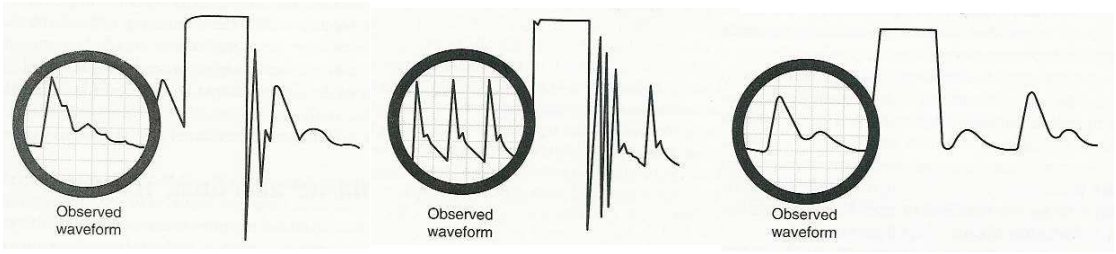


Figure 49b Over damped - square wave test input

Figure 50c Under damped - square wave test input, detailing enlarged section of trace

Monitoring systems on wards are attached to people inputting their own arterial pressure waves, so there is no constant steady-state baseline. In this instance an empirical observation is made. It is achieved by pulling and releasing the fast-flush tab to inject a unit step pulse. The resulting waveforms are observed on the bedside patient monitor. I performed this test on the adult Intensive Care Ward and also identified and removed air bubbles from a patient connected system.



Normal Underdamped Overdamped

Figure 51 Varying responses from flush test

Table 20 Causes and Effects of damping on measurement

Problem	Consequence	Causes
Overdamped Waveform	• Underestimates systolic and overestimates diastolic blood pressure • Mean arterial pressure remains the same	Air bubbles, compliant tubing, catheter kinks, blood clots, stopcocks, no fluid or low flush pressure
Underdamped waveform	• Overestimates systolic and underestimates diastolic blood pressure • Mean arterial pressure remains unchanged	Long tubing Increased vascular resistance.

5. ELECTRICAL SAFETY

5.1 Rationale

What effect does electricity have on the body? How can those effects be reduced and the risks managed in a healthcare system? Those questions will now be tackled.

5.2 Physiological Effects of Electricity

If the body forms part of an electric circuit a current will flow which is proportional to the voltage applied between the two contact points (skin surface electrodes for example) and inversely proportional to the complex impedances of the various tissues that complete the circuit.

The musculature and nervous system are the main physiological systems operating under ionic charge flow and therefore most susceptible to electrical disturbance. At very low currents (0.5mA at 50Hz; 2-10mA DC) [25] a tingling sensation can be felt as sensor nerve fibres are stimulated. This is called the '**threshold of perception'** and is known to any schoolboy who has touched a 9volt battery with their tongue, stimulating chemo-receptors. As the current increases, motor nerve fibres are increasingly recruited and stimulated. This may cause fatigue, discomfort and/or pain. Within the region of 6mA at 50Hz[25] the strength of involuntary muscle stimulation becomes greater than voluntary withdrawal actions. Here, the **'let-go current'** is defined as the maximum current at which the subject can voluntarily withdraw. If the back of the hand touches a conductor carrying the let-go current, there is less risk of physical injury, because the fingers will not wrap around and grip the conductor. With increasing current flow, involuntary muscle contraction affects the respiratory muscles (the diaphragm and intercostals), causing asphyxiation. Respiratory arrest, the cessation of normal tidal flow, has been shown to occur at currents of 18-22mA [25]. This is accompanied by increased pain and fatigue.
Gas filled lungs and bony ribs have the lowest conductivities in the body ($0.13Sm^{-1}$ and $0.04Sm^{-1}$ respectively, at 433Hz) [45], offering some protection to the intricate electrophysiology of the heart. When an externally applied current is sufficiently great to reach and stimulate part of the heart, the rhythmic wave of propagation becomes disrupted, resulting in a disorganised and tachycardic rhythm called **ventricular fibrillation**. The brain remains oxygenated for approximately 6-7 minutes before death occurs. Ventricular fibrillation is the major cause of death due to electric shock and takes place within the region of 75-400mA [25]. A defibrillator can be used to discharge a capacitor across the heart, applying a brief peak biphasic current of 50Amps at 7500volts [46]. A **complete myocardial contraction** will occur with the application of 1–6 Amps. This causes the entire heart muscle to contract and when the stimulus is removed, synchronised re-polarisation may force the heart back into a normal rhythm.
As the current increases beyond 10Amps, the high skin resistance at the electrode contact points cause resistive heating and skin burns. High current densities arise from small electrodes, exacerbating the effects. The nervous system fails to function and muscle contraction is so strong that muscles are ripped away from the bone. There is great variability across the population to these effects; small people with oily skin are more conductive than large people with dry skin and as such will experience effects at the lower end of the ranges quoted.

5.3 Fault Conditions

The following definitions are encountered in the design and use of medical electrical equipment.

A **macroshock** arises from an externally applied device presenting an unwanted current to the body. The current follows a high resistance path, a proportion of which may reach the heart. A **microshock** is experienced if a current follows a direct conductive path to the cardiac muscle via an applied part. An invasive catheter may create such a path. A microshock requires a lower current to cause ventricular fibrillation than a macroshock. The impact of the shock will vary with the path impedance of local skin and body structures, moisture level and the points of entry of the current.

A **single Fault Condition** is a "condition in which a single means for protection against a SAFETY HAZARD in EQUIPMENT is defective or a single external abnormal condition is present" [47]. A single fault condition may involve:

- A broken earth
- A broken neutral line
- A reversed polarity supply

If a single fault condition occurs, the risk of a macroshock is increased if a person touches the chassis of the faulty device. For this reason continuity of the ground wire must be tested periodically.

An **applied part** is: "a part of the EQUIPMENT which in NORMAL USE:- necessarily comes into physical contact with the PATIENT for the EQUIPMENT to perform its function; or - can be brought into contact with the PATIENT; or - needs to be touched by the PATIENT" [47]. The flow of energy between patient and device through an applied part may be bi-directional.

5.4 Safety Standards

Gaining an understanding of the fault paths and expected effects is the first step in managing risk. The next stage is to build safety into a set of standardised working practises. Safety is an international theme and the standards will inevitably become harmonised across the various continents, the following sections focus on Europe.

The Medical Devices Directive (MDD 93/42/EEC) [48], is one of three European Directives in force concerning medical devices and applies to medical devices not covered by the Active Implantable Medical Devices Directive (AIMDD 90/385/EEC) [49] or the In Vitro Diagnostics Directive (IVDD 98-79-EEC) [50]. The Medical Devices Directive is supplemented by 2000/70/EEC and 2001/104/EEC. These are embedded in UK law by Statutory Instrument No 618 (the Medical Devices Regulations 2002, MDR) [51] and by Statutory Instrument No 1697 (the Medical Devices (Amendment) Regulations 2003) [52]. The Commission Electrotechnique Internationale's 60601 standard is an authoritative standard harmonised across the United States, Canada, the European Union, Japan, Australia and New Zealand. For products within the European Union that comply with IEC60601 (or the UKs BS

EN60601) there is a “presumption of conformity” to the Medical Devices Directive 93/42/EEC. The IEC 60601 comprises four parts:

- The 60601-1 base standard is the core of the standard and with the collateral standards covers all of the general requirements for electrical medical products. For example collateral standard IEC60601-1-2 covers aspects of electromagnetic compatibility.
- The 60601-2 group are particular standards that include particular device-specific standards. For example collateral standard IEC 60601-2-2 covers high frequency surgical devices.
- The 60601-3 group are performance standards that cover performance requirements for specific devices. For example IEC60601-3-1 contains requirements for the performance of transcutaneous oxygen and carbon dioxide partial pressure monitoring equipment.

5.5 Classification of Medical Devices

BS EN 60601-1 defines three classes for medical electronic instrumentation:

Class	Definition	Designated Symbol
I	• Devices powered by an external source, provided with a reliable **protective earth** such as a complete metal enclosure that is protectively tied to the ground pin of a three pin power plug • A **fuse** is in place in the live path	No symbol
II	• As above, without a protective earth, relies on **double insulation** or **reinforced insulation** to protect against electric shock	
III	• Safety extra low voltage (SELV) devices • Externally powered <24$V_{a.c.}$ or < 50 V_{DC} • Not recognised as medical devices	No symbol

Figure 52 Classification of medical devices

The class designates the level of electrical protection a device has. Three further classification types exist for patient connected equipment or ‘applied parts’ that come into direct physical contact with the patient.

Type	Definition	Designated Symbol	
		Applied Parts	Defibrillation-proof applied parts
B	An internal or external connection (excluding direct cardiac connection)		
BF	Type B with a floating (isolated) input		
CF	Suitable for direct cardiac application		

Figure 53 Type classification of medical devices

5.6 Safety Testing

5.6.1 Visual inspection

70% of faults are identified during visual inspection [53]. The inspection includes:

- Housing / enclosure - look for damage or cracks.
- Cabling – look for cuts, misconnections, exposed wires, incorrect colour coding.
- Fuse rating – check value against device specification
- Markings and labelling – check the integrity
- Mechanical parts – check integrity or obstructions

The following measurements are then used to ensure a medical device complies with 60601 standards. Earth bond or ground bond testing, tests the integrity of the current path from the earth pin at the mains plug to any conductive surface on the device. If a fault condition occurs to cause the casing to become live, a low resistance path to earth will reduce the risk of shock if a person touches the case.

5.6.2 Earth leakage current measurement

The earth leakage current is "current flowing from the MAINS PART through or across the insulation into the PROTECTIVE EARTH CONDUCTOR" [47]. Leakage currents exist by virtue of capacitive coupling between the mains lines. Figure 54 shows the path of an earth leakage current through a grounded device. This assumes that the ongoing ground path from the device to the mains is unbroken. St George's Hospital require all extension leads to be tested for compliance to 60601.

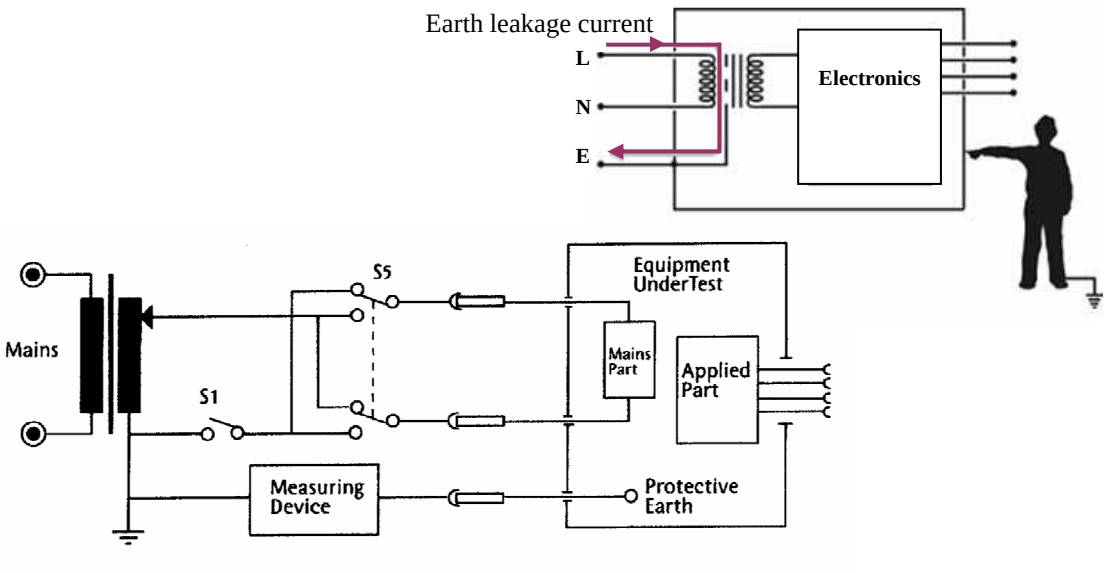
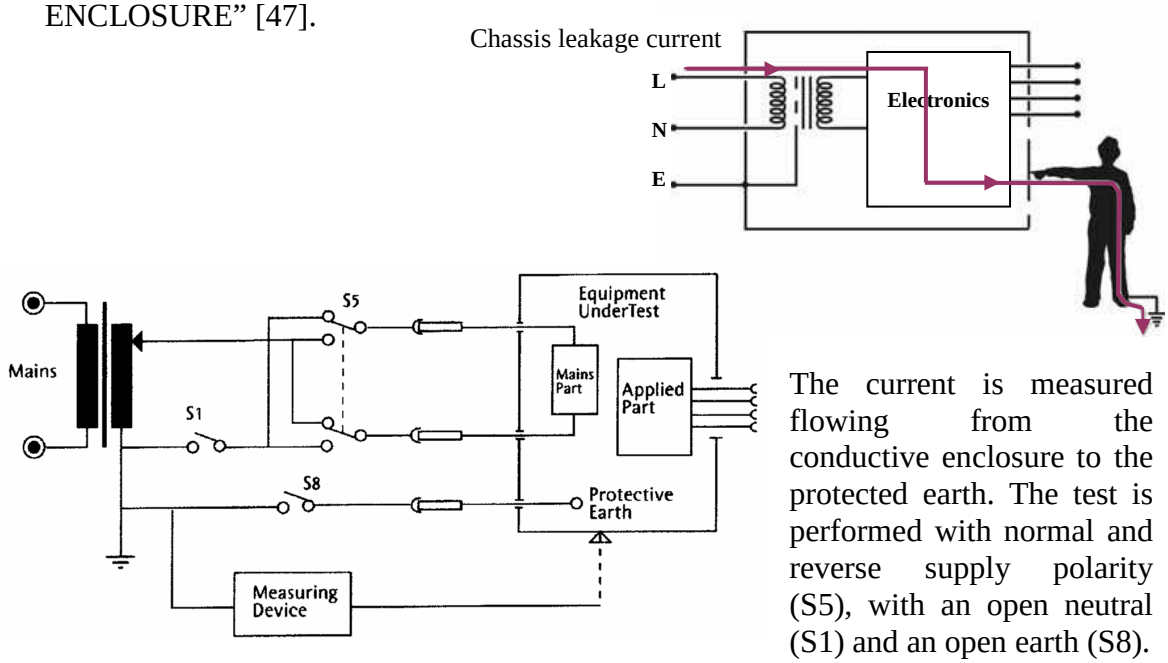


Figure 54 Earth leakage current and its measurement

The measuring device measures the current flowing through the device's protective earth conductor. The test is conducted under normal and a reverse polarity fault condition using switch S5 (Figure 54) and with an open neutral line fault condition using switch S1. A 25A test current is commonly used. Test limits are shown in Table 21.

5.6.3 Chassis (or enclosure) leakage current measurement

Enclosure leakage current is “current flowing from the ENCLOSURE or from parts thereof, excluding APPLIED PARTS, accessible to the OPERATOR or PATIENT in NORMAL USE, through an external CONDUCTIVE CONNECTION other than the PROTECTIVE EARTH CONDUCTOR to earth or to another part of the ENCLOSURE” [47].

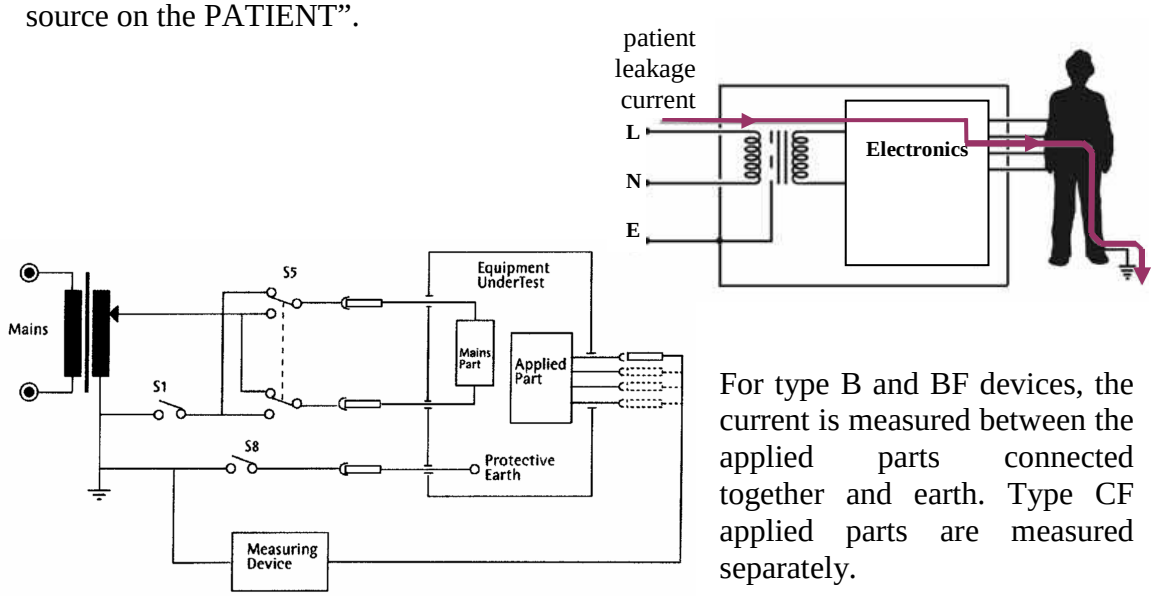


The current is measured flowing from the conductive enclosure to the protected earth. The test is performed with normal and reverse supply polarity (S5), with an open neutral (S1) and an open earth (S8).

Figure 55 Chassis (or enclosure) leakage current and its measurement

5.6.4 Patient leakage current measurement

Patient leakage current is “current flowing from the APPLIED PART via the PATIENT to earth, or flowing from the PATIENT via an F-TYPE APPLIED PART to earth originating from the unintended appearance of a voltage from an external source on the PATIENT”.



For type B and BF devices, the current is measured between the applied parts connected together and earth. Type CF applied parts are measured separately.

Figure 56 Patient leakage current and its measurement

The mains voltage supply is also connected to the applied parts and the leakage current that would flow from an external source into the patient circuits is measured. The purpose is to ensure there is no danger of electric shock to the patient who may be raised to a potential above the earth when connected to the applied parts of the device under test.

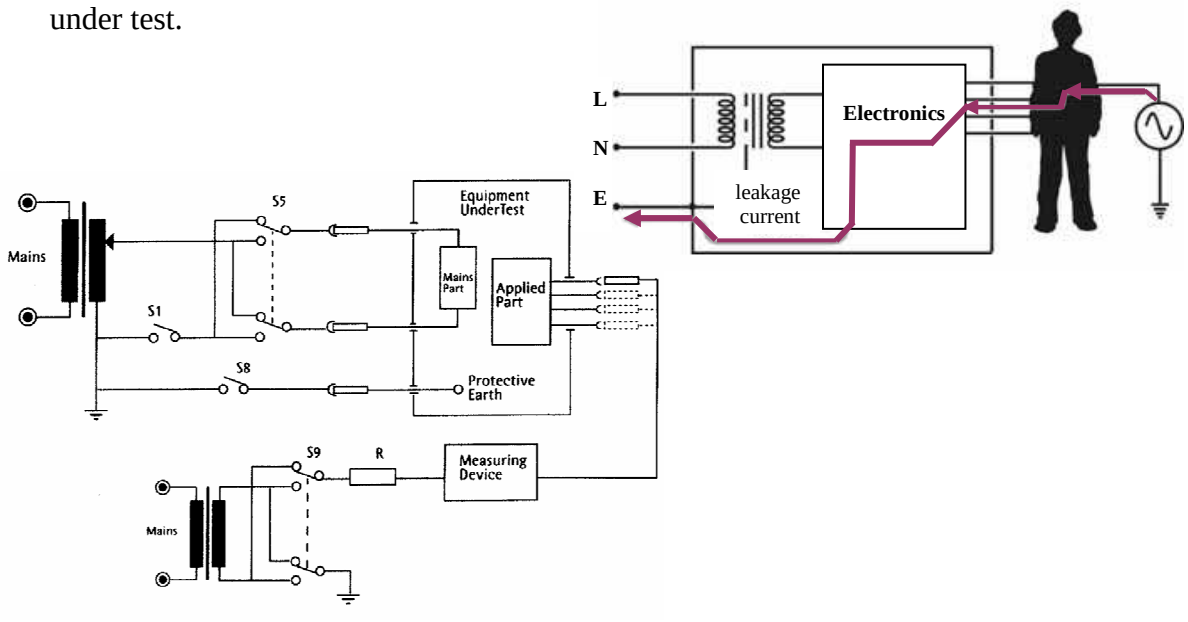


Figure 57 Mains on applied part patient leakage current and its measurement

5.6.5 Patient auxiliary current measurement

Auxiliary current is “current flowing in the PATIENT in NORMAL USE between parts of the APPLIED PART and not intended to produce a physiological effect”. Examples include the bias current of an amplifier or the current used in impedance plethysmography.

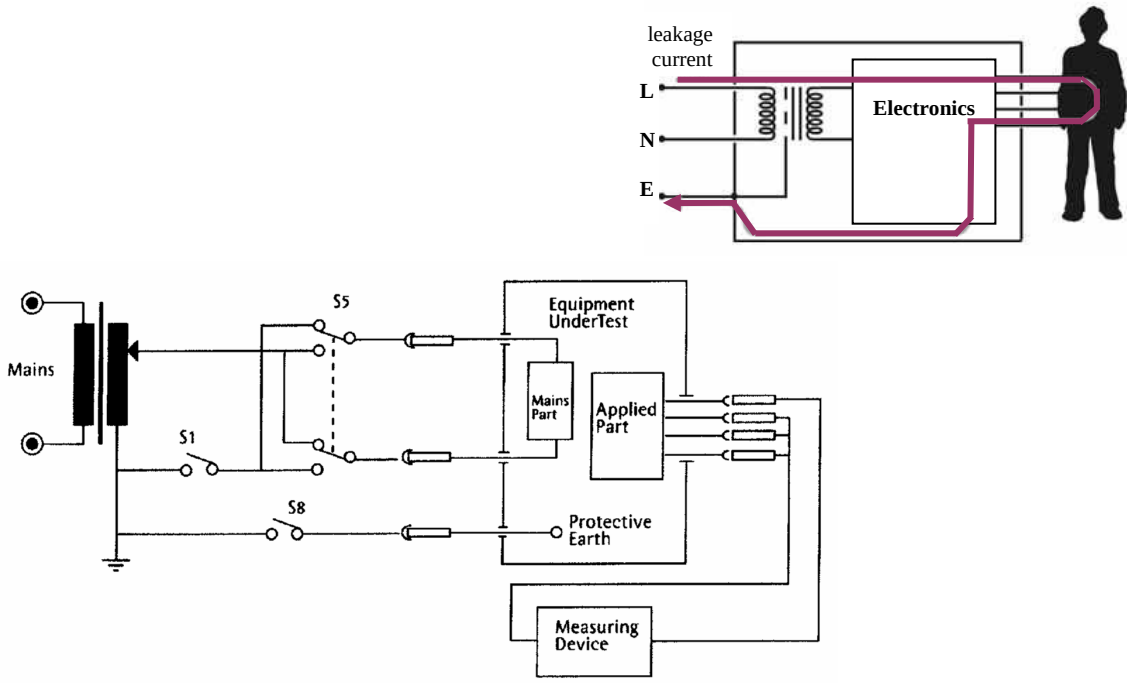


Figure 58 Patient auxiliary current and its measurement

The following table summarises BS-EN IEC60601 leakage current limits, (all values are given in mA). Note the earth leakage current limit occurs at around the threshold of perception. Auxiliary currents and those experienced in type CF applied parts are 50µV.

Table 21 IEC60601-1 Test Limits

Non-detachable power cord	<0.1Ω					
Detachable power cord	<0.2Ω					
	Type B Applied Parts		**Type BF Applied Parts**		**Type CF Applied Parts**	
Leakge Current Type	NC	SFC	NC	SFC	NC	SFC
Earth leakage	0.5	1	0.5	1	0.5	1
Enclosure leakage	0.1	0.5	0.1	0.5	0.1	0.5
Patient leakage (DC)	0.01	0.05	0.01	0.05	0.01	0.05
Patient leakage (a.c.)	0.1	0.5	0.1	0.5	0.01	0.05
Patient leakage (mains on applied part)	NA	NA	NA	5	NA	0.05
Patient leakage (mains on signal input or signal output)	NA	5	NA	NA	NA	NA
Patient Auxiliary Current (DC)	0.01	0.05	0.01	0.05	0.01	0.05
Patient Auxiliary Current (a.c.)	0.1	0.5	0.1	0.5	0.01	0.05

5.6.5 Medical Electrical Systems

It is important to stress that the act of attaching a medical device to a non-medical device constitutes the process of manufacturing a new medical device, which must then be tested for compliance to 60601, this can be as simple as plugging a lamp into an extension lead. This aspect of safety is covered under BS EN 60601-1-1:2001 “Safety Requirements for medical electrical systems”.

Since the recent loosening of restrictions on the use of mobile phones in hospitals, some concern has been raised that hospital visitors may plug mobile phone chargers into hospital electrical circuits and potentially unplug incorrect devices. The 60601 regulations is particularly pertinent in such cases, with the requirement that enclosure leakage currents from or between parts of the system with the patient environment shall not exceed 0.1mA. The patient environment is a clearly defined envelope extending 1.5 metres around the bed and 2.5 metres above the bed. This equally applies to visitors televisions and devices produced during research and development.

5.7 INVESTIGATION - Compliance Test of an ECG Monitor

I conducted the following test on an HP Merlin Monitor with 3-lead ECG monitor, using the Bio-Tek 601 Pro electrical safety tester. All tests comply with BS EN IEC60601. The monitor is a class 1, type CF device. Serial number: 3051G04784.

Table 22 Test results for ECG monitor compliance to 60601

Test no	Test Description	Results
0	Plugs and cables in satisfactory condition?	Yes
1	Mains voltage and current	249.3V and 0.4A

		Measured Value	Unit	Result	Accepted Limit
2	Insulation resistance (mains to earth)	Over	MΩ(min)	Pass	>50MΩ
3	Insulation resistance (applied parts to earth)	Over	MΩ(min)	Pass	>50MΩ
4	Earth continuity	0.125	Ω(max)	Pass	<0.2Ω

		Normal	Reverse	Unit	Result	Accepted Limit
5	Earth leakage (normal condition)	66	55	µA	Pass	0.5mA
6	Earth leakage (single fault, supply open)	59	58	µA	Pass	1mA
7	Enclosure leakage (normal condition)	1	1	µA	Pass	0.1mA
8	Enclosure leakage (single fault, earth open)	66	55	µA	Pass	0.5mA
9	Enclosure leakage (single fault, supply open)	0	0	µA	Pass	0.5mA
10	Patient leakage (normal conditions)	0	0	µA	Pass	0.01mA
11	Patient leakage (single fault, earth open)	2	2	µA	Pass	0.05mA
12	Patient leakage (single fault, supply open)	0	0	µA	Pass	0.05mA
13	Patient leakage (mains on applied part)	9	9	µA	Pass	0.05mA
14	Patient auxiliary current (normal conditions)	0	0	µA DC	Pass	0.01mA
15		2	2	µA a.c.	Pass	0.01mA
16	Patient auxiliary current (single fault, earth open)	0	0	µA DC	Pass	0.05mA
		0	0	µA a.c.	Pass	0.05mA

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APPENDICES

Appendix A Calibration limits of accuracy for Neonatal Cortical AEPs

General Notes:

1. The measurement methods advocated in IEC 60645-3 shall be used for clicks and tone pips (2:1:2 cycle tone bursts) and be calibrated to reference zero values published on the NHSP website until ISO 389-6 is formally available;
2. Tone burst stimuli (lasting > 20 ms or having > 10 cycles) should be calibrated by extending the plateau to provide a near-continuous tone, and be calibrated to reference zero values given in ISO 389 parts 1-4.
3. Equipment is deemed to be *within calibration* if its performance lies within specified limits. Where possible, the limits shall be analogous to those given in IEC 60645-1 for a type 1 instrument. The limits are listed below.

Item	Accuracy	Achieved?
Signal level (All signals & transducers listed in part 1)	±3dB	
Masking level	+5dB to –3dB	
Tone frequency	±1%	
Attenuator linearity	±1dB per 5dB	
Total harmonic distortion (250Hz & 1kHz) (CERA tone bursts. All transducers listed in pt 1)	2.5% AC 5.5 % BC	
Visual check of waveform at max O/P for freedom from clipping / saturation / obvious distortion	check	
Stimulus polarity (clicks) (All transducers listed for clicks in part 1)	check	
Attenuator breakthrough	<-70dB	
R/L crosstalk	<-70dB	
Cursor amplitude accuracy (using internal or external test pulse)	±10%	
Cursor latency accuracy (using internal or external test pulse)	±1%	
Electrode impedance accuracy (5kΩ)	±10%	

Part 3: Calibration Correction Values

Tables of correction values have been provided for: ☐ ABR stimuli

☐ CERA stimuli

Date of calibration:

Calibrated by: on behalf of:

Source: Wood S. NHSP Guidelines for ERA Equipment Calibration. NHS Newborn Hearing Screening Programme. 2007.

Appendix B Subject Information Sheet for ABR Normal database Project

INFORMATION SHEET

Wandsworth Teaching Primary Care Trust NHS

Auditory Brainstem Response Test (ABR)

You have been asked to volunteer as a subject in an ABR test:

What is the ABR test? It is a routine clinical test that measures the electrical activity following the path of sound from your ear, through the brain, to the auditory cortex. The test is automated so it doesn't require a response from the patient. It is therefore used to assess the hearing of babies and other people who would not normally respond to the standard hearing test.

What is the purpose of this study? I am collecting data from a wide range of people with no reported hearing problems, in order to build up a table of data of normal ABR patterns. It will also form part of my training as a Trainee Clinical Scientist.

How will your data be used?

- As part of a table of data which will be used as a reference when assessing patients clinically with the ABR test
- A summary of the results will be included in my training portfolio

I will require your date of birth for information purposes only. All data will be anonymous, kept confidential and only used within Queen Mary's Hospital.

What does the test involve? Four electrodes will be placed on your head (one behind each ear and two on your forehead) and you will be asked to listen to some clicking sounds through a pair of headphones. While the test is being carried out, you are asked to relax on a bed. This is because the signal is very small (a few micro volts) and tense muscles generate unwanted noise that could hide the signal we are looking for. But you do need to stay awake!

Are you eligible? Yes if you aged between 18 – 50 and have no current hearing problems (tinnitus, glue ear, grommets, hearing aids etc)

Who, where and when?

The test will be conducted by me under the supervision of the Senior Audiologist at Queen Mary's. I am a postgraduate engineer and Trainee Clinical Scientist coming to the end of my training.

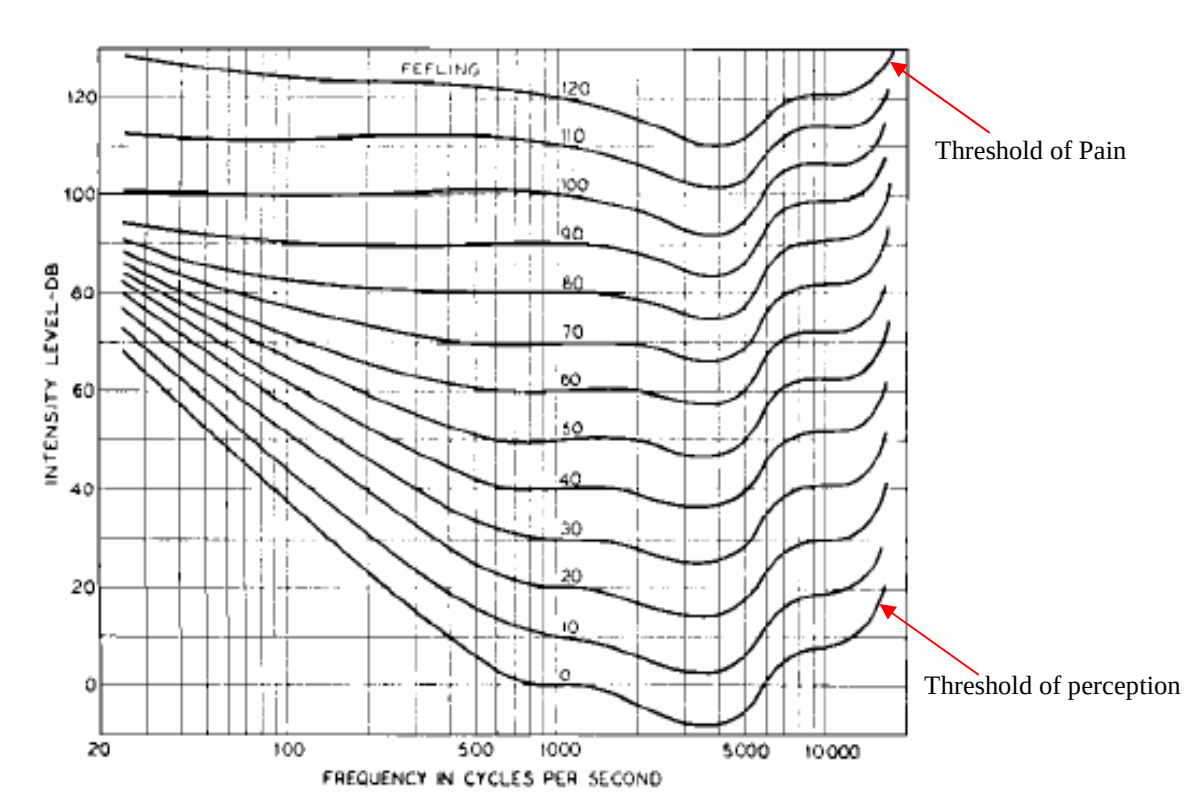
It takes about 1 hour, most of the time you'll be sitting listening to clicking sounds in the Audiology Department on the Ground floor (straight passed the coffee shop from the main entrance, first on the right).

If you have any questions please don't hesitate to contact me, Graham Webb on extension 6101, or Justine Sweet Head of Audiology on 020 8487 6381

Appendix C ABR Normal Database Project - Subject Data

Appendix D Equal Loudness Curves

In 1933 Fletcher and Munsen studied subjective loudness by comparing an increasing intensity stimulus against a 1 kHz reference tone, across the physiological frequency range (20Hz to 20kHz), the results were averaged across a large subject group [1]. They produced the 'equal loudness contour', which is used today to normalise acoustic intensity with frequency. Fletcher and Munsen's data has been added to over the years to derive a number of filtering functions for acoustic applications. Two commonly used filters in audio measurement are the A-weighting and C-weighting functions. The A-weighting filter is defined by International Standards Organisation (ISO) and subsequently by British Standards (BS) and is commonly applied to continuous time-varying sound pressure levels. It closely imitates the spectral response of the human ear by de-emphasising lower and higher frequencies (0-1 kHz and 5-16 kHz) and emphasizing mid-range frequencies (1-5 kHz). The C-weighting permits a broader bandpass and is applied to the measurement of impulse sounds [2].



Loudness level contours for an increasing number of phons. A phon is the unit of perceived loudness, (1 phon = 1dBSPL at 1kHz).

Sources:

[1] Fletcher H., M. W. (1933). "Loudness, Its Definition, Measurement and Calculation." J. of the Acoustical Society of America **5**: 82-108.

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Appendix E Spyrometer Reference Values

Reference values for lung volumes and forced ventilatory flows for adults of European descent. The equations apply to men and women of European descent, aged 18-70 years of age and were derived from studies carried out on subjects who were non-smokers without (previous) disease, that would have affected their ventilatory function and used tests and equipment similar to that employed today. The height range of the subjects in these studies was 1.55-1.95m in men and 1.45-1.80m in women. (Note: For measurements of PEF, RV, FRC and TLC it was not ensured that smokers and ex-smokers were consistently excluded from the studies). Accompanying notes can be found in Ruppel G. (1994). Manual of Pulmanory Function Testing, 6th Edition. Mosby-Year Book, Inc.

Table 1 Summary equations for lung volumes and ventilatory flows in adults*. The lower 5 or upper 95 percentile are obtained by subtracting or adding the figure in the last column from the predicted mean.

Variable	Unit	Regression Equation	RSD	1.64RSD
MEN				
IVC	1	6.10H-0.028A-4.65	0.56	0.92
FVC	1	5.76H-0.026A-4.43	0.61	1.00
TLC	1	7.99H-7.08	0.70	1.15
RV	1	1.31H+0.022A-1.23	.040	0.67
FRC	1	2.34H+0.009A-1.09	.06	0.99
RV/TLC	%	0.39A+13.96	5.46	9.0
FRC/TLC	%	0.21A+43.8	6.74	11.1
FEV_1	1/sec	4.30H-0.029A-2.49	0.51	0.84
FEV_1/VC	%	-0.18A+87.21	7.17	11.8
$FEF_{25-75\%}$	1/sec	1.94H-0.043A+2.70	1.04	1.71
PEF~	1/sec	6.14H-0.043A+0.15	1.21	1.99
MEF_{75}	1/sec	5.46H-0.029A-0.47	1.71	2..81
MEF_{50}	1/sec	3.79H-0.031A-0.35	1.32	2.11
MEF_{25}	1/sec	2.61H-0.026A-1.34	0.78	1.28
WOMEN				
IVC	1	4.66H-0.026A-3.28	0.42	0.69
FVC	1	4.43H-0.026A-2.89	0.43	0.71
TLC	1	6.60H-5.79	0.60	0.99
RV	1	1.81H+0.016A-2.00	0.35	0.56
FRC	1	2.24H+0.001A-1.00	0.50	0.82
RV/TLC	%	0.34A+18.96	5.83	9.60
FRC/TLC	%	0.16A+45.1	5.93	9.80
FEV_1	1/sec	3.95H-0.025A-2.60	0.38	0.61
FEV_1/VC	%	-0.19A+89.10	6.51	1.07
$FEF_{25-75\%}$	1/sec	1.25H-0.034A+2.92	0.85	1.40
PEF~	1/sec	5.50H-0.030A+1.11	0.90	1.48
MEF_{75}	1/sec	3.22H-0.025A-1.60	1.35	2.11
MEF_{50}	1/sec	2.45H-0.025A-1.16	1.10	1.61
MEF_{25}	1/sec	1.05H-0.025A-1.11	0.69	1.13

H: standing height (m); A: age (yr); RSD: residual standard deviation. *Between 18 and 25 yr in the equations. ~Mixture from (mini) Wright peak flowmeter and pneumotachometer: more work is needed.

Table 2 Approximate conversion factors for adjusting European reference values for application to men of other ethnic groups

Population	**FEV_1**	**FVC**	**Reference**
Oriental			
Hong Kong Chinese	1.0	1.0	[247]
Japanese American	0.89	-	[248]
Polynesian	0.90	0.90	[249]
N. Indian & Pakistani	0.90	0.90	[250]
N.Indian, see Africans			[251]
African descent or subtract (1)	0.87 0.45* or	0.87 0.70*	[252] [241]
*The corresponding volumes for women are 0.41 and 0.61			

Table 3 Summary equations for total lung capacity and transfer factor for adults age 25-70 yr. The upper and lower percentiles are obtained by adding or subtracting the figure in the last column from the predicted mean.

Index	**Regression equation**	**1.64 RSD**
Men		
TLCO SB*	11.11H-0.066A-6.03	2.32
TLC(1)	7.99H-7.08	1.15
Women		
TLCO SB*	8.18H-0.49A-2.74	1.92
TLC(1)	6.60H-5.79	0.99
*mmol min^{-1} kPa^{-1}; H: standing height (m); A: age (yr); RSD: residual standard deviation. Between 18 and 25 yr: substitute 25 yr in the equations.		

Sources:

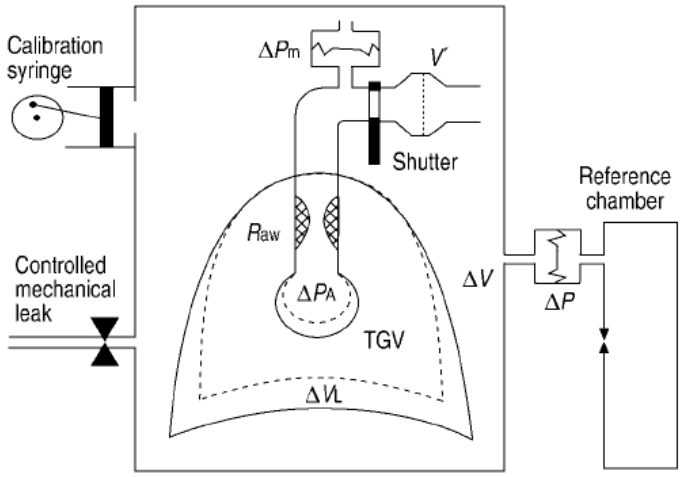
Quanjer Ph.H, Tammeling GJ, Cotes JE, Pedersen R, Yernault J-C. (1993). Report working party. Standardisation of lung function tests European Community for steal and coal: Lung Volumes and Forced Ventilatory Flows. European Respiratory Journal, Vol. 6, Suppl 16, pp 4-40

Quanjer Ph.H, Chinn DJ, Cotes JE, Roca J, Yernault J-C. (1993). Report working party. Standardisation of lung function tests European Community for steal and coal: Standardisation of the measurement of transfer factor (diffusing capacity). European Respiratory Journal, Vol. 6, Suppl 16, pp 41-52

Appendix F Equipment specification: 'Body Box'

Flow measurement:	JAEGER Pneumotach
Range	0 - ±20 l/s
Accuracy	0.2 - 12 l/s ±2 %
Resistance	<0.05 kPa/(l/s) at 10 l/s
Volume determination:	digital integration
Range	0 - ±20 l/s
Accuracy	5 ml
Mouth pressure:	JAEGER pressure transducer
Range	±20 kPa
Accuracy	< ±2 %
Box pressure:	JAEGER pressure transducer
Range	±1 kPa
Accuracy	< ±2 %
Box:	aluminium/acrylic glas
Principle	volume-constant
Volume	830 litres
BTPS simulation	ASC electronics
He Analyser:	Electrochemical cell
Range	0-9.5%
Accuracy	0.05%
CO Analyser:	Electrochemical cell
Range	0-4%
Accuracy	0.0003%
Calibration:	CAL-Pack, automatically
Space Requirements:	
Box	90 * 90 cm
Trolley	70 * 90 cm

Appendix G Derivation of Intrathoracic gas volume



The constant volume (or variable pressure) plethysmograph.

Parameters:

V	volumetric flow rate
ΔPm	change in mouth pressure
ΔPA	alveolar pressure
TGV	Thoracic gas volume
ΔV	change in box volume
Raw	airway resistance
ΔP	change in box pressure

Isothermal conditions can be assumed since the wall thermal time constant is greater than rate of physiological heat transfer. The leakage time constant is also greater than the rate of breathing. Therefore Boyles law, which states that P.V is constant (under isothermal conditions), can be applied to the lungs as follows:

$$P_A.TGV = (P_A - \Delta P)(TGV + \Delta V)$$

The inspiratory or expiratory effort against the closed shutter will decrease or increase P_A by ΔP_A, and increase or decrease TGV by the small volume change ΔV.

Rearranging: $$TGV = \left(\frac{\Delta V}{\Delta P_A}\right)\left(P_A - \Delta P_A\right)$$

Since ΔP_A « P_A (<2%) it is usually omitted in the differential term and $P_A = P_{bar} - P_{H_2O,sat}$.

So $$TGV \approx \left(\frac{\Delta V}{\Delta P_A}\right) \cdot \left(P_{bar} - P_{H_2Osat}\right)$$

When respiratory efforts are made against the closed shutter ΔP_A can be recorded as ΔPm. ΔV is measured by the calibrated plethysmograph gas pressure transducer.

Appendix H Volume-flow loops for cases TD and FD

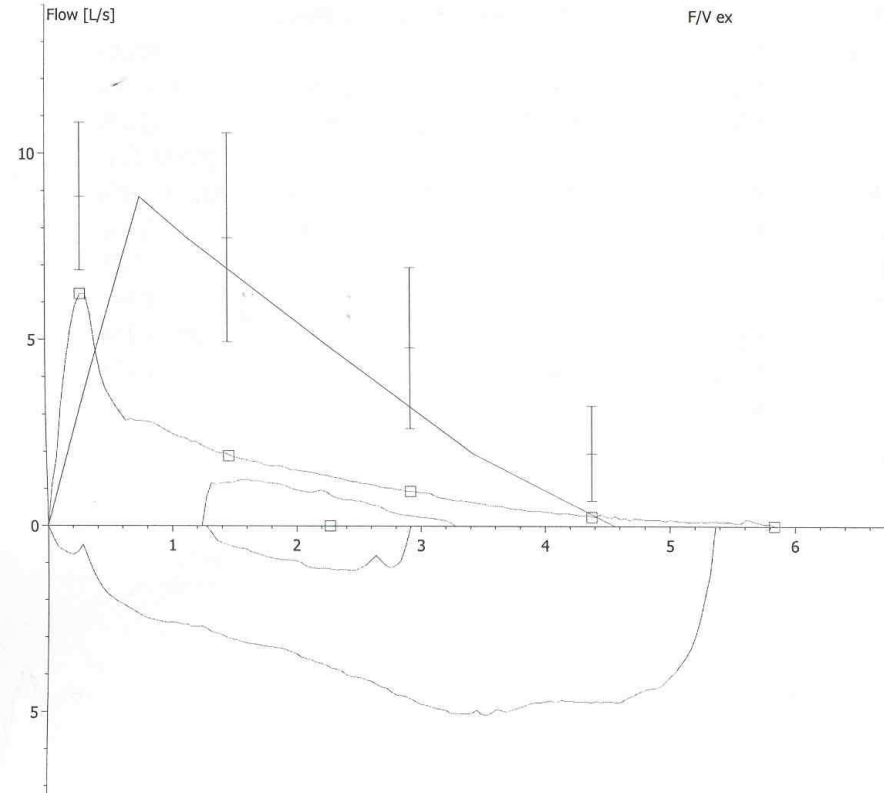


Figure 59 Volume flow loop result from TD

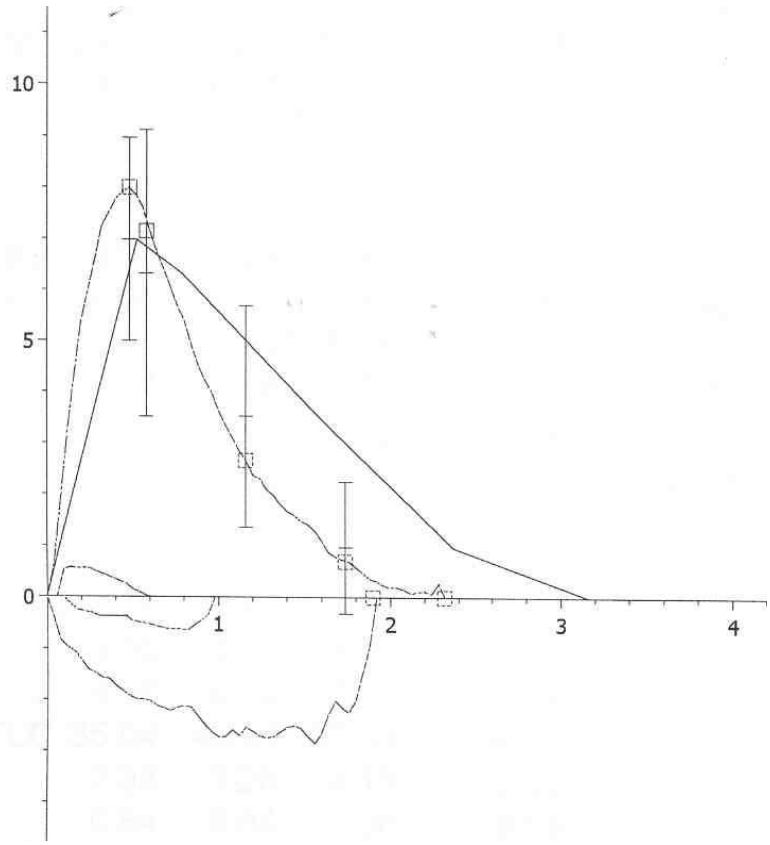


Figure 60 Volume flow loop result for FD

Appendix I Risk Assessment for 'body box'

Device: Jaeger Tonnies' "Master Screen Body Equipment"

Assessment Questionnaire taken from ISO14971:2000.

Q1. What is the intended use / intended purpose and how is the medical device to be used?

- To assess the lung function, volumes and capacities of patients attending the lung function clinic.
- Mental and Physical factors: to accommodate wheelchair users, users with claustrophobia and those who easily over exert, mentally and physically.
- Skill and training: users are coached by staff trained according to Association for Respiratory Technology and Physiology standards.
- Ergonomic factors: height adjustable seat with adjustable back rest to promote correct erect posture for measurement. Transparent box for comfort.
- Environment: clinical setting, not in glare of direct sunlight, stable ambient pressure and temperature.
- Influence of patient on control of medical device: patient can remove the mouthpiece and end the tests at any time. Patient has no other control over functional operation.

Q2. Is the medical device intended to contact the patient or other persons?

- Yes, via a mouthpiece that is sterilised and a disposable anti-viral, anti-bacterial filter.

Q3. What materials and/or components are incorporated in the medical device or used with, or are in contact with, the medical device?

- A rubber mouthpiece, a disposable anti-viral anti-bacterial filter and a length of tubing to connect the pneumotachometer head to the analyser.

Q4. Is energy delivered to and/or extracted from the patient?

- Yes. A test gas is delivered to the patient and the patient exerts a mechanical energy through the act of ventilation.

Q5. Are substances delivered to and/or extracted from the patient?

- Yes. A test gas (BOC MA 0735 / 0011R) supplied by BOC Special Products Division under licence, is delivered to the patient. Gas constituents: 0.28% CO, 9% H_e, 19% O_2, 71.72% N_2.

Q6. Are biological materials processed by the medical device for subsequent re-use?

- No.

Q7. Is the medical device supplied sterile or intended to be sterilised by the user, or are other microbiological controls applicable?

- Yes. The device is not supplied sterile, but the mouthpieces and tubing are sterilised on a daily basis. General aspects of the equipment are disinfected when required or on a weekly basis, whichever is sooner.

Q8. Is the medical device intended to be routinely cleaned and disinfected by the user?

- Yes. See Q7.

Q9. Is the medical device intended to modify the patient environment?

- Yes. The device will occlude patient airflow for the duration of two breaths.

Q10. Are measurement taken?

- Yes. Lung function, volumes and capacities are taken during spirometry, plethysmograpy and single breath gas diffusion testing.

Q11. Is the medical device interpretive?

- No. Trained clinical staff present results to Consultant Cardiothoracic Physicians for interpretation.

Q12. Is the medical device intended for use in conjunction with medicines or other medical technologies?

- Yes. The device is to be compliant with pacemakers and other body worn implants. The device is intended for use with bronchodilators such as Salbutamol and Ventilin.

Q13. Are there unwanted outputs of energy or substances?

- Yes. The device may emit electromagnetic radiation.

Q14. Is the medical device susceptible to environmental influences?

- Yes. The device function is sensitive to ambient pressure and temperature variations.

Q15. Does the medical device influence the environment?

- Yes. See Q13.

Q16. Are there essential consumables or accessories associated with the medical device?

- Yes. Nose pegs, mouthpieces, anti-viral anti-bacterial filters and airway tubing.

Q17. Is maintenance and/or calibration necessary?

- Yes. The device is maintained under a commercial planned maintenance contract.
- The device is calibrated daily, or more frequent if appropriate.

Q18. Does the medical device contain software?

- Yes. Embedded software within the control box and a high level user interface on the host PC.

Q19. Does the medical device have a restricted shelf-life?

- No.

Q20. Are there any delayed and/or long-term use effects?

- Yes. Momentary breathlessness or fatigue following immediate use.

Q21. To what mechanical forces will the medical device be subjected?

- The seat post and base will see a force of no greater than 200kg.
- The pneumotachometer head and supporting struts will be subject to general movement wear.
- The door hinges will be subject to general wear and tear.
- The side panels may be knocked.

Q22. What determines the lifetime of the medical device?

- Senescence through the introduction of new and novel techniques.
- Spare part obsolescence.

Q23. Is the medical device intended for single use?

- No. The device will be used approximately ten times per day.

Q24. Is safe decommissioning or disposal of the medical device necessary?

- No.

Q25. Does installation or use of the medical device require special training?

- Yes. Installation is carried out under supervision of the Medical Physics and Bioengineering Department.
- The device is used by staff trained according to Association for Respiratory Technology and Physiology standards.

Q26. Will new manufacturing processes need to be established or introduced?

- No.

Q27. Is successful application of the medical device critically dependent on human factors such as the user interface?

- Yes. Data is collected in accordance with Association for Respiratory Technology and Physiology guidelines.

Q27.1 Does the medical device have connecting parts or accessories?

- Yes.

Q27.2 Does the medical device have a control interface?

- Yes.

Q27.3 Does the medical device display information?

- Yes.

Q27.4 Is the medical device controlled by a menu?

- Yes.

Q28 Is the medical device intended to be mobile or portable?

- No.

Appendix J Risk Management in the Trust

Wandsworth PCT classifies risks as either Acceptable or Unacceptable. An acceptable risk is "one which has been accepted after proper evaluation and is one where appropriate controls have been implemented. The risk must not only be identified, but also quantified to the maximum practicable, analysed and communicated to the appropriate level of management"[1]. Action should be taken to reduce any unacceptable risks to an acceptable level. All acceptable risks are measured according to their likelihood (or frequency) and severity (or consequences) and entered into a risk matrix (Table 23). The acceptance of a risk should therefore represent an informed decision to accept the consequences and likelihood of that risk.

Table 23 Risk Matrix

Likelihood (frequency)	Severity (consequences)				
	1 (Insignificant)	2 (Minor)	3 (Moderate)	4 (Major)	5 (Catastrophic)
5 Certain	5 Y	10 Y	15 R	20 R	25 R
4 Likely	4 G	8 Y	12 Y	16 R	20 R
3 Possible	3 G	6 Y	9 Y	12 Y	15 R
2 Unlikely	2 G	4 G	6 Y	8 Y	10 Y
1 rare	1 G	2 G	3 G	4 G	5 Y

Table 24 shows the descriptors used to quantify risk likelihood and Table 25 shows the risk severity descriptors.

Table 24 Likelihood (frequency or probability) or risk occurring or repeating.

SCORE		DESCRIPTION
1	RARE	Do not believe will happen, one off. Exceptional circumstances
2	UNLIKELY	Not expected but possible. Could occur at some time
3	POSSIBLE	May/should occur at some time
4	LIKELY	Will probably occur.
5	ALMOST CERTAIN	Likely to occur on many occasions. A persistent issue

Table 25 Risk severity levels identified by the National Patient Safety Agency

Description	Impact on individual	Impact on organisation	Person affected at any one time	Financial Impact Complaint Litigation
1 Insignificant	No injury	No risk to the PCT No impact on service No impact on environment	None	Theft/loss up to £1k Complaint unlikely Litigation risk remote
2 Minor	First Aid Minor Injury or Minor illness up to 1 month	Minimal risk to PCT Slight impact on service Slight impact on environment	Very few 1-2	Theft/loss between £1k - £5K Complaint possible Litigation <£50K
3 Moderate	Temporary incapacity. Short term monitoring. Additional Medical treatment required up to 1 year	Some service disruption. Potential for adverse publicity Moderate impact on environment	Small numbers 3 - 15	Theft/loss £5k - £25k Complaint expected Litigation possible >£50k - £500k
4 Major	Major Injury (reportable) major clinical intervention Permanent incapacity	Service restriction Adverse publicity Loss of reputation Major impact on environment	16 – 50	Theft/loss £25k – £200k Litigation >£500 - £1m expected
5 Catastrophic	Death	National Media Interest. Severe loss of confidence	50+	Theft/loss over £200k Litigation >£1m

Source:

[1] Caulfeild-Stoker, D. (May 2006). Risk Management Strategy, Wandsworth Teaching PCT.

Appendix K PCT Risk Assessment Form

Wandsworth NHS
Teaching Primary Care Trust

Wandsworth PCT

Risk Assessment Form

Section 1 – Process Description

DIRECTORATE:	**DEPARTMENT:**
Process/Activity:	

Risk/Hazard	Persons at Risk	Existing Control Measures	Severity (consequences)	Likelihood /Frequency	Risk Rating SxL
1					
2					
3					
4					
5					

Section 2 – Identifying Risks/Hazards

Complete Action Plan and attach to Risk Assessment form.

Is any employee health monitoring required?	Yes □	No □
Is a more detailed assessment (e.g. COSHH, Manual handling) required?	Yes □	No □
Is further information or investigation required to complete risk assessment?	Yes □	No □

Assessor's Name:	Job Title:
Date of Assessment:	Reassessment Date:
Assessor's Signature:	Manager's Signature:

DIRECTORATE: **DEPARTMENT:**

Risk/Hazard	Risk Score	Action Required to Control Risk	Lead Person	Action By	Comments

Completed By:
Date:
Manager's Signature:

Appendix L International 10-20 System of Electrode Placement

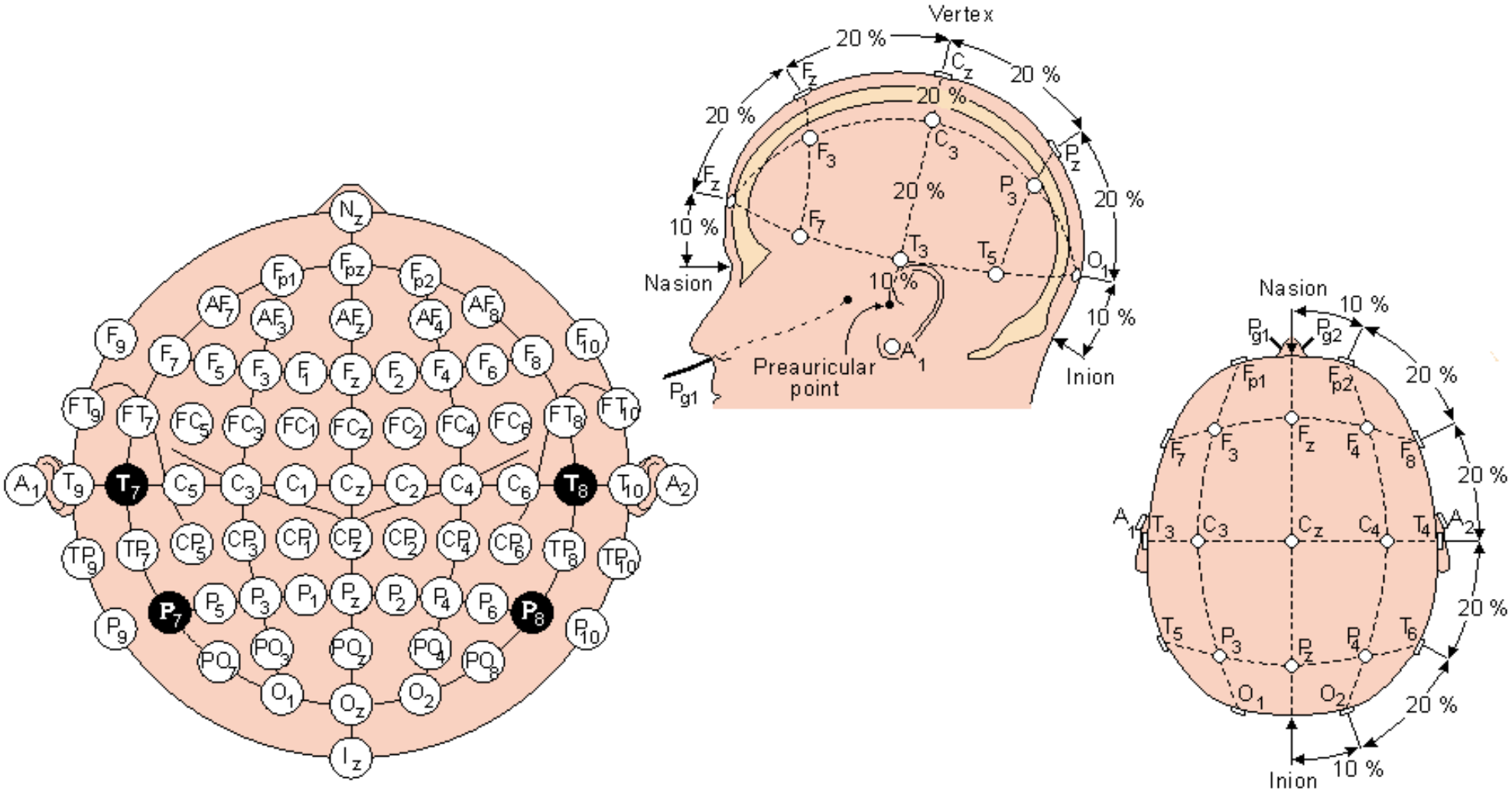
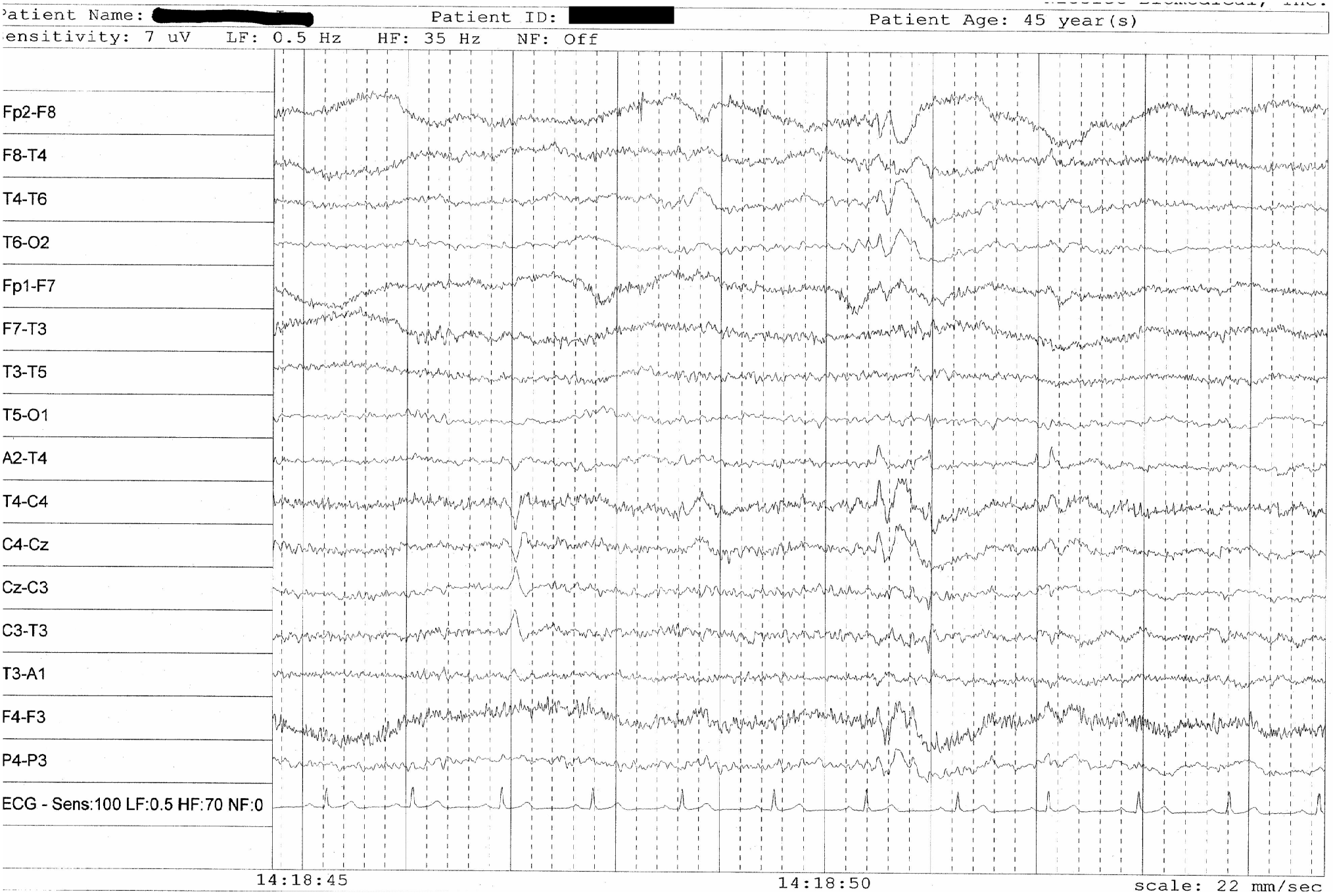


Figure 61 The international 10-20 system. A = Ear lobe, C = Central, Pg = nasopharyngeal, P = Parietal, F = Frontal, Fp = Frontal Polar, O = Occipital.

Appendix M EEG Results for CASE IM

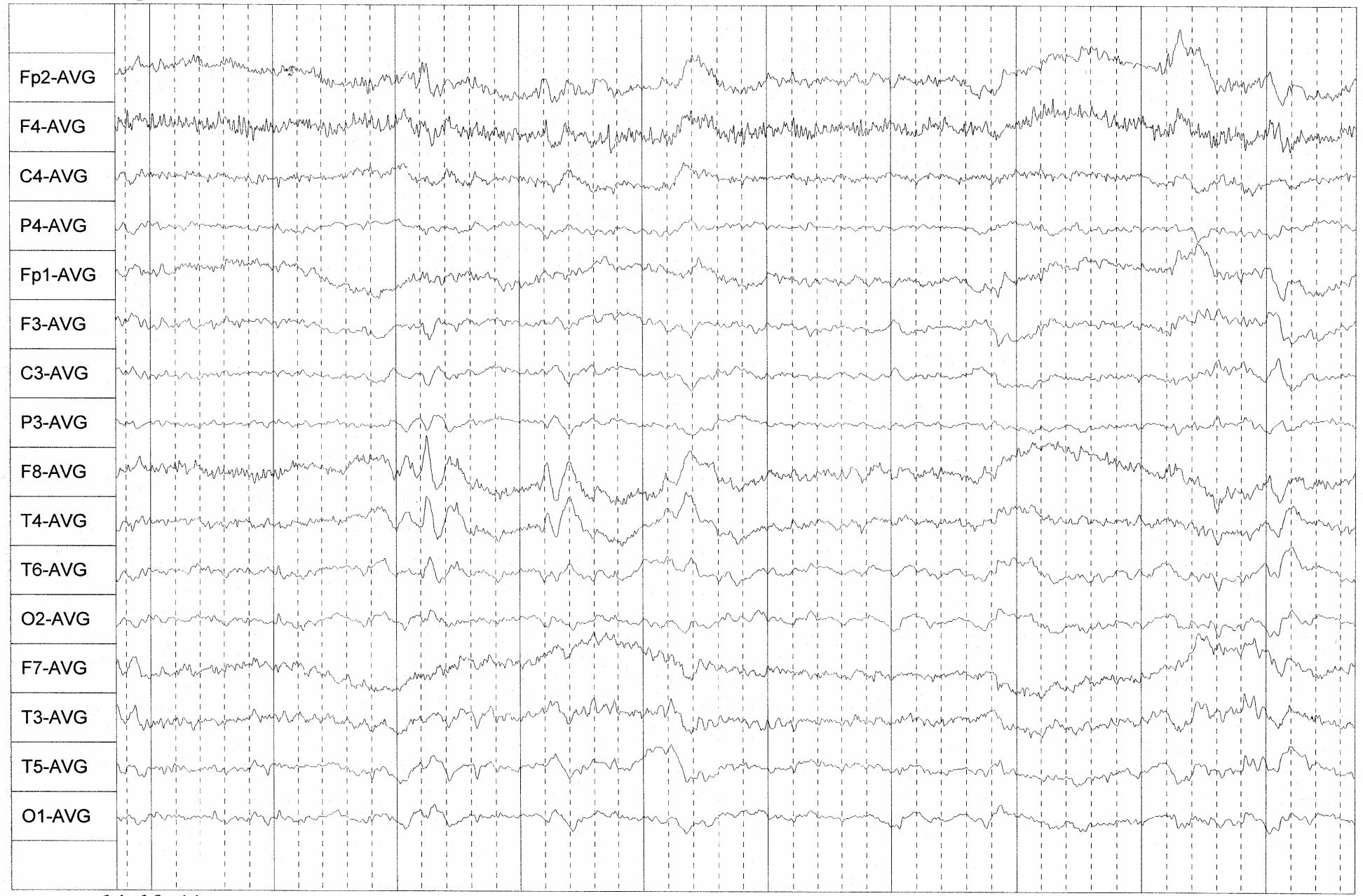


NicoletOne

Nicolet Biomedical, Inc

Patient Name: [redacted] Patient ID: [redacted] Patient Age: 45 year(s)

Sensitivity: 7 uV LF: 0.5 Hz HF: 35 Hz NF: Off

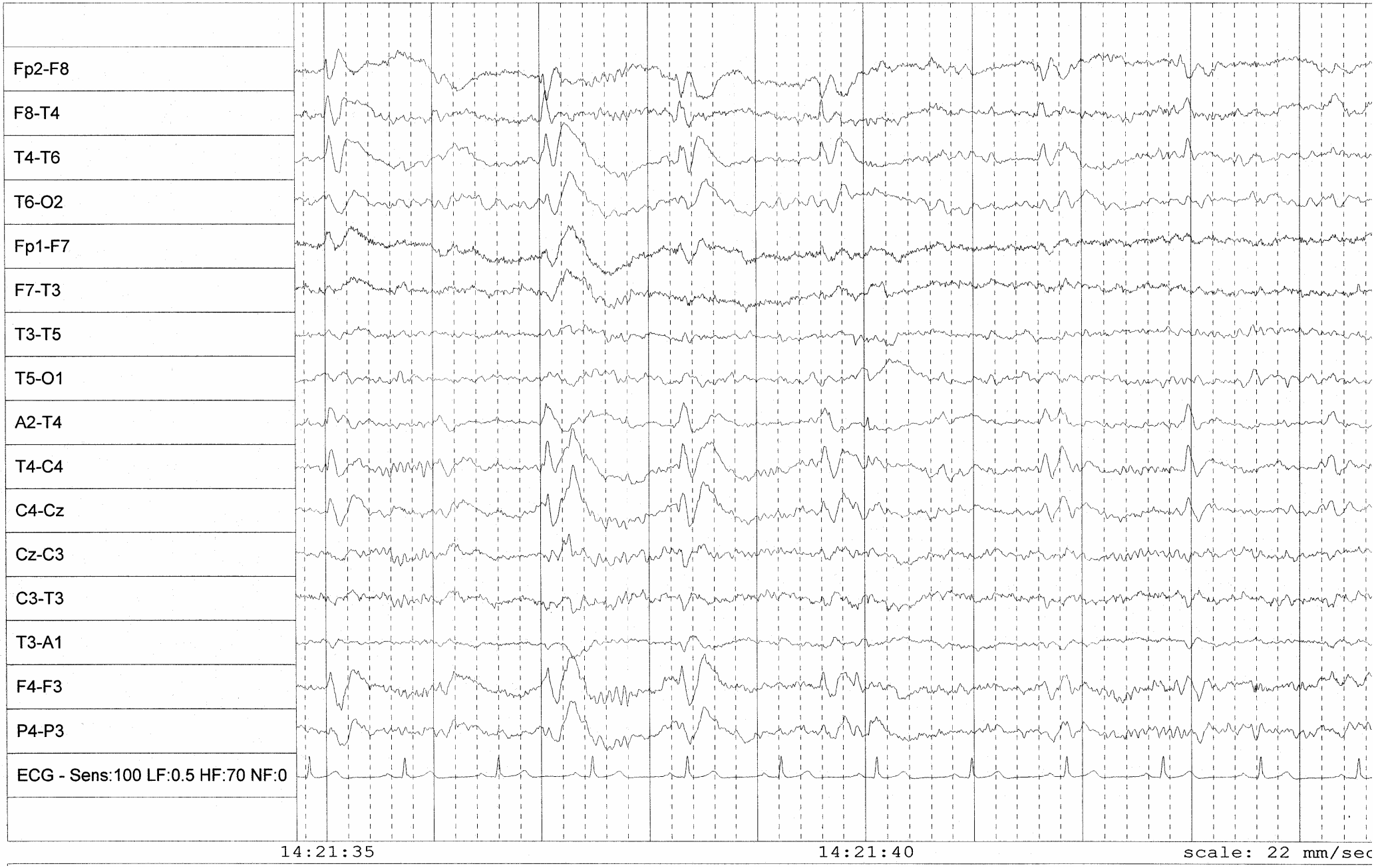


scale: 24 mm/se

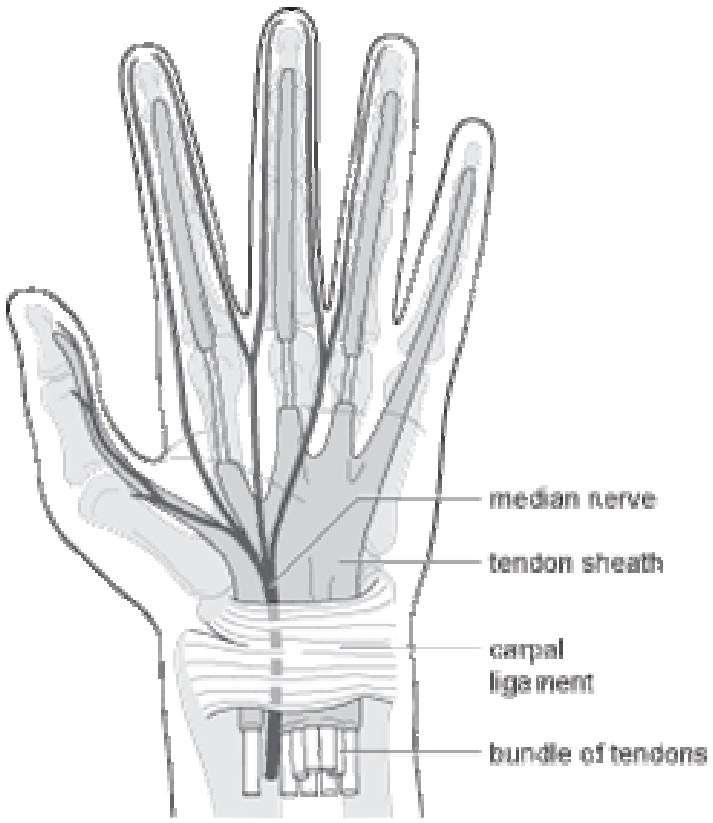
NicoletOne Nicolet Biomedical, Inc.

Patient Name: [redacted] Patient ID: [redacted] Patient Age: 45 year(s)

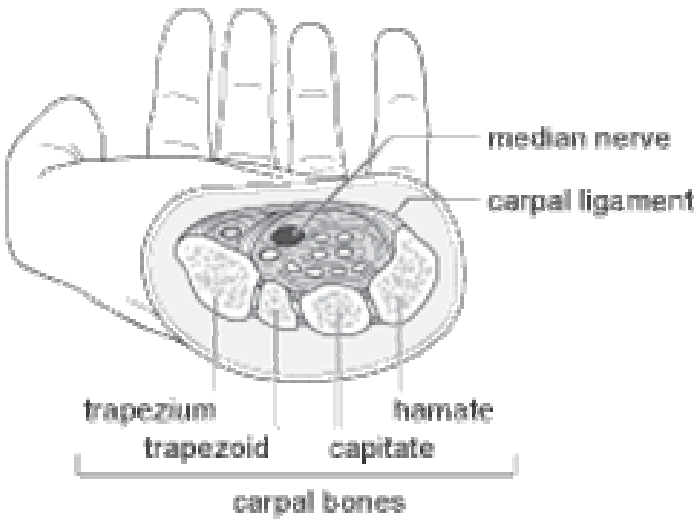
Sensitivity: 7 uV LF: 0.5 Hz HF: 70 Hz NF: Off



Appendix N Carpal Tunnel Anatomy



Frontal view of left hand detailing aspects of interest in CTS



Transverse view of left hand

Appendix O Equipment specification: blood pressure monitor

The following is the specification for pressure measurement and other general features of the Draeger "Infinity Delta" series patient monitoring system, in use throughout the intensive care units at St George's.

Noninvasive Blood Pressure (NBP)
Displayed parameters Systolic, mean and diastolic pressures
Measuring method Oscillometric utilizing step deflation
Modes of operation
Manual (single measurement); Interval and Continuous (5 minutes)
Interval times 1, 2, 2.5, 3, 5, 10, 15, 20, 25, 30, 45, 60, 120 and 240 minutes
Measuring range (default)
Adult (Adult 270 mmHg)
• Heart rate 30 to 240 bpm
• Systolic 30 to 250 mmHg
• Mean 20 to 230 mmHg
• Diastolic 10 to 210 mmHg
Pediatric (Pediatric 180 mmHg)
• Heart rate 30 to 240 bpm
• Systolic 30 to 170 mmHg
• Mean 20 to 150 mmHg
• Diastolic 10 to 130 mmHg
Neonatal (Neonatal 140 mmHg)
• Heart rate 30 to 240 bpm
• Systolic 30 to 130 mmHg
• Mean 20 to 110 mmHg
• Diastolic 10 to 100 mmHg
Cuff pressure
Default inflation pressure
• Adult (270): 160 mmHg ± 10 mmHg
• Pediatric (180): 120 mmHg ± 10 mmHg
• Neonatal (140): 110 mmHg ± 10 mmHg
Inflation pressure after a valid measurement
• Adult (270): Systolic + 25 mmHg ± 10 mmHg
• Pediatric (180): Systolic + 25 mmHg ± 10 mmHg
• Neonatal (140): Systolic + 30 mmHg ± 10 mmHg
Maximum inflation pressure
• Adult (270): 265 mmHg ± 5 mmHg
• Pediatric (180): 180 mmHg ± 10 mmHg
• Neonatal (140): 142 mmHg ± 10 mmHg
Minimum inflation
• Adult (270): 110 mmHg ± 10 mmHg
• Pediatric (180): 90 mmHg ± 10 mmHg
• Neonatal (140): 70 mmHg ± 10 mmHg
Connector Quick-release connector with single airway

Invasive Blood Pressure
Displays up to 8 pressures
Measuring method Resistive strain gauge transducer
Display resolution 1 mmHg
Measuring range -50 to 400 mmHg
Frequency ranges DC to 8 Hz, DC to 16 Hz, and DC to 32 Hz (user-selectable)
Zero balance ± 200 mmHg
Transducer specifications Dräger Medical-approved transducers with a resistance of 200 to 3000 and an equivalent pressure sensitivity of 5µV/V/mmHg ± 10%
Accuracy ± 1 mmHg or ± 3%, exclusive of transducer (whichever is greater)
IBP alarms User-selectable upper and lower limits for systolic, mean and diastolic pressures
Accessories Dräger Medical-approved pressure transducers

Display Specifications
Type Thin Film Transistor-Liquid Crystal Display Active Matrix (TFT-LCD)
Size (Delta) 264 mm (10.4 in.) diagonal
Channels 5 channels standard, 6, 8 channels optional
Viewing area 211 x 158 mm (8.3 x 6.2 in.)
Resolution 640 x 480 pixels
Size (Delta XL) 310 mm (12.2 in.) diagonal
Channels 6 channels standard, 8 channels optional
Viewing area 246 x 184.5 mm (9.7 x 7.3 in.)
Resolution 800 x 600 pixels

User Interface
Rotary knob
Easy-to-use menu structure and fixed keys
Three alarm levels Life-threatening, Serious and Advisory

Connections
MultiMed® Pod, HemoMedTM Pod, Pod Comms (Delta: 1 standard, 1 optional;
Delta XL: 2 standard), NBP-input, etCO2 module, Infinity Docking Station, analog
output, QRS sync output, RS 232, remote keypad, and Scio® multigas module via X8.

Analog Output
Signals ECG, arterial blood pressure
Delay ≤25 msec

Physical Specifications
Cooling Convection
Size (Delta) H x W x D 253 x 365 x 190 mm (10.0 x 14.4 x 7.5 in.)
Weight (Delta) 5.8 kg (12.7 lbs.) with external battery 6.4 kg (14.0 lbs.)
Size (Delta XL) H x W x D 272 x 384 x 190 mm (10.7 x 15.1 x 7.5 in.)
Weight (Delta XL) 6.2 kg (13.6 lbs.) with external battery 6.8 kg (14.9 lbs.)

Information Management Capabilities
Data storage 24 hours of trended parameter information
Data resolution 30-second sampling
Trend tables 1-, 5-, 15-, 30- or 60-minute display formats
Trend graphs 1-, 2-, 4-, 8-, 12- or 24-hour display formats

Electrical Specifications
Input voltage 11 to 15 VDC
Power consumption ≤70 watts (fully loaded)
Patient leakage current ≤10µA

Power adapter
Power requirements 100 to 120V AC, 3.4A or
200 to 240V AC, 1.7A (switch selectable)
Frequency 50 to 60 Hz
Chassis leakage current <300µA @ 120V AC
<500µA @ 220V AC

Battery Specifications
Internal battery
Battery type Lithium-ion
Battery capacity 180 minutes
Charging time 6.5 hours at 25°C
External auxiliary battery
Battery type Sealed lead-acid
Battery capacity 50 minutes
Charging time 3.5 hours at 25°C
Size (external auxiliary battery)
H X W X D 62 x 182 x 24 mm
(2.4 x 7.2 x .9 in.)
Weight 0.635 kg (1.4 lbs.)
Battery life is specified for new batteries with monitor running under the following
conditions: ECG, SpO2, four invasive blood pressures, two temperature probes,
NBP measurement every 15 minutes connected, display at 50% brightness.
Battery life may diminish after extended use.

Environmental Requirements
Temperature range
Operating 10°C to 40°C
Storage -20°C to 40°C
Relative humidity
Operating 20% to 90%, non-condensing
Storage 10% to 95% (with packaging)
Atmospheric pressure
Operating 525 to 795 mmHg
(70 to 106 kPa)
Storage 375 to 795 mmHg
(50 to 106 kPa)
Standards
IEC 60601-1 and applicable particular and collateral standards,
IEC 60601-1-2, Electromagnetic compatibility CISPR 11, Class B
The Delta and Delta XL monitors comply with Medical Devices Directive (MDD) 93/42 EEC and bear the CE mark.