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## REPORT OF THE HON. SECRETARY TO THE ANNUAL GENERAL MEETING, 10 MAY 2002

### SCIENTIFIC MEETINGS HELD IN LONDON (October 2001 – April 2002)

#### 5 OCTOBER 2001

Presidential Address: *The Magic Numbers: Unravelling the Code.*

#### Professor Anthony Adams

A finger on the spacebar unleashed the presentation with a rousing musical introduction. We were then transported to another world of numbers, codes and ciphers. The recent premier of the film *Enigma* gave the presentation remarkable topicality and proved to be its backdrop. How were these 'Magic numbers' so beloved of the code breakers at Bletchley Park going to weave their way into Professor Adams' presentation the audience wondered?

We were then allowed a flashback, not to the Second World War on this occasion, but to rural Exeter and to the SHO boyhood of our President and the beginnings of his life-long love for anaesthetic circuits (even those, incidentally, with CO<sub>2</sub> absorption!) and ventilators. We could almost see him pondering over the Radford nomogram and twiddling the Sturme Archer gearbox of the East Radcliffe ventilator. Perhaps nothing changes after all?

A diversion, but keeping with the numbers theme, allowed one of the great gurus of anaesthesia to enter into the story, William Mapleson. Here it was fame associated with circuits and the simple letters of the alphabet, ABCDEF. We began to see a connection when the President recalled London in the '70s, this time, London, Canada and the University of Western Ontario (UWO) where he was a Visiting Professor. In 1972 a new streamlined breathing system was heralded in the *Canadian Anaesthetists' Society Journal* with a paper from UWO by none other than Bain and Spoerel. The latter was the Professor and we were told of his German naval origins (another clue?). Indeed he was an anti-Nazi and became a POW near Oxford, before being accepted by Canada and eventually spending 30 years as Professor in London.

The Bain circuit obviously captured the imagination of our President. Magic numbers such as 70 ml kg<sup>-1</sup> and 35 mmHg began to enter the story. We were told of elegant work performed on the circuit and after considerable redesign, the final production of the UK Bain circuit manufactured by Penlon in Abingdon, Oxford. It was clear that the magic numbers of 70 ml kg<sup>-1</sup> and 100 ml kg<sup>-1</sup> as FGF gave consistent CO<sub>2</sub> readings. Henville and Adams studied 100 patients to support the work of Bain and Spoerel, 70 ml gave a PaCO<sub>2</sub> of 40 and 100 a PaCO<sub>2</sub> of 34 mmHg. So influential was their work that a survey showed that 68% of all anaesthesia departments in the UK used Bain circuits with the Penlon modification.

Other benefits of this predictable CO<sub>2</sub> were discussed such as maintaining cerebral blood flow lowering intra-ocular pressure and ease of getting the patient to breathe at the end of the case without the dangerous habit of CO<sub>2</sub> administration. However, the click, wheeze, clunk, splutter of the venerable East Radcliffe with its innumerable water bottles, pistons and chains was just not the right ventilator for this innovatory circuit.

It was at this point that PneuPac Ltd., its redoubtable founder, George Weiss together with a portable resuscitation ventilator entered the fray. George also fits nicely into the picture as he had come to the UK as a refugee from Austria and, incidentally (although not mentioned by the President), is the father of the beautiful actress Rachel Weiss! Anyway, I digress! To cut a long story short, the PneuPac mechanism was incorporated into a new ventilator that was made appropriately enough by Penlon. We were also told the happy story of how the name Nuffield came to be applied to the ventilator after the support of Professor Macintosh. This coincided with the arrival of our President as Professor of Anaesthesia at Guy's Hospital. It was then that I realised why the President had been presented with a working Penlon Nuffield ventilator at his retirement Party at Guy's.

Examination candidates, and indeed examiners, were then treated to a masterly explanation of how the Penlon Nuffield works and the subsequent innovation of Dr Newton and his valve in allowing the ventilator to be used in infants and small children. Appropriate to our meeting this evening was the question of gas dilution from the ventilator driving gas, the 1-metre hose and awareness. With scrupulous scientific diligence, aided and abetted by a fast oxygen electrode which had been invented by Dr Hahn, the President and his co-workers were able to demonstrate that yes, indeed, 1 metre was enough!

How were we going to return to the Magic numbers? Reference back to aircrew in the Second World War heralded the return of Macintosh and his worthy assistant, EA Pask. They used none other than the precursor of a Bain circuit to devise experiments, which, under anaesthesia, allowed life jackets to be developed that kept the aircrew's head upright in the event of landing on water in the unconscious state. The videotape footage of Pask floating, anaesthetised and intubated in the wave tank at Ealing film studios will long remain in the memory.

So the talk comes full circle with U-boats and the Enigma code machines. Many of us will now have seen in the film how the miraculous code breakers, even with odds of at least 159 million million million to one, were able to do their job. For those that did not choose to see the film the elegant exposition by our President of the intricacies of these machines made a very good substitute.

The presentation ended as it began with rousing music during which the names of those who had co-authored papers in the long and distinguished career of our President were up on the screen.

The Immediate Past President, Dr. Anne Florence, gave a vote of thanks and the company retired to the Conservatory for the Presidential Reception.

Over 100 members and their guests attended.

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## **9 NOVEMBER 2001**

**Minutes of the November meeting which was a joint meeting between the Section of Anaesthesia and Obstetrics and Gynaecology and was held in the Barnes Hall on November 9<sup>th</sup> at 18:00 hours. 97 Members and Guests were present.**

The meeting was opened by the President of the Section of Obstetrics and Gynaecology, Professor Linda Cardozo, and commenced with readings of the minutes of the sections' previous meetings.

Professor Anthony Adams took the chair for the first half of the meeting that concerned *Consent and Awareness*.

### ***Consent Without Awareness: Dr Stephen Yentis of the Chelsea and Westminster Hospital, London.***

Dr Yentis' lecture concerned the difficulties of obtaining consent for obstetric procedures. The dilemmas included the amount of information to give, what information and when it should be given. Studies from the UK had shown that women got most of their information from midwives and the media and in the USA, in addition, from obstetricians. However, for epidurals, midwives were more worried about their effects on progress of labour and delivery and backache, but these were not the main concerns of anaesthetists. The information which patients got from the media was perverse with some lurid and scandalous distortions of the truth.

It was clear that proper information in the form of leaflets and videos were needed but this had proved too expensive for his Trust to issue. Lectures received antenatally had not been properly assessed they probably provided little long-term information to the parturient.

As far as consent to obstetric procedures is concerned, do the patients want to know what could go wrong? Should we give the patient what they want? How do we resolve the issue of rarity and severity of complications? Studies have shown that women want to know everything in Canada and also at the Chelsea and Westminster Hospital. At what stage should this information be given? Do they remember when it is given antenatally? Can women give informed consent in labour? What DO we tell them?

Complications raised included headache, inadequate block, backache and neurological damage. Dr Yentis felt that the future was better education with perhaps an exhaustive information sheet given to every patient as was done in some hospitals in the USA.

He concluded that consent without awareness is common, that most women want to know more and, not surprisingly, there are more questions than answers to this difficult area.

***Awareness Without Consent: Professor Alan Aitkenhead, University Hospital, Nottingham.***

Professor Aitkenhead is Medical Advisor to the Medical Defence Union on anaesthesia. There were major problems with legal action and awareness. He explained the relationship between awareness and memory and the role of implicit memory that was related to depth. The main problem area was spontaneous recall without amnesia. He noted that the original two general anaesthetics in Boston in 1846 had an incidence of 50% awareness!

Awareness in non-obstetric practice in the last decade was nearly 0.1 to 0.2% of cases. Total intravenous anaesthesia showed a similar incidence. Awareness during LSCC was now about 1.3%. Patient satisfaction surveys in Australia had revealed high satisfaction but 0.9% were dissatisfied. Moderate or severe pain had a 4 x odds ratio of causation of dissatisfaction, as did nausea and vomiting, and these were the main reasons for complaint. However, awareness was 54 times more likely to lead to dissatisfaction and litigation.

In Professor Aitkenhead's practice it was the third most likely cause of litigation and there were about eight cases per year with no decrease year on year so far. Obstetrics and Gynaecology cases account for a quarter each of all complaints. Awareness is still a prominent issue. For the non-anaesthetists in the audience, he explained the triad of anaesthesia pointing out that the analgesic component is the same as reflex suppression. There was a continuum of anaesthesia where as it deepens beyond the analgesic stage, amnesia occurs and then there is loss of autonomic responsiveness and then loss of motor response. However, even at this stage consciousness can occur due to surgical stimulation, the bane of stable anaesthesia!

Complaints from patients who were aware included hearing what was going on, vision, touch, paralysis, the fear of anticipation of surgery and of impending death. Main causes of awareness included faulty technique and failure to check apparatus. False claims are rare but exaggerated claims are more common. Sometimes there is genuine misinterpretation of the facts, e.g. awareness on recovery from anaesthesia. Sometimes there are justified risks, e.g. low blood pressure, haemorrhage, anaphylaxis, total spinal and difficult intubation where saving life is more important than preventing awareness.

Why avoid awareness? This was for humanitarian reasons and the avoidance of Post Traumatic Stress Disorder rather than simply to avoid litigation. Management of awareness occurring during anaesthesia was important. Accept a report of awareness as credible, listen to the patient, provide counselling but only if it is requested. Follow up the patient and keep the GP informed.

In his practice, 20 out of 117 cases were defensible and mainly related to postoperative events. Civil litigation is uncommon with only six cases going to trial. For compensation and successful litigation there needs to be proved a breach of duty, injury occurring as a result and a causal link between the two. Although awareness during GA is low there was an increasing need to warn patients of pain during LSCS under epidural and the risk of failure. In 20% of cases this can lead to stress disorder.

***Awareness With Consent: Archive film by Michael Tunstall presented by Dr. David Green, King's College Hospital, London.***

The film concluded the first session and provided an enlightening historical perspective to the whole subject of awareness. Tunstall had popularised the isolated forearm technique in obstetric anaesthesia whereby one arm was isolated from the effect of the neuromuscular blocking drug, suxamethonium. He found many cases of 'amnesic wakefulness' where the patient was transiently awake but could then be deepened to an ideal level of anaesthesia where she was unresponsive. Patients could not recall such events postoperatively. During one such episode, the patient was so purposeful that, with her consent, he allowed her to remain awake until the baby was born. For a subsequent LSCS she requested the same technique, i.e. Awareness by Request and it was the process of this anaesthetic that was the subject of the film.

**A 45-minute break was taken for a hot buffet Dinner.**

***Professor Michael De Swiet, Obstetric Physician at Queen Charlotte's Hospital, London.***

Prof. De Swiet gave a brief overview of *The Place of Regional Block in Medical Complications of Pregnancy*. He appeared confident that the likelihood of serious neurological complications due to bleeding from epidural/spinal needles was almost non-existent. This view was not generally supported — and certainly not by the anaesthetists — because he did not acknowledge the care that was currently taken to avoid the risk of such a disastrous complication.

The final speaker was:

**James Drife, Professor of Obstetrics & Gynaecology in the University of Leeds.**

His subject was *Lessons from the Confidential Enquiries Into Maternal Deaths*. He explained that the next Triennial Report on this subject was due to be published in a month's time and was embargoed so he could not divulge all the details, at least not on his slides. He thus had to confine himself to 'asides' during his excellent presentation which gave the audience indications of what aspects of contributions to maternal mortality were improving and which were becoming proportionately worse. He demonstrated the enormous reduction in maternal deaths in the UK during the 20<sup>th</sup> century, and the smaller reduction in neonatal loss, and contrasted this to a worldwide situation where a very high maternal mortality rate persisted in many countries. The lessons from the CEMD still needed to be applied in many parts of the world.

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**7 DECEMBER 2001**

**Minutes of the December meeting which was held at 17:30 hours in Barnes Hall, RSM.**

**It was entitled: *Neuropathic Pain: One Treatment Fits All?***

**42 Members and Guests were present.**

The President opened the meeting and asked for the minutes of the previous meeting to be read by the Hon Sec. He then introduced the evening's programme paying particular thanks to Dr. Wolfgang Hamann who had organised the meeting and to Pfizer Ltd., the sponsors.

**The first presentation was by Dr WC Hamann and his lecture was entitled: *The Pathophysiology of Acute Pain Conditions*.**

As in other clinical conditions there was a need to link mechanisms to forms of treatment. Classification of pain includes nociceptive, behavioural and neurogenic, of which neuropathic pain is a subset. Assessment of the neuropathic pain patient includes history, clinical tests, imaging, sensory testing, intravenous testing, nerve blocks and conduction studies. The mechanism of peripheral sensitisation includes down regulation of  $\mu$  receptors, less descending inhibition and the effects of central sensitisation.

Dr Hamann commented that now we recognise phantom limb pain as a clinical entity it is now increasingly diagnosed with a 50% incidence following amputation in some series.

His group had noted a sustained increase in mast cells appearing in injured nerves that became more intense for up to 6 months. Their precise role in neuropathic pain was as yet to be established. Particularly interesting was the mosaic of responses to pain testing procedures in patients with neuropathic pain. These included hypo- and hyperalgesia as well as allodynia and spontaneous pain — all occurring in a confined area. It was clearly not an all or none response.

The differences between stump pain versus phantom limb pain and the effect of opioids were elegantly demonstrated with remifentanyl. Its effect on stump pain is proportional to dose but has little effect on the continuous phantom limb component.

He finished with images of brain PET scans in a patient with mononeuropathy. Clearly there was not as much knowledge of the cause of central pain as with peripheral pain.

**The second presentation was by Professor Troels Jensen of Aalborg University and his lecture was entitled: *Drug Treatment of Neuropathic Pain*.**

Although the prevalence of neuropathic pain was not rare the numbers were fairly small numbers, e.g. painful diabetic neuropathy had an incidence of 0.5/1000. However, the incidence of neuropathic pain following surgery is much higher than is generally thought, e.g. following CABG it is 56%, thoracotomy up to 61% and hernia repair up to 11%. Symptoms may include ongoing or continuous pain and evoked pain. The mechanism and treatment of these two components is different. In addition, removal of a neuroma may lead not only to pain but also hyperalgesia and allodynia. Sprouting C fibres lead to central sensitisation in the spinal cord with A fibre changes leading to an expansion of the pain area to beyond the site of injury. Professor Jensen pointed out that hyperexcitability, exhibited by hyperalgesia and allodynia were a main component of neuropathic pain and could be modified by anti-hyperalgesic drugs

Types of drugs that could be used include:

- Traditional anti-depressants
- Na<sup>+</sup> channel blockers, such as lamotrigine and gabapentin, which are sometimes very effective in diabetic neuropathy
- NMDA antagonists, such as ketamine, that work in the dorsal horn
- Opioids
- Sympatholytic, such as clonidine
- Topicals, such as lignocaine and capsaicin.

McQuay and Moore have proposed that the number needed to treat (NNT) should be universally adopted to record a particular effect for analgesic drugs. The NNT50 is the NNT to achieve 50% pain relief. This is a very useful concept and allows us to compare the effectiveness of different drugs in neuropathic pain, a figure of 2 or 3 indicating a useful analgesic effect. Indeed, for most antidepressants, the NNT50 is 2 to 4, whilst for the Na<sup>+</sup> channel blocker mexiletine, the figure is approaching 10.

**The final presentation was by Dr Nicholas Padfield and his lecture was entitled: *Invasive Treatments of Neuropathic Pain.***

Dr Padfield explained the role of invasive treatment was in those patients who had low compliance with treatment, where there was a failure of medication or a desire for a 'cure'. Great pressure is placed on pain specialists to be invasive in these circumstances and certain caveats should be noted. Patients have unrealistic expectations of invasive treatment so do not promise a cure. A reduction in pain is not necessarily curative, as it may be a small part of a multidimensional problem.

He then elaborated on types of treatment explaining the role of surgical neuroablative procedures is much diminished now. The success and safety of techniques such as chemical and thermal neurolysis, neuromodulation and pulsed radiofrequency lesions are much improved due to better equipment. Many are now done percutaneously.

It is most important for the doctor and patient to weigh up potential benefits against side-effects of treatment. For example, an intrathecal phenol block may effectively reduce pain but lead to loss of bladder control and difficulty in walking. Patients must be counselled about these possibilities.

Dr Padfield then took us through a wide spectrum of different techniques such as chemical and thermal neurolysis, lumbar facet rhizotomy and cordotomy. Following the latter PET scans had shown increased blood supply in the hemithalamus contralateral to the lesion. This was evidence of effectiveness. He ended his presentation by describing peripheral nerve stimulation, spinal cord stimulation and intraventricular drug delivery. New implantable stimulators are fully programmable and controllable percutaneously. And he might have added, very expensive!

**I FEBRUARY 2002**

**Minutes of the February 2002 meeting which was held at 17:30 hours in Barnes Hall, RSM.**

**It was entitled *The Laryngeal Mask.***

**45 Members and Guests were present.**

The President opened the meeting and asked for the minutes of the previous meeting to be read by the Hon Sec. He then introduced the evening's programme paying particular thanks to Dr. Archibald Brain and to Intavent Orthofix, the sponsors.

**The introductory presentation was by Dr Archie Brain, the inventor of the Laryngeal Mask and his lecture was entitled: *History and Introduction.***

Dr Brain began his presentation by considering the risks of introducing new techniques and skills. He pointed out that there had been more than 7000 prototypes tested before the clinical introduction of the laryngeal mask. Many of these had required use in real patients and this 'experimental phase' had been enabled by one ethical committee decision. He wondered whether, in the pursuit of excellence, that such trial and error approaches to the development of new apparatus would be possible and what chance would a new 'inventor' have of evolving a new device in the future?

He then gave an insight into the development, initial testing and introduction of the first laryngeal mask (LM). The crucial role of Dr John Nunn was alluded to. The LM and its insertion had appeared to be deceptively simple but Dr Brain emphasised the importance of 'hands on' versus video training in gaining expertise and reducing complications. Although the use of IPPV through the LM was still questioned in some countries, evidence

overwhelmingly attested to it being safe practice. Dr. Brain finished his presentation on aspects of development and use of the new LMs, the Fastrach, Proseal and disposable versions.

**The main presentation of the evening was by Dr David Ferson of the M. D. Anderson Cancer Center, Houston, Texas, USA and his lecture was entitled: *New Development, New Uses.***

Dr Ferson had gained considerable experience in the use of the LM in the hospital setting. He illustrated his experience with the liberal use of photographs and video clips. He divided clinical use of the LM into various areas, such as:

**Outside the OR:** in the radiology department for scanning, the cardiac department for Transoesophageal echocardiography and the endoscopy suite. He noted that the LM could be used alongside large 'scopes such as for TEE and OGD although in the former the TEE had to be placed first.

**Head and neck:** he noted the pioneering contribution of Dr Paul Bailey in use of the LM during tonsillectomy and the extension for patients with difficult airways or intubation. In particular, the LM allowed more accurate and safe use of the laser on airway tumours.

**Thoracic surgery:** fiberoptic bronchoscopy preceded thoracotomy in all patients in Houston so that inoperable tumours could be excluded. However, the presence of the ET tube had prevented some high lesions being visualised. Use of the LM in association with fiberoptic bronchoscopy had allowed a better visualisation of the upper airway and had revealed inoperable tumours that would otherwise have proceeded to a pointless thoracotomy. In a similar way, it had allowed for the use of high stents in airway obstruction.

**Neurosurgery:** the LM was particularly useful for stereotactic surgery and surgery where speech had to be assessed during the procedure. It was also used for replacing the endotracheal tube (ETT) at the end of surgery so that the patient would wake up with the LM rather than the ETT that tended to cause straining and coughing.

**Difficult airway:** Dr Ferson pointed out the evolutionary role of the LM in the ASA *Difficult Airway Algorithm* from a last ditch use to one where the LM is now recommended at most of the stages of the algorithm. The development of the Fastrach device together with the specially designed ET tube had revolutionised difficult airway management especially in the trauma situation.

At the end of his excellent presentation, Dr Ferson considered some of the problems of the LM such as whether or not to ventilate, problems with aspiration and the role of the Proseal mask. To conclude, he had used the LM to provide airway management in the chimpanzee and thus noted that the LM, having been safely used in humans, was now appropriate for animal use too!

The meeting ended with a few questions from the floor.

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## **1 MARCH 2002**

**Minutes of the March 2002 meeting which was held at 17:30 hours in Barnes Hall, RSM.**

**It was entitled: *Neuropathic Pain: Meeting 2: Chronicity and Acute Pain.***

**50 Members and Guests were present.**

The President opened the meeting and asked for the minutes of the previous meeting to be read by the Hon Sec. He then introduced the evening's programme paying particular thanks to Dr. Magdi Hanna of King's College Hospital, London, the organiser of the meeting.

**The first presentation was by Professor Martin Koltzenburg of the Institute of Child Health, University College London and National Hospital for Neurology and Neurosurgery.**

Professor Koltzenburg began by explaining that acute pain pathways are transient and very high fidelity, e.g. the pain of venepuncture. However, tissue damage such as in arthritis and chronic conditions such as post-herpetic neuralgia could lead to chronic pain and this involved nociceptor sensitisation. Peripheral changes could trigger central changes and primary nociceptors are the main site of this sensitisation.

A great advance was the cloning of the capsaicin receptor in 1997. It is also activated by heat and protons. Since then a number of receptors have now been identified including VR1 (the capsaicin receptor) and CMR 1 that responds to cold. Identification of these receptors leads to the possibility of antagonists that are being explored by pharmaceutical companies.

Professor Koltzenburg then outlined the changes in nociception following injury. Normally nociceptors respond to strong stimuli. In inflamed tissue you get spontaneous discharge and a stronger than normal response. This leads to increased pain, hyperalgesia and allodynia.

Paw withdrawal tests in animals lacking the VR1 receptor show no response to inflammatory stimuli demonstrating the importance of this receptor. Bradykinin is the most potent substance involved in nociception but trkA and nerve growth factor are also implicated in chronic pain. He explained that primary hyperalgesia is mediated by nociceptors and peripheral sensitisation and second hyperalgesia by central sensitisation

Current analgesics have problems with lack of effect, side effects and a high value of number needed to treat (NNT). NSAIDS had increased side effects with increasing dose and age of patient. Both renal failure and chronic heart failure are exacerbated.

In conclusion he looked at the activity of NMDA as being an important receptor that could allow new drugs to be developed. Alongside NMDA, the NK1 receptor is another avenue that is being explored but has not yet produced any patient benefit.

**The second presentation was given by Dr John Brown of King's College Hospital, London, and his lecture was entitled: *Can Effective Management of Acute Pain Prevent the Development of Chronic Pain?***

The transition from acute, surgical pain to a sub-acute condition with added illness and anxiety was a chronic transition involving complex processes of peripheral and central sensitisation. 'Prolonged pain burns its way into the CNS'.

He began by tackling the subject of pre-emptive analgesia (PEA) that was successful in animals but has not been fully substantiated in human trials. Various techniques were described, such as local anaesthetic blocks, caudals and epidurals given prior to surgery, e.g. hernia repair, but few of the studies showed a positive pre-emptive effect. In studies using systemic opioids prior to abdominal hysterectomy, some showed a PEA effect whilst others did not. In dental studies, NSAIDs had shown no PEA effect. A better response was found using multi-modal techniques, e.g. using combination treatment in thoracotomy. Why is PEA not effective in human beings? Maybe it is not clinically significant or general anaesthesia and opioid, such as fentanyl, mask it. He explained the *Hydra principle* by which postoperative pain itself can trigger sensitivity and thus must be completely blocked for PEA to show any effect.

How do we define chronicity? A period of 2 months is often cited but it depends on the operation itself and patient factors. Causes of chronicity were outlined, e.g. type of incision, haematoma and even type of suture. Different operative sites had different potential to produce chronic pain and in breast surgery Kalso has related the intensity of chronic pain to the severity of postoperative pain. Dr Browne then discussed the role of PEA in thoracotomy and inguinal hernia where chronic pain may be present in up to 63% of patients after 1 yr and in 10 to 20% the pain is severe.

Finally he discussed chronic pain states such as post-amputation pain where the evidence to date of prevention with epidurals is not conclusive. In post-herpetic neuralgia prevention by acute treatment with antiviral drugs may possibly alter the duration of pain if not the incidence.

**The third and final presentation was by Dr Sue Peat, also of King's College Hospital, London, and the title of her lecture was: *Recent Advances in the Management of Chronic Pain.***

Neuropathic pain (as a form of chronic pain) is often described by patients as a 'burning, tingling sensation like an electric shock'. The general principles underlying treatment is that a complex situation with multiple causes and mechanisms therefore needs a number of different drugs. The concept of number need to treat (NNT) or to harm (NNH) was useful and indicated how relatively ineffective these drugs were in comparison to general anaesthetics, for example where the NNT is 1, unlike 4 or 5 with some chronic pain drugs. Treatment usually involved what Dr Peat described as 'rational polypharmacy'.

Most of the drugs are not licensed for the use to which they are put in chronic pain as they are not analgesics. Patients must know this and accept the risks of 'off license' prescribing. Dr Peat completed her presentation by describing the use of some of these drugs in therapy. These included carbamazepine and oxcarbazepine its new keto analogue that demonstrates no auto-induction. Gabapentin has a licence for neuropathic pain but although much lower doses are needed than in epilepsy, patients still get side effects! Topiramate is another promising new drug with some studies showing 70% improvement. A major side effect is weight loss that may be dramatic. The drug also improves glycaemic control in diabetics.

Questions from the floor followed each presentation.

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## 5 APRIL 2002

**Minutes of the April 2002 meeting which was held at 17:30 hours in the Barnes Hall, RSM.**

**It was a meeting organised by the Surgical Outcomes Group.**

**52 Members and Guests were present.**

**The first lecture was entitled: *Does Perioperative Management Make a Difference?* given by Professor David Bennett, Department of Intensive Care, St. George's Hospital, London.**

He began by saying that tissue PO<sub>2</sub> and PCO<sub>2</sub> had been measured during major operative procedures. In some cases very low PO<sub>2</sub> and pH values had been recorded. Experimental work in animals had shown that prolonged hypoxia produces deleterious effects on capillary endothelium. If tissue PO<sub>2</sub> was less than 2 kPa this leads to reduced adenylyl cyclase, cAMP and cGMP. The result was increased capillary permeability, pro-coagulation and production of activated protein C. Increased expression of adhesion receptors together with polymorph (PMN) activation and hypoxic vasoconstriction leads to tissue damage. When tissue blood flow and oxygen is restored (e.g. during resuscitation) the deleterious effects of reperfusion become evident. Reactive oxygen intermediates (ROIs) are the prime cause of this damage. The major source is the xanthine oxidase system and is exacerbated by PMN activation. ROIs accentuate organ injury. The implications of hypovolaemia and metabolic stress with tissue hypoxia led researchers to wonder whether there was a window of opportunity to prevent the hypoxia developing.

A study of supplementary, perioperative oxygen therapy published in the *New Engl J Med* suggested a reduction in wound infection in colorectal surgery. In this study, 30% O<sub>2</sub> or 80% O<sub>2</sub> was administered during surgery and for two hours afterwards. Patients were evaluated until discharge. Subcutaneous PO<sub>2</sub> and muscle PO<sub>2</sub> were much higher in the 80% O<sub>2</sub> group. The effect was to halve the number of wound infections. However, there was no difference in hospital stay.

Warming has also been shown to reduce wound infection and antibiotic use. One study showed a significant reduction in hospital stay.

In higher risk patients, Shoemaker had shown that if O<sub>2</sub> delivery is low (< 300 ml m<sup>-2</sup>) patients have high mortality, which goes down to 10% at 500 ml m<sup>-2</sup> and when over 600 ml m<sup>-2</sup> there was zero mortality. He found that O<sub>2</sub> debt is also a predictor of mortality especially if the O<sub>2</sub> debt is increasing. Predictive criteria of higher risk patients were then produced such as massive blood loss, chest infection, respiratory failure and so on which were shown to worsen outcome.

Can we do something about it? Is it a real problem? 30,000 patients die postoperatively in the UK, mostly in the early postoperative period but some die later up to 30 days or more following surgery. He had studied ICU mortality using the England and Wales ICU ICNARC data set looking at all patients going to 90 ICUs who had undergone major surgery. There were 26,000 elective and 12,000 emergency cases. There was 24% mortality, overall, with 11% and 37% in the elective and emergency surgery. Median length of stay (LOS) was over 24 days for these patients and thus there is massive consumption of resources. Interestingly, Scotland has much lower mortality in ICU than England and Wales and this maybe due to higher resources for the Scottish healthcare system.

These findings have led to what became known as *goal directed therapy*. Shoemaker produced a classic, but controversial, paper in 1988 that showed a reduction in mortality from 30% to 4% in the control versus the treatment group. Wilson *et al* in the BMJ 1999 also showed a large reduction with preoperative dopexamine or adrenaline plus fluids pre treatment. These were elective patients. Bed days were reduced with dopexamine and adrenaline. A similar finding was seen in a Brazilian study.

Using the Oesophageal Doppler Monitor (ODM) and goal directed fluid administration; Sinclair *et al* also showed a large reduction in hospital stay in fractured neck of femur patients from 15 to 9 days. Reduction in organ dysfunction and ICU stay and also re-admission to ICU was shown in a study in cardiovascular patients by Polonen *et al*. A 50% reduction in mortality was noted for up to 12 months after surgery.

How then do we reconcile the results of the famous Mangano study of *beta blockade* with atenolol that also showed a beneficial effect? This is of course likely to reduce Q and O<sub>2</sub> delivery. Bisoprolol has also showed similar effects in vascular surgery. Reduction in mortality from 17% to 3% and also of non-fatal MI was observed.

## **Conclusion**

There are 3 million operations a year in the UK from which 30,000 patients will die in the first 28 days. Wound infection increases hospital stay and tissue hypoxia may be a significant causative factor. Normothermia and high PO<sub>2</sub> can also have a profound beneficial effect. If the patient has myocardial disease, beta-blockade may also be effective. Increased O<sub>2</sub> delivery can decrease mortality. and meta analysis of well-controlled studies (of which there are few) shows evidence that this is effective.

## **The second lecture was by Dr Mike Grocott, formally Research Fellow, Surgical Outcomes Research Centre, UCLH. His lecture was entitled: *Can Surgical Outcome Be Measured?***

Why measure surgical outcome? A number of studies have shown that mortality can be reduced. International comparisons of mortality show marked differences with the UK often doing worse than other countries. In the future we are going to be required to measure outcome, e.g. following the Bristol Inquiry into cardiac surgery, outcome by individual cardiac surgeon is now required. Other surgical specialties outcomes will also be measured in the future.

How well do our patients actually do? We often don't know. What is surgical outcome and how can we quantify it? We can use cost, length of stay, quality of life but we are surely more interested in morbidity and mortality. Measurement of mortality and adverse effects in the perioperative period show that death rates although low are significant

Length of stay in hospital or ICU is not entirely dependent on the patient as it also reflects other factors such as discharge criteria, availability of step-down facilities, and so on. Studies show quite marked difference in outcomes for similar patient groups.

Postoperative morbidity following surgery has been assessed using patient interview and then categorised by an independent panel. Nine morbidity sub scales can be identified such as gastrointestinal (GIT), pulmonary and renal. Prospective studies of major surgical patients at Duke University in the USA showed that GIT dysfunction was a major cause of morbidity. Other factors included age, ASA, length of operation and blood loss.

Dr Grocott then outlined a prospective study that is now being undertaken at UCLH. The need to have a definite measure for morbidity was noted and one must have a believable measure and then make sure it varies with the things that you expect it to vary with e.g. severity of the illness. POSSUM (Physiological Operative Severity Score for the enumeration of mortality) may be able to predict outcome).

## **Conclusion**

Surgical outcome is worth measuring because it can be improved. There are many dimensions and it needs validation by prospective studies.

## **The third lecture was given by Professor Monty Mythen of UCLH and was entitled: *Variation In Surgical Outcome — An International Comparison.***

He began by reflecting on the fact that published mortality is twice as high for abdominal aortic aneurysm and cardiac surgery in the UK versus the USA. However, in coronary artery bypass graft surgery mortality is nearly the same, but slightly higher in the UK. He commented that this was despite the fact the service delivered, equipment and staffing is the same for cardiac surgery in both countries. All patients traditionally go to ICU despite the low mortality in this group (2 to 3%) versus emergency general surgery where the mortality is 30%.

Comparative multi-centre trials suggest that the UK is not as good as other centres in Europe. In one study, mortality was four times higher than some other countries despite similar POSSUM scores preoperatively. The ITU literature, however, shows that UK patients are just as likely to survive which suggests that the level of care is as good once you get to ICU. However, the big difference is the much smaller chance of getting into an ICU bed in the UK in the first place!

Are things getting any better? For NCEPOD 2000, a 10% random sample (cf. 20% from 1990) was taken of the 19,000 deaths following surgery. Fifty percent of patients who died postoperatively never saw an ICU; 5% were refused admission; 50% of hospitals had no HDU facilities. Thirty-three percent of those who died had not seen a consultant surgeon or anaesthetist. This was obviously unacceptable. The media has exploited the how death rates seemingly vary between hospitals, e.g. Kettering and Rotherham with 1/6th to twice the average number of deaths respectively.

However, in most studies, including those alluded to earlier, no case mix adjustment was made except for major surgery. Adjustment scoring systems include the Parsonnet and POSSUM scores for surgery and Apache scoring for ICU. With POSSUM one can compare the observed to expected ratio to compare your results with what is predicted. If the ratio is 1 or less you are doing fine.

He then presented the results of a trial entitled *Case mix adjuster mortality following major surgery*. This compared the USA and the UK with two prospective cohorts of 1000 (USA) and 1500 (UK) patients. Mount Sinai, New York and Portsmouth in the UK were the chosen sites. Although the UK had slightly sicker patients the patients seemed comparable. Results showed that UK patients were four more times likely to die than the US cohort, a highly significant result. The higher incidence of mortality in the UK may be due to the following differences in practise. In Mount Sinai no patients are refused admission to ICU and consultant surgeons and anaesthetists see all the patients.

Could the results also be due to better funding? A recent study published in the *BMJ* of Kaiser Permanente Health Maintenance Organisation (KPHMO) in California versus the NHS contradicts this view. The KPHMO budget is similar to the NHS and thus the study does not support lack of funding as the inherent cause of poor outcome in the UK.

### **Conclusion**

However, health resources are much lower in the UK with 3.5 times more ITU beds in the USA. At the end of the day, life expectancy is as long in the UK or USA and infant mortality is lower. Difficulties in comparison with the US emerge due to lack of sources of published data and the predominantly private versus public health care systems. There are, for instance, no similar data sets to NCEPOD in the USA.

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## **FRIDAY 7 JUNE 2002**

### **New Monitoring in the Operating Suite and State of the Art. What should we be using?**

#### **WINDOWS ON THE BRAIN AND CIRCULATION**

#### **MORNING SESSION: WINDOWS ON THE BRAIN**

**Chair:** *Professor Anthony P Adams, President of the RSM Section of Anaesthesia*

#### **MEASURING OXYGENATION TO THE BRAIN**

##### **Introductory remarks**

*Professor Anthony P Adams, President of the RSM Section of Anaesthesia*

The President opened the meeting by explaining that exciting developments in monitoring were now taking place resulting in new non invasive or minimally invasive monitors. The main emphasis was on patient safety. The morning session would look at monitoring the central nervous system and the afternoon session the cardiovascular system.

##### **The Invos Cerebral Oximeter: a new trend monitor of brain haemodynamics**

*Harvey L Edmonds, Professor and Director of Research, Department of Anesthesia, University of Louisville, Kentucky, USA*

Professor Edmond's lecture concentrated on brain injury and its prevention and the role of the Somanetics cerebral oximeter. This device measures regional oxygen saturation in the brain (RSO<sub>2</sub>, see later). Recent studies have shown that persistent neurocognitive decline (NCD) following surgery has a much greater impact on overall morbidity than previously recognised. After adult cardiac surgery it is up to 40% and even after general surgery in the over 65's the incidence is 11%.

It is clearly a cause for concern. Current practice relies on so called 'mid range' normal data based on population means rather than individual values. Animal experiments with the RSO<sub>2</sub> show that a value of less than 30% is associated with ATP decline and EEG changes. This may be associated with NCD and brain injury. In cardiac surgery 38 to 50% of patients at some point will suffer a decline in RSO<sub>2</sub> to 25%.

There is some confusion as to the impact of these changes. In the legacy literature clinicians made decisions based on oxygen input and output (e.g. using jugular venous oxygen tension, JVPO<sub>2</sub>) and did not look at changes at tissue level or in the capillary bed. This is because the technology for measuring this has only recently become available. For example, an AV shunt could cause regional hypoxia yet the a-vO<sub>2</sub> difference and JVPO<sub>2</sub> would be normal. This has been demonstrated in animal studies where dogs anaesthetised with isoflurane had catheters to measure PO<sub>2</sub> in the sagittal sinus and electrodes inserted to measure brain cortical tissue PO<sub>2</sub>. The same degree of hypotension obtained by increasing isoflurane or using sodium nitroprusside (SNP) showed no change in venous PO<sub>2</sub> (cf. JVPO<sub>2</sub>). However, cortical tissue PO<sub>2</sub> showed a marked decline with SNP hypotension but not with isoflurane. This indicates that tissue PO<sub>2</sub> is a much more sensitive discriminator of hypoxia than the overall

supply/demand value obtained with  $JVPO_2$ . Thus it is only with the new technology such as  $RSO_2$  that we can pick up these changes during anaesthesia.

The ability to measure  $RSO_2$  depends on two facts. The first is that the human skull is translucent to visible and infrared (IR) light and the second is that photons don't have to travel in a straight line but can be bent. The Cerebral Oximeter has two spectrophotometric probes which pulse visible and IR light at two angles. One is reflected by the brain surface and the second by the deep cortical tissues in the 'watershed area' between the territory of the middle and anterior cerebral arteries. These reflected signals are picked up by a single sensor. The differential gives an overall value of tissue  $O_2$  saturation (or  $RSO_2$ ) in this area that is neither venous nor arterial nor pulsatile. It thus has no direct relationship to values obtained from  $JVPO_2$  or pulse oximetry. The value obtained is constant for an individual but there is no absolute 'normal' value. In 250 patients where  $RSO_2$  was measured before surgery, the mean was 65 but the standard deviation was 9 giving a normal range 47 to 83. The value is less in liver disease or CCF. The  $RSO_2$  is dependent on total Hb, oxygenation and the degree of perfusion. Thus, the value will fall if any of these values decrease. At a constant Hb and arterial saturation the value will depend on cerebral perfusion e.g. during carotid endarterectomy the value will fall following clamping of the ipsilateral artery if the collateral circulation is inadequate, thus predicting the need for a shunt.

Chromophores may affect the signal. Melanin and skin pigmentation have no effect but dyshaemoglobins may affect the reading as only two wavelengths are used in the transmitter. Indocyanine green and methylene blue do alter the signal and may produce spurious results.

What is the evidence that the device works? In radiological balloon occlusion of major cerebral vessels (e.g. in giant aneurysm) there is a large fall in  $RSO_2$ . The deVries study of  $RSO_2$  versus  $JVPO_2$  during cardiac pacemaker testing showed that the former tracks falls in cerebral perfusion during experimental ventricular fibrillation rather than after the event with the latter. The fall in  $JVPO_2$  only occurs after flow is restored. Thus the  $RSO_2$  measures imbalance in oxygen supply and demand in a localised area.

Other uses of the cerebral oximeter were presented. The US Air Force had problems with pilots during high G forces blacking out. A 100 tonne centrifuge capable of inducing 14G forces is used as a test bed. The  $RSO_2$  value falls just before blackout and in 90% this presyncopal fall precedes full syncope by 5 seconds. On a simpler level, using a tilt table test for syncope, a 30% decline in  $RSO_2$  also predicts impending syncope.

Changes in perfusion and  $O_2$  delivery can be demonstrated by  $RSO_2$  during respiratory obstruction, vasospasm, hypoxia and anaemia. Falls may also be seen unexpectedly following head rotation for central venous line insertion and is corrected by returning the head to the neutral position. If prolonged this may lead to NCD. Other uses include aortic aneurysm repair and antegrade cerebral perfusion during circulatory arrest. Here retrograde flow using the venous system is sometimes employed with drainage through the open arteries into the chest. Both transcranial Doppler and Cerebral oximetry predict the effectiveness of flow.

Transfusion strategy can be based on  $RSO_2$  information during major blood loss as there are great differences in the effect of blood loss on  $RSO_2$  between patients. Those patients who maintain  $RSO_2$  can have transfusion withheld. As the  $RSO_2$  is a measure of supply/demand, reducing  $O_2$  consumption, e.g. by using propofol can improve  $RSO_2$  in an almost dose-related manner. In some patients a small fall in BP can cause a major fall in  $RSO_2$  due to a waterfall effect. In other patients the same fall produces no change, thus there may be adequate autoregulation in one patient but not in another.

There are limitations as the cerebral oximeter only looks at a small area of brain so other areas may become ischaemic without any change in  $RSO_2$ . However, it does 'look at' the watershed area which is an area most prone to becoming ischaemic due to falls in perfusion. The probe can't be placed on skin with hair and must go directly onto smooth skin (like an ECG electrode) so this also limits the parts of the brain it is 'looking at'. There is no absolute 'normal' value (like  $SpO_2$ ) and it is therefore only a trend monitor. It is affected by dyshaemoglobins and dyes (see above).

How does it impact on practice? Although there are no published randomised controlled trials, evidence is accumulating in cardiac and other surgery that interventions to normalise  $RSO_2$  reduces neurological and NCD, time for postoperative ventilation, time in ICU, hospital stay and, most importantly, cost! Although massive multicentre trials would be needed to establish that  $RSO_2$  monitoring reduced the incidence of stroke (say) much smaller trials would probably demonstrate major changes in NCD and overall patient cost.

## **AWARENESS IN ANAESTHESIA**

### **Introductory remarks**

*Professor Anthony P Adams, President of the RSM Section of Anaesthesia*

The President opened this session by stressing the increasing importance of this subject in anaesthesia. Although the overall incidence of awareness seems small at 0.1 to 0.2% of all general anaesthetics, it is higher with TIVA techniques and in certain types of surgery such as cardiac, emergency and obstetric. Indeed the Medical Defence Union and the Medical Protection Society are very concerned as claims are increasing in the UK.

### **The Aspect Bispectral Index Monitor**

*Professor Peter Sebel, Professor of Anesthesia, Emory University, Atlanta, Georgia, USA*

Professor Sebel stressed that use of 'awareness' monitors such as the Aspect BIS had implications in anaesthetic practice beyond awareness. It was really about monitoring optimal depth of unconsciousness. In the traditional 'balanced anaesthetic', Gray's triangle stressed hypnosis, analgesia and muscle relaxation. He felt that this should be restated as hypnosis, unconsciousness and lack of memory for intraoperative events.

What is BIS? It is essentially a Fourier transformation of the very complex EEG signal. This is integrated to give a BIS number or 'score' by use of proprietary analysis and algorithms. The number is graded from 100 awake to 0 which occurs when there is a flat line EEG. A level of 70 indicates light anaesthesia with little chance of memory, a value of less than 49 suggests deep hypnosis.

In the USA there have been over 5 million uses of the BIS and it is employed in 9% of all operating theatres. There have been 400 peer reviewed papers. In the UK approximately 8000 cases are monitored per year and in the USA 0.5 to 1 million per year.

Professor Sebel emphasised that studies to indicate the relevance of BIS score must equate clinical endpoints of unconsciousness, codified observer's assessments and drug levels. With BIS during propofol anaesthesia the EEG changes, BIS score, drug level using target controlled infusion and unconsciousness level are consistent. Using the isolated forearm technique (IFT), BIS can also be used to track return of consciousness. At a BIS level of 64, only 13% of patients show adequate response.

Memory performance during anaesthesia has been determined in a Trauma study with varying levels of anaesthesia using etomidate, isoflurane and fentanyl. Tagged words at known BIS levels were presented during anaesthesia. The higher the BIS score the higher the likelihood of memory for the word and procedure. A BIS below 50 showed that memory was unlikely to occur.

Sandin's prospective study of awareness in more than 11,000 patients showed an explicit awareness rate of 0.1% without and 0.2% with the use of neuromuscular blockade. In the Emory system where 50,000 general anaesthetics are administered this would indicate 100 possible cases of awareness. If this incidence of 0.2% awareness was reduced to 0.1% by BIS a properly powered study would need 50,000 patients to show a significant difference.

In the Aspect company database, 5 million cases have been monitored with 82 reports of awareness where BIS monitors have been used. When analysed, 48 had high BIS levels above 65. In others the retrospective data was not available. Although it is likely that aberrant patient behaviour would be seen in the 5 million uses so far it has not actually been found to date. In the latest Sandin study there have been two cases of awareness but with BIS levels above 65. This represents a 78% reduction in awareness. This study is not yet in print. Professor Sebel reported an ongoing trial called Aim which is trying to identify the incidence of awareness in 50,000 patients. 16,000 cases have been enrolled as of May this year (2002).

What about clinical utility studies where BIS has been used versus standard practice? These studies have shown 23% less propofol, faster response to awakening and extubation and quicker discharge or bypass of the postanesthesia care unit (PACU). At Emory a BIS introduction programme with prior training then using a BIS decision matrix has been introduced. In another study of 2,000 patients there was a 30% reduction in operative time and a 60% reduction in intubation rates in PACU. In addition postoperative nausea and vomiting (PONV) was reduced, possibly due to less drug being used?

In previous models of the BIS, EMG interference could interfere with the BIS score so that a high level with high EMG activity would be reduced by the simple administration of vecuronium. The new XP model addresses this problem.

In summary, apart from reducing the incidence of awareness, use of BIS leads to less drug usage, faster wakeup, more alert patients with earlier discharge from PACU and a reduced incidence of PONV. There are, however, limitations. BIS is a state monitor not a predictor of response to a surgical stimulus. This should always be born in mind when keeping to a particular BIS level in the absence of a surgical stimulation.

### The Fathom Sinus Arrhythmia Monitor (SAM)

*Dr Chris Pomfrett, University Department of Anaesthesia, Manchester*

Dr. Pomfrett explained that he was the co-inventor of the SAM with Professor Healy and Dr. Robert Sneed of Manchester University in 1990. He emphasised that the SAM was now licensed to Amtec medical company in Northern Ireland but the monitor was not as yet freely available. Very little research had been carried out on the monitor (in comparison with BIS) due to lack of availability of monitors. The underlying principle was a loss of the normal slowing and speeding up of the heart which occurs during inspiration and expiration in normal patients. As anaesthesia deepens this 'beat to beat' variation diminishes and is seen as a predictable sign of the onset of unconsciousness. Dr. Pomfrett emphasised that although it was well known that the absence of haemodynamic responses to surgery were a poor predictor of awareness, SAM was completely different. The changes were very small and not visible by looking at an ECG. Although it seems like a very logical device it is dependent on an intact neural pathway of the vagal afferents and efferents so will not work in a denervated heart or when there are dysrhythmias. A spin off of the technology is its use in cattle to predict the onset of new variant Creutzfeld-Jacob disease and Dr. Pomfrett mused on the possibility that this may turn out to be its most useful application.

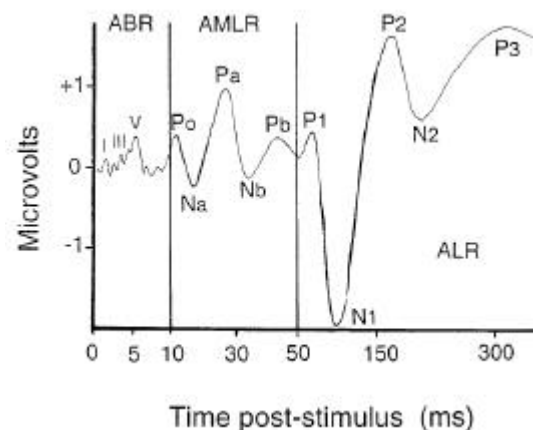
### The Alaris AEP (Auditory Evoked Potential) Monitor

*Dr Douglas Newton, Imperial College School of Medicine at St Mary's, Northwick Park Hospital, London*

Dr. Newton explained that, since 1980, AEPs have been used as a systematic research physiology based tool to monitor awareness at Northwick Park Hospital where his main co-researcher is Dr. Christine Thornton. The AER produces a very small signal versus the EEG. To process a  $0.5 \mu\text{v}$  signal is more demanding than the  $200 \mu\text{v}$  from the EEG. Unlike the BIS, simple electrodes may be used in the future so removing a potential funding stream.

When recording evoked potentials it is necessary to average several hundred individual responses to several hundred repetitions of the stimulus in order to pull the tiny signal evoked by the sound stimulus out of the much larger generalized EEG 'noise' (which may very well carry biologically important information, but which is random with respect to this particular stimulus). A cycle of 256 clicks is needed to pick up the data. A diagram of the general shape of the auditory evoked response in human subjects is shown in the figure. The AER consists of a series of positive and negative waves or peaks.

Fig. 1 shows the three phases of the auditory evoked response. The first phase is the response obtained from the auditory nerve and brainstem (ABR). The second phase is the early cortical response or auditory mid latency



**Figure 1.** The auditory evoked response.

response (AMLR or mid latency auditory evoked response, MLAER). The third phase, the auditory late response (ALR) is obtained from the sensory cortex and represents auditory interpretation. ABR is very insensitive and ALR too sensitive, showing depression even under light sedation. Studies have shown that it is only the MLAER that is sensitive to general anaesthetics and the transition from unconsciousness to consciousness. The latency is increased and the amplitude decreased for waves Pa and Nb in the presence of volatile anaesthetics with these changes becoming more pronounced as end-tidal concentrations of anaesthetic increase until eventually the waveforms are abolished.

During recovery from anaesthesia the amplitudes of Pa and Nb recover. Later studies specifically addressed the relationship of MLAERs to awareness, using response to a verbal command to raise a finger or squeeze the experimenter's hand as a measure of awareness. These studies showed a statistically highly significant relationship between response to command and both latency and amplitude of waves Pa and Nb. The association of MLAER with awareness was also found using the anaesthetic propofol and the measure of awareness was response to a verbal instruction to squeeze the investigator's hand. Repeated transitions from responsiveness to unresponsiveness and back again for each subject showed statistically significant increases in Na, Pa, and Nb latencies when responsiveness disappeared and decreases when it reappeared again.

Historical problems with the MLAER as a useful monitor of awareness revolved around the general method of extracting AEP by use of a Moving Time Average (MTA). However, the MTA is time consuming because a large number of repetitions is needed to produce an estimate of the AEP which could take at least 2 minutes. Hence, changes occurring over a small number of sweeps will not be detected by the MTA average. The AEP was thus a research and not a clinically useful assessment of rapid changes in the conscious state as occur during real time surgery. However, in 1996 Jensen *et al.* described a system-identification method, an autoregressive model with exogenous input (ARX) model, which, together with the availability of increased processing power was to produce a second, sweep-by-sweep estimate of the AEP. This A line ARX (AAI) index is now the basis of the A line AEP monitor. As with BIS, a value of 70 to 100 is found in awake patients and a level below 30 is associated with surgical anaesthesia.

In conclusion, the AER shows graded changes with depth and is similar for different anaesthetics (except, like BIS, ketamine) and responses to noxious stimuli. It is modified by opioids, indicates awareness and can be successfully automated

The claims for the monitor are similar to the BIS but the AER represents a response to stimuli rather than a continuous physiological signal as with the EEG. The only stimulus to which BIS responds is the surgical stimulus itself. The transition from awake to unconsciousness shows some overlap with BIS but not with the Alaris AEP. However, the literature for BIS is much greater than the Alaris AEP so this needs further confirmation. He commented that there were 15 years study of AEPs in Northwick park with virtually no other papers. Now there is a big increase in clinical research in anaesthesia with AEP and most work seem promising.

## **AFTERNOON SESSION: WINDOWS ON THE CIRCULATION**

**Chair:** *Dr D. W. Green, Honorary Secretary of the RSM Section of Anaesthesia*

### **Introductory remarks**

*Dr D. W. Green, Honorary Secretary of the RSM Section of Anaesthesia*

The Hon Sec opened the afternoon session by re-emphasising the relatively non-invasive nature of the monitors which have been discussed during these sessions. Clearly, some of these monitors would be accepted into mainstream anaesthesia and intensive care practice and others would not. He mentioned the case of the pulse oximeter which is now accepted as a *sine qua non* of monitoring during anaesthesia as much as the ECG or the automatic oscillometer. However, it was nearly 8 years following its introduction that it was routinely used at his hospital. This meant that about 100,000 patients had been anaesthetised without the benefit of oximetry. Thus, there are some monitors which get accepted but take too long to become routine whereas there are others such as the Finapres blood pressure monitor or conjunctival oxygen monitor which are now forgotten but seemed so impressive at the time of their introduction. He urged the audience to think about which of the monitors discussed would be successful and which would go the way of the Finapres. An interesting conundrum?

### **The PiCCO Less Invasive Advanced Haemodynamic Monitor**

*Dr Julia Wendon, Consultant Hepatologist and Intensivist, King's College Hospital, London*

Dr. Wendon began by saying that the goals of monitoring the cardiovascular system is to improve cardiac output, perfusion and outcome and to avoid detrimental effects. However, she emphasised the importance of one's own clinical experience in determining treatment. Thus experience counts and one must look at the overall patient. Any monitor must be easy to use and have a low complication rate.

Although pre-optimisation is a very topical subject at the moment, most of the work has been done by optimising vascular pressures. However, CVP or LVEDP (PCWP) does not address volume changes if cardiac compliance is abnormal. PiCCO measures cardiac index, contractility index, preload, blood volume index and extra vascular lung water index (EVLWI) which is a measure of lung leak. It is simple to use requiring a CVP and an arterial line in the femoral or axillary artery. Although it is applicable in children it cannot be used with the intra-aortic balloon pump, aortic aneurysm repair or pneumonectomy. It produces trends rather than absolute values.

Dr. Wendon then explained the physics of the system and emphasised good comparisons with the gold standard thermodilution.

Volumetric rather than pressure monitoring is thus possible with the PiCCO. In working out a methodology for different cohorts of patients different strategies are needed. For example with sepsis and large blood volumes the patient is filled until the stroke volume plateaus. Pressures alone do not indicate how the patients respond to volume whereas intrathoracic blood volume, obtainable with the PiCCO, is a much better correlation. In addition, EVLWI is a valuable added adjunct in patients with hypoxia. If it is normal then volume replacement will not be detrimental to oxygenation, whereas if it is high more volume is likely to worsen indices with an increase in EVLWI and an increase in mortality. However she emphasised that we should not stop using CVP and venous oxygen saturation.

From the anaesthetic point of view, PiCCO has been found useful in the operating theatre with laparoscopic surgery, liver transplantation and liver resection. Stroke volume variation (SVV) is a useful measure which gets less with adequate volume status. PiCCO allows continuous SVV to be monitored allowing graded fluid loading using 100ml 6% hydroxyethyl starch infusions over 2 minutes. A SVV greater than 9.5% predicted the response to a specific volume load, especially in cardiac patients where comparisons of SVV, CI and SV correlated well. It is also useful for determining or guiding overall fluid and vasoactive drug strategy.

### **Measurement of Cardiac Output by Lithium Dilution and Arterial Waveform Analysis**

*Dr Robert Linton, Consultant Anaesthetist, St Thomas' Hospital, London*

Dr. Linton is the co-inventor of the Lithium dilution cardiac output (LIDCO) monitor and together with his son in developing the algorithm for determining stroke volume (SV) from arterial wave form or pulse contour analysis. Determination of stroke volume in this way (as is also used with the PiCCO) produces statistical problems when trying to compare values obtained with those of the gold standard thermodilution (TD) method. Although correlation coefficients are often used they are usually used for sets of readings and are no good for determining whether an individual reading of SV with the LIDCO is consistent with that obtained with TD at the same time. Thus, CI obtained with the PiCCO may show excellent correlation with that obtained by TD. However, he then presented data which showed that if percentage change is measured in each a completely different result is obtained. This may be due to a failure of the PiCCO algorithm to take into account that the aortic waveform is reflected at the periphery and comes back again to give a secondary peak. This peak may be interpreted as increased SV. The position of the tip of the arterial catheter in the PiCCO is right at the point of maximal reflection especially in elderly patients. This may also occur during vasoactive treatment. For example, Rodig's study using phenylephrine and PiCCO appeared to show an increase in cardiac output following the drug whereas the true effect was a fall in cardiac output (Q). This misinterpretation was due to the PiCCO taking the increased reflected wave due to vasoconstriction as part of the stroke volume. Thus, the area under the curve (AUC) of the arterial waveform is no guide to Q.

He then explained the mechanism of the LIDCO and the fact that it is accurate, safe and easy to use. The calibration sequence uses miniature ion (Lithium) sensitive electrodes which are connected to a radial arterial line by a set of tubing and a pump. The pump is switched on which then draws arterial blood past the sensor and then inject 0.3 mmol of Lithium. The lithium dilution curve is then used to compute Q which is then used as calibration for the pulse contour wave analysis. He showed examples, using videotapes, of its use in human beings, horses and giraffes.

### **The Deltex Oesophageal Doppler Monitor (CardiacQ) - the Continuous, Non-invasive Cardiac Function and Volume Status Monitor**

*Dr Howard Wakeling, Consultant Anaesthetist, Worthing*

Christian Doppler in 1842 described the shift in frequency on which all Doppler techniques of measuring flow are based. The oesophageal Doppler probe (ODP) of the CardioQ is placed at the level of the descending thoracic aorta and measures real time blood flow. The set up and adjustment is easy and involves placing the probe in the oesophagus and adjusting the probe position by rotation or up and down to get the highest peak on the waveform. Autogain adjusts the gain to get the best possible picture with the whole process taking 45 seconds or so. To obtain flow from the velocity and thus stroke distance (SD) of the blood flow knowledge of the aortic cross sectional area (ACSA) is required. This is not measured directly but is estimated from the patient's height and weight. ACSA multiplied by SD gives volume. The CardioQ assumes a constant 30% of blood goes to the arms and the brain thus Q can thus be calculated from this volume.

One of the main variables which the CardioQ calculates is the corrected flow time (FTC). This is obtained from the arterial waveform and is a measure of fluid status of the patient. The width of the arterial waveform broadens

as volume is depleted and is corrected for changes in heart rate. A value of 340 to 440 for FTC suggests normovolaemia. Hypovolaemia is suggested by a low SV, FTC and Q. Administration of colloid increases all these factors. Clinical studies have involved assessment and correction of hypovolaemia, especially in the elderly. Correction of hypovolaemia has specific beneficial effects on reducing gastrointestinal dysfunction and reducing morbidity and length of stay in elderly patients following fractured neck of femur.

**'Vasotrac' A new concept in 'continuous' non-invasive blood pressure monitoring**

*Dr. Philippa Veale, Specialist Registrar in Anaesthetics & Clinical Research, Queen's Medical Centre, Nottingham*

Why do we need a new non-invasive BP monitor? Many patients need the placement of a continuous arterial catheter but there are disadvantages resulting from the placement of a catheter in an artery. The 'Vasotrac' provides a near continuous NIBP but without the need for an invasive catheter. It works by a patented process that pressurises a membrane that compresses the radial artery overlying the head of radius. This position is crucial because it needs underlying bone to compress the artery but it is not totally occlusive. The waveform is displayed on the monitor and a true value for systolic, diastolic and mean pressure is updated every 15 seconds. It works on adults and children down to 2 years of age. Shortly a probe for infants and neonates will be produced. The evidence that it works comes mainly from the multicentre study of Belani *et al.* Only 80 surgical patients were studied with 17,468 measurements. This study showed good agreement with invasive monitoring.

In her studies, Dr. Veale was concerned to see whether different operators obtained the same readings on the same patient. In a preliminary usability study, five operators assessed the reproducibility of the data. She found that 95% agreement is excellent and operator variability very low. In another study in children there was good agreement for systolic, diastolic, mean and heart rate. However, she found that systolic BP from the Vasotrac over-reads at low BP values and under-reads at higher BP values *cf.* intra-arterial BP monitoring. Diastolic BP is more satisfactory with the limits of agreement of 12–15 mm Hg.

Her conclusions were that the monitor seemed useful but her results were not as tight as Belani *et al.* in the series mentioned above. Their results need to be substantiated by other studies.

**Dr DW Green, MB, FRCA, MBA**  
*Hon. Secretary, Section of Anaesthesia*