Brian J Pollard

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Brian Pollard was a MRC research scholar at the Clinical Research Centre, Harrow, in Middlesex, from 1971-72, after qualifying BPharm; MB. ChB in 1977, FFARCS in 1981. After anaesthesia training in Nottingham, he became senior lecturer in Manchester in 1985[i]. He was appointed to a personal chair in 1996 and became head of department in 1997[ii].

His first paper, published in 1973, was as a MRC research scholar and was a study involving the rat phrenic nerve-diaphragm preparation, a commonly used animal model that was used in Nottingham in the same period. It was used to study the effects of inhalational anaesthetic agents. Diethyl ether, methoxyflurane and trichloroethylene caused depression; cyclopropane just potentiation and chloroform and halothane potentiation followed by depression. Halothane caused the least depression. Studying the preparation with tubocurarine, direct stimulation of the muscle and denervation of the muscle suggested that the effects of the inhalational agents were a non-junctional process [1].

Further work was done using this technique in 1983, *"Interactions between tubocurarine, pancuronium and alcuronium demonstrated in the rat phrenic nerve-hemidiaphragm preparation"* [8]. They found that mixtures with tubocurarine were synergistic; alcuronium mixed with pancuronium was not. This type of work was continued later in Manchester

Nottingham: 1973 – 1985

Brian and the author shared time in Nottingham and therefore the names of the co-authors are very familiar.

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[i] Studies Concerning the Interactions between the Neuromuscular Blocking Agents. MD Thesis, University of Sheffield, 1991

[ii] J F Nunn. British Journal of Anaesthesia. 1999; 83(6): 916

1976: This was a description of the efficacy of nasogastric feeding, with a group of physicians interested in metabolic processes, for patients in intensive care; they used a constant drip method [3].

A series of case reports followed. 1982/'83/'85:

1. A baby born with a paralysed hemidiaphragm and congenital hypothyroidism [5].
2. Reversal of biliary sphincter spasm with glucagon [7].
3. Fatal pulmonary embolism due to limb exsanguination [9].
4. Subtotal tracheal resection: a case report and review of airway, anaesthetic and post-operative problems [12]. An intrathoracic tumour required tracheal resection; the 'gap' was replaced with a musculocutaneous flap[iii].

Manchester 1987 -

Tom Healy, ex-reader Nottingham, was professor in Manchester and BJP followed in '87.

The major theme in BJP's work was neuromuscular blockade, particularly the combination of agents:

Neuromuscular Blockade

1987: "*The neuromuscular blocking effect of trimetaphan alone and in combination with different non-depolarizing muscle relaxants in the rat*". Trimetaphan produced potentiation of neuromuscular blockade when given with tubocurarine, metocurine and pancuronium but produced an all-or-none block when the concentration exceeded a threshold value [21]. Trimetaphan (Arfonad) was commonly used during anaesthesia to induce hypotension and thus knowing its effect on the neuromuscular junction was important.

1988: "*Differing interactions between hexamethonium and tubocurarine, pancuronium or alcuronium at the neuromuscular junction*". Hexamethonium in low concentrations opposed the neuromuscular blockade produced by tubocurarine, pancuronium and alcuronium. Higher doses produced a dose-

[iii] The author was the lead anaesthetist and needed a very competent trainee to assist; BJP was the one. In retrospect this procedure should probably have been done using cardiopulmonary bypass. The tracheal airway was maintained using a rigid bronchoscope and the patient oxygenated using a venturi injector. Anaesthesia was maintained with intravenous agents.

dependent block. The inhibition of cholinesterase in the tissue of the preparation only occurred at concentrations higher than that that resulted in antagonism and therefore could not explain the results. The possible mechanisms were discussed [30].

1988: "*Prolonged neuromuscular blockade with vecuronium in renal failure"* [32]. A case report: the title speaks for itself – full recovery took 90 hours.

1988: "*Interactions of vecuronium and atracurium in an in vitro nerve-muscle preparation*" [33]. In this study atracurium and vecuronium were combined and the combination's log dose response curves indicated an increased potency. This synergy was considered consistent with multiple receptor site and different modes of action hypotheses.

1989: "*Concentrations of atracurium and laudanosine in cerebrospinal fluid and plasma during intracranial surgery*" [34]. Atracurium was given by infusion for up to four hours during neurosurgery. Atracurium and laudanosine concentrations were measured in the cerebrospinal fluid and plasma. It was apparent laudanosine accumulated in both plasma and CSF; laudanosine is known to predispose to convulsions.

1989: *"The effect of acutely administered phenytoin on vecuronium-induced neuromuscular blockade"* [35]. Phenytoin administered intravenously during steady state neuromuscular blockade caused a significant increase in the neuromuscular blockade; possible mechanisms are discussed.

1989: "*Use of atracurium or vecuronium to prolong the action of tubocurarine"* [37]. When atracurium or vecuronium was given after some recovery from tubocurarine, the first dose always had a more intense effect and lasted longer than subsequent equal doses.

1989: "*Priming with alcuronium and tubocurarine accelerates the onset of neuromuscular block"* [38]. Priming[iv] accelerated the onset of neuromuscular block.

1989: "*Should vecuronium be used in renal failure?"* [40] A letter.

1989: "*Use of continuous prolonged administration of atracurium in the ITU to a patient with myasthenia gravis*" [41]. A case report - muscular relaxation was with atracurium 5 mg h^{-1} and on cessation power recovered rapidly.

[iv] Priming is where a sub paralyzing dose of the drug is given prior to the main dose.

1989: "*Doxapram and the neuromuscular junction*" [43]. This was another rat phrenic nerve-diaphragm study. Doxapram enhanced neuromuscular transmission. Cholinesterase inhibition was excluded as an underlying mechanism. However, dose-related depression occurred if there was a partial neuromuscular block. Using agents with known presynaptic activity it was suggested that doxapram had a presynaptic facilitatory effect and in the presence of partial neuromuscular block an inhibitory action, post-junctional. It was pointed out that these effects were at concentrations much greater than those used in clinical practice.

1990: "*Concentrations of atracurium and laudanosine in cerebrospinal fluid and plasma in three intensive care patients*" [51]; a follow-up paper to a similar report in 1989. There were no adverse effects attributable to laudanosine.

1992: "*Atracurium block prolonged by low dose tubocurarine*" [68]. This was a detailed letter about the potentiation of atracurium by tubocurarine and could be used to reduce the dose of atracurium required and possibly avoid the use of anticholinesterases at the end of surgery. Presynaptic inhibition may be the cause; it would enhance agents acting on post-synaptic receptors.

1992: "*Effect of doxapram on neostigmine evoked antagonism of vecuronium neuromuscular block*" [70]. Recovery was prolonged in the presence of doxapram, but it was not statistically significant.

1992: "*Neuromuscular blocking drugs and renal failure*" Editorial [71].

1993: "*Neuromuscular blocking drugs in the intensive care unit: introductory remarks"* [77] and "*Neuromuscular blocking agents in intensive care*" [81] and were leading articles in an 'Intensive Care Medicine' supplement, followed later by "*Which drug - steroid or benzylisoquinolinium?*" [80].

1993: "*The onset of alcuronium and tubocurarine: alone and in combination*" [85]. The rate of block onset with the 50% combination was faster than the agents given alone. However, the small effect was considered unlikely to be of clinical use.

1993: "*Extending a pipecuronium neuromuscular block. Increments of atracurium or vecuronium as an alternative to pipecuronium*" [86]. A dose response relationship was constructed for pipecuronium. Further patients then received a dose to produce a >90% block. Small repeated doses of atracurium, vecuronium or pipecuronium were then given on recovery to 90% block. The

block with pipecuronium was constant but the duration of the blocks with atracurium or vecuronium became less with subsequent increments.

1995: *"Molecular mechanisms of neuromuscular blocking agents: is the increased understanding of importance to the practising anaesthetist?"* [91] This was a review article which explained that the precise mode of action of muscle relaxants remained unclear. It reviewed the ways in which other drugs used during anaesthesia "*may modify this system*".

1995: "*Prediction of infusion rates of vecuronium using the bolus test dose technique*" [94][v]. Neurosurgical patients were given a loading dose of vecuronium (0.1 mg kg^{-1}). On recovery of the first twitch in response to the train-of-four electrical stimulus various boluses were given. The duration of 2mg and 4mg boluses were capable of predicting the required infusion rate. They suggested the initial dose could also be used for prediction.

1995: "*The role of muscle relaxants in total intravenous anaesthesia*" [95]. This describes the use of muscle relaxants during total intravenous anaesthesia and does emphasise the need to monitor neuromuscular function.

1995: "*Intubation conditions and time-course of action of low-dose rocuronium bromide in day-case dental surgery*" [97]. Rocuronium, relatively new, was compared with atracurium and vecuronium. They used relatively small doses and so full relaxation was unlikely at 60s. The rocuronium group had the better conditions for intubation and the duration was of the order of 22 minutes.

1995: *"Pulmonary function and head lift during spontaneous recovery from pipecuronium neuromuscular block"* [93].

1996: "*The infusion requirements and recovery characteristics of cisatracurium or atracurium in intensive care patients*" [106]. Cisatracurium is an isomer of atracurium and makes up a small proportion of the atracurium formulation.

[v] The author is somewhat biased about this paper as it reproduces some work he had published in the Br J Anaes 1990;64:287-293. He is not sure about the statement that a prediction can be made using the first dose as the first dose is in mg kg^{-1}. It was shown in 1989 that weight determined dosage of vecuronium bromide can result in different durations depending on whether the patient is fat or slim. Anaesthesia 1989;44:692.

Both drugs were administered as an infusion for a minimum of 24h, the mean infusion rate of cisatracurium was a third of the atracurium rate. The recovery time was the same and there were no side effects and so it was considered a satisfactory drug for this use. The following report in 1997 is similar.

1997: "*A comparison of cisatracurium (51W89) and atracurium by infusion in critically ill patients*" [109].

1997: "*Rocuronium and cisatracurium*" [111]. This is an overview of rocuronium and cisatracurium, the most recent additions to the anaesthetic armamentarium.

1997: "*Mivacurium or vecuronium for muscular relaxation in day-case surgery*" [112]. Day case surgery is for relatively short procedures thus the muscle relaxants used need to be of short duration and recovery. The other agents used were propofol, fentanyl, nitrous oxide and isoflurane. The maximum block and ease of intubation was similar with the two test drugs but recovery was much faster in the mivacurium group; all the patients given vecuronium were given neostigmine. It was considered that mivacurium was possibly the better agent for day-case surgery.

1999: "*Antagonism of rapacuronium using edrophonium or neostigmine: pharmacodynamics and pharmacokinetics*" [117]. Half of the patients were given 1.5 mg kg^{-1} rapacuronium and the others 1.5 mg kg^{-1} rapacuronium plus three further doses of 0.5 mg kg^{-1}, all were given neostigmine or edrophonium. The results suggested that a three-compartment pharmacokinetic model was justified. Clearance was 4.4 ml kg^{-1} min^{-1}, initial volume of distribution 94.8 ml kg^{-1}. Clearance in females was 38.5% less and V1 was 25% less for those over 65 years of age.

The result of all this work demonstrated the effects of drugs used in anaesthesia on the pharmacodynamics of muscle relaxants, on the interactions between muscle relaxants, confirming the ability to predict infusion rates in individual patients and, hopefully, enhancing the likelihood of improving the frequency with which neuromuscular monitoring is used.

Heart rate variability (HRV)

HRV is the variation in the interval between heartbeats. The variations are largely due to vagal modulation; the parasympathetic tone normally exceeds the sympathetic effects. The vagal activity is the major contributor to the high frequency (HF) component; the low frequency (LF) component includes both sympathetic and vagal influences.

Previous work in the Manchester Department had revolved around the assessment of sinus arrhythmia as a depth of anaesthesia index[vi], CJ Pomfrett being a common co-author.

1998: "*The influence of premedication on heart rate variability"* [114]. This is an interesting collaboration with anaesthetists and cardiologists from the University Hospital, Iraklion, Crete. They used HRV to study the autonomic effects of midazolam, morphine and clonidine as premedicants. The patients were studied 60 mins before and after premedication using a Holter device. The low-frequency and high-frequency components were calculated. A ratio of <1 signified parasympathetic dominance. Power decreased with premedication whereas it did not with the placebo. Morphine and clonidine caused the low- to high-frequency ratio to decrease suggesting parasympathetic dominance.

2004: "*Perturbation of heart rate variability in cattle fed BSE-infected material*" [133]. The highest concentration prions that cause BSE are found in the brainstem, particularly nuclei in the medulla oblongata which are involved in the modulation of HRV. The level of high-frequency HRV was significantly different between control cattle and those exposed to BSE.

Another interesting collaboration.

2007: "*The vagus nerve as a conduit for neuroinvasion, a diagnostic tool, and a therapeutic pathway for transmissible spongiform enceph- alopathies, including variant Creutzfeld Jacob disease"* [139]. This is obviously a follow up to the 2004 paper and it is a little unusual as it is published in the journal called Medical Hypotheses. To quote from their website – **"***Medical Hypotheses* is a forum for

[vi] Pomfrett CJ, Barrie JR, Healy TEJ Br J Anaesth. 1993;71(2):212-7

ideas in medicine and related biomedical sciences. It will publish interesting and important theoretical papers that foster the diversity and debate upon which the scientific process thrives."

It was hypothesised that the vagus nerve is an important conduit for infective neuroinvasion during the incubation of certain transmissible spongiform encephalopathies. They proposed that HRV analysis of vagal function may indicate early functional signs of infection and may be used to measures the effect of new therapies.

2001: "*Delta sleep-inducing peptide*" [121]. DSIP is a delta sleep-inducing peptide which can cross the blood-brain barrier. Its effect is similar to natural sleep.

2009: "*Delta sleep-inducing peptide alters bispectral index, the electroencephalogram and heart rate variability when used as an adjunct to isoflurane anaesthesia"* [144]. It was thought that it might be useful as an anaesthetic supplement and so was studied using BIS, the EEG and HRV. Half of the subjects were given a placebo and the other half DSIP – all had a standard anaesthetic. The results were counterintuitive. The active group developed a tachycardia, increased BIS, reduced delta activity on the EEG, and decreased HRV (reduced parasympathetic tone). It lightened rather than deepened anaesthesia. It just reinforces the need for physical experimentation rather than thought experiments.

Day case surgery

There were four publications that audited and discussed anaesthesia for day case surgery.

2002 "*Anaesthetic agents in paediatric day case surgery: do they affect outcome?"* [124] The aim of day case surgery is to minimise post-operative morbidity, most commonly postoperative delirium, nausea and/or vomiting. The induction of anaesthesia, cardiovascular effects, recovery and postoperative nausea and vomiting were all addressed. The use of sevoflurane, halothane, propofol, desflurane and nitrous oxide, and combinations of them were all assessed.

2002/3 *"Which anaesthetic agents are cost-effective in day surgery? Literature review, national survey of practice and randomised controlled trial"* and *"Clinical and economic choices in anaesthesia for day surgery: a prospective randomised controlled trial"* [123, 126]

2003 "*Anaesthesia for day case surgery: a survey of adult clinical practice in the UK"* [127] In 2000 a national postal survey of the range and variation of anaesthetizing patients for day case surgery was carried out, particularly urology and orthopaedic day cases. They used a structured postal questionnaire. Premedication was used by between 6 and 12% of practitioners, propofol was the preferred induction agent and isoflurane the preferred maintenance agent. Prophylactic antiemetics were only used in between 32 and 41% of patients and a laryngeal mask was used by between 86 and 93%.

2003 "*Anaesthetic agents in adult day case surgery".* [128] This was a meta analysis of "*all comparative published studies of adult day case anaesthesia in the English language up to December 2000*". One hundred-and-one studies were analysed. Propofol was found to be equal to sevoflurane and desflurane and better than methohexital, etomidate and thiopental. Isoflurane and halothane were considered the worst for maintenance. Propofol was the induction and maintenance agent of choice and avoiding nitrous oxide might help reduce the incidence of postoperative nausea and vomiting.

2008 "*The effect of anaesthetic agents on induction, recovery and patient preferences in adult day case surgery: a 7-day follow-up randomized controlled trial."* [141] This was a study of the longer term effects of day case anaesthesia. It was a randomized controlled trial of four standardized techniques. "... *propofol induction and maintenance, propofol induction with isoflurane/N_2O, or sevoflurane/N_2O maintenance, or sevoflurane/N_2O alone*". The bottom line was that the differences in outcome were transient and patients base their preferences on the method of induction; it is, after all, that which they are most aware of.

Cardiac output

1987: "*The effects of calcium-blocking agents on sympathetic responses to acute haemorrhagic shock in dogs"* [19]. Verapamil, nifedipine and diltiazem were studied to determine their effects on the cardiovascular in response to

haemorrhagic shock; this study was in dogs. Cardiac output, mean pulmonary artery pressure, mean right atrial pressure and pulmonary capillary wedge pressure all fell; adrenaline and noradrenaline activity increased; and the calcium antagonists did not make a difference. However patients receiving the higher doses of verapamil or nifedipine had a greater rise in renin levels.

1991: "*Bioimpedance versus thermodilution cardiac output measurement: the Bomed NCCOM3 after coronary bypass surgery"* [62].
The gold standard for cardiac output measurement, thermodilution, was compared with measurements using bioimpedance (Bomed NCCOM3) after coronary artery bypass surgery. Cardiac output measured by bioimpedance was significantly lower than with thermodilution during their time in intensive care. The limits of agreement were also large. Later measurements, with all patients breathing spontaneously, there was no difference.

1991: "*Measurement of transthoracic electrical impedance"* [65] This letter highlights some of the problems with bioimpedance measurement. It is affected by both intra- and extravascular fluid and the packed cell volume. As they stated, it "*may explain why a technique that was well described more than 20 years ago has yet to find its way into routine clinical use.*"

1991: "*Non-invasive measurement of cardiac output during induction of anaesthesia and tracheal intubation: thiopentone and propofol compared."* [66]. In this paper they investigated haemodynamic changes during standard anaesthesia with the bioimpedance monitor. The cardiac index decreased after induction and decreased further after tracheal intubation. There was no difference between the two groups. Mean arterial pressure and systemic vascular resistance were stable after thiopentone but both increased after tracheal intubation. Propofol caused a decrease in both mean arterial pressure and systemic vascular resistance.

Cerebral Function

BIS asymmetry [125, 130, 135]

It has been shown that there is an asymmetry in BIS recording between the left and right brain in children. It has been thought that it was immaterial which side of the head was used for measurement. These three studies investigated the pnenomenon.

Functional electrical impedance tomography by evoked response (fEITER) [146, 148-150]
These describe the use of a portable new non-invasive device for brain imaging. The aim was to study "sub-second mechanisms underlying consciousness". A sinusoidal current was passed between electrode pairs; non-current electrodes were used for voltage measurements. The sub-second responses to single flashes enabled construction of 3D maps of conductivity. Large changes in trans-cerebral impedance occurred during propofol induction[vii].

Case reports

1982: Paralysis of the right hemidiaphragm. [5]
1983: Reversal of biliary sphincter spasm with low dose glucagon during operative cholangiography. [7]
1983: Fatal pulmonary embolism secondary to limb exsanguination.[9]
1985: Subtotal tracheal resection: a case report and review of airway, anaesthetic and post-operative problems. [12]
1988: Prolonged neuromuscular blockade with vecuronium in renal failure.[32]
1989: Use of continuous prolonged administration of atracurium in the ITU to a patient with myasthenia gravis.[41]
1989: Anaesthesia in the testicular feminisation syndrome.[39]
1989: The use of flumazenil (Anexate, Ro 15-1788) in the management of drug overdose.[42]
1989: Anaesthesia in myotonia dystrophica.[44]
1989: Renal transplantation and diabetic autonomic neuropathy. [45]
1989: Obstructive sleep apnoea.[47]
1989: Failure to cannulate the epidural space.[49]

1989 was obviously the year for the reporting of clinical problems. Brian Pollard was involved at this time with the writing of the manuscript for "*Anaesthesia for Uncommon Diseases*" with the author. In fact 1989 was a very busy year – there were at least fourteen publications.

[vii] See http://www.wellcome.ac.uk/News/2011/News/WTVM051674.htm

He was the Editor-in-Chief of *Current Anaesthesia and Critical Care* (1989 to 2003–13 editorials), was involved with the *European Journal of Anaesthesiology* from 1997-2009 (Editor-in Chief 2004 to 2009) and Editor-in-Chief, "*Trends in Anaesthesia and Critical Care*" 2010 to 2014 (10 editorials). There were many more invited articles for a variety of non-indexed publications.

He was president of the Section of Anaesthesia of the Royal Society of Medicine, Council member of the Association of Anaesthetists and an Executive Committee member of the Anaesthetic Research Society. He was also a Senate Member of the European Academy of Anaesthesiology.

Books:

Anaesthetic Management: A Rule-Based Guide. 1986 BJ Pollard, MJ Harrison and R. M. Jones. Butterworths (1986).

Anaesthesia for Uncommon Diseases. 1989 BJ Pollard, MJ Harrison. Blackwell Scientific Publications, (1989)

"Aids to Anaesthesia" Second edition.T.E.J.Healy and B.J.Pollard Churchill Livingstone, 1999

Edited books

Applied Neuromuscular Pharmacology (1994), The Muscle Relaxant Handbook, (1995) and Handbook of Clinical Anaesthesia, three editions 1996 – 2011 and twenty-five chapters in various books,

Odds and ends:

Nasal CPAP: [47, 63, 75, 76], Assisted conception: [58, 113, 115] and the QT interval: [104, 105]

And ... to finish:

"Beards, academia and anaesthesia: a controlled study" [103]. This was published in the pre-Christmas edition of the BMJ, usually a jocular publication. It's worth a read!

"*Anaesthesia acronyms and abbreviations*" [132]

"The impact of increasing oximetry usage in India: A pilot study" [147]

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