J. P. Payne
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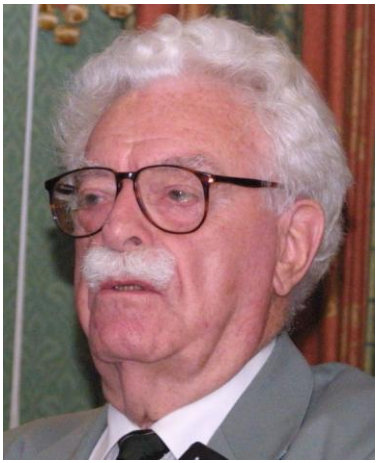
 [i]

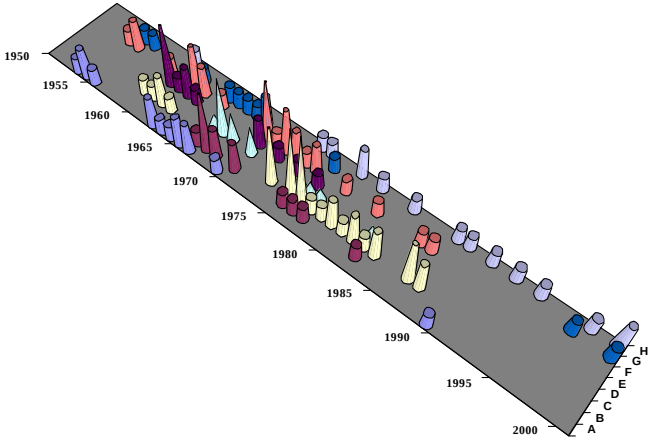
JP Payne, over a period of more than forty years, wrote on a variety of topics relating to anaesthesia; these fall into several main categories, hypoxia, the measurement of alcohol in blood and breath, the effect of drugs on the neuromuscular junction, physiological measurement and various papers on ethics and education.

Payne was part of a team and the following names should be recognised as eminent team members: JA Bushman, CM Conway, DW Hill, R Hughes and N Sugai. Their references [1-76] are discussed and references [77-150] refer to his papers not discussed in the text.

Hypoxia

Payne published a paper , in 1953 in the British Journal of Anaesthesia, based on a presentation to the Scottish Society of Anaesthetists titled 'Controlled hypotension in theory and practice'[1]. He was then working in the Department of Anaesthesia at the University of Manchester. This is an interesting article comparing the clinical use of 'bleeding' for hypovolaemic hypotension with the use of ganglion blocking agents. It included case reports of bleeding patients up to two litres for the facilitation of neurosurgery. He commented on the ease with which anaesthesia was

[i] Photograph courtesy of Alistair McKenzie and the History of Anaesthesia Society



A	B	C	D	E	F	G	H
Oxygenation	Alcohol	NMB	Technology	Measurement	Pharmacology	Clinical	Ethics etc.

Publications from Payne's team from 1950 – 2000

maintained at low blood pressures and postulated that at <50mmHg (?systolic) patients might remain asleep without anaesthesia. There followed a discussion on consciousness, in terms of philosophy, physiology and pharmacology; tissue metabolism was considered of importance in maintaining consciousness.

Two papers in 1954 investigate oxygen consumption in cats[2 3]. The second one showed that the reduction in VO_2 due to bleeding was greater than due to sympathetic blockade, possibly due to the vasoconstriction, and suggested that the fall in VO_2 during sympathetic blockade was secondary to the absence of the "normal complement of adrenaline".

In a joint investigation with Nunn in 1962 [4] Payne reported postoperative hypoxaemia; oxyhaemoglobin saturations of 90% (PO_2 of 65mmHg). This was a landmark paper. Both Nunn and Payne were lecturers at the Postgraduate Medical School, Hammersmith. They described how it stayed low for

24 hours, and the lowest PO_2 recorded was 39 mmHg, the highest 82mmHg. They suggested that this was a strong case for the use of oxygen masks in the postoperative period. Another paper published in 1963 supports this work[5]. Oxyhaemoglobin desaturation was found in every patient and was relieved by oxygen enriched air. It was not related to a particular anaesthetic agent or surgery. The PCO_2 values were normal so it was not thought to be due to under-ventilation. Further work on hypoxaemia in the perioperative period was published in late 1964[6]. Hypoxia during surgery, as well as in the postoperative period was a surprise finding, and some patients were found to be hypoxaemic before surgery[7]. Atropine was thought to be the causative agent[8]. Intramuscular atropine was shown to reduce oxyhaemoglobin saturation compared with control patients. This is contrary to a later paper where it was shown that only atropine administered subcutaneously seemed to cause a fall in PaO_2[9], it was not seen with the intravenous or intramuscular routes. The mode of action was thought to be mechanical, secretions or a change in surfactant. The magnitude of the fall was in PaO_2 was related to age. $PaO_2 = 102.5 – 0.22$ (age)

Over these early years, mid fifties and sixties, JPP seemed to have affection for chloroform; he was chloroform's last champion [10]. Several papers seemed to be fighting a rearguard action for its continued use; its arrythmogenicity being a complication of too light anaesthesia.

Alcohol[ii]

Payne, Hill and King (Research Department of Anaesthesia, Royal College of Surgeons, England) in 1966 produced a report on the distribution of alcohol in blood, breath and urine[11]. The work was carried out in anaesthetised dogs and volunteer humans (a choice of whisky, gin or rum). Great attention was paid to the validation of the measurement techniques and the following conclusions were

[ii] The breathalyser was introduced in Britain on October 9th 1967, it is partly responsible for reducing alcohol related deaths from 13000 pre-1967 to 2500 in 2006.

reached. Absorption of alcohol from the stomach was slower than from the duodenum and the rate of absorption varied widely, the uptake being related to the dose and the rate of drinking.

Carotid jugular equilibrium occurred quickly but the measuring of venous blood alcohol was only of value after the peak arterial concentration had been reached. The uptake of alcohol by the tissues would lead to an underestimate of arterial alcohol before the peak.

Analysis of alcohol in the breath tended to underestimate arterial levels, possibly due to V/Q abnormalities, or possibly due to uptake of alcohol by mucus. If alcohol was in the mouth an artificially high level was recorded.

A good correlation between blood and urine only held if the peak concentration in the urine had passed and the bladder had been emptied 20 – 30 minutes earlier. The advice given was that two samples should be collected at thirty minute intervals and if the concentration in the first was greater than the second then accuracy was assured.

In 1967 the accuracy of gas chromatography method for alcohol was assessed and confirmed the work by Curry et al.[iii] that gas chromatography combined with an internal standard and an integrator could determine alcohol content accurately in five minutes. An investigation was carried out into the conversion factor of 1.33 for blood levels from urine levels[12], similar work was published in France, Spain and Germany[13-15]. Stevens et al.[iv] however showed the methodology to be invalid in the "less rigidly controlled environment of a busy police station". The range of blood: urine ratios were such that miscarriage of justice was possible. The value of alcohol in capillary samples of plasma and red cells was also discussed. The plasma's alcohol concentration was not the same as blood alcohol concentration as the red cells contain a lower concentration[16]. The blood concentration would depend on the haematocrit. It was suggested that venous

[iii] Curry AS, Walker GW, Simpson GS. Determination of ethanol in blood by gas chromatography. *Analyst* 1966;91(88):742-3.

[iv] Stevens PJ, Mason JK, Bowden CH. Comparative ethanol concentrations in blood and urine during social drinking.. Medicine, Science and the Law 1966;*6(2):96-102*

blood should still be used. The mean urine: blood ratio was 1.44:1 with a range of 1.10 to 2.44.

The Ministry of Transport Road safety Act, 1967, stated that 107 mg of alcohol in 100ml urine should be treated as equivalent to 80 mg of alcohol in 100ml blood.

Further work, in 1968, on the plasma vs. blood alcohol problem confirmed the work of others and reinforced the view that plasma/breath ratios, or plasma/urine ratios should be used as these were independent of haematocrit. Numerous allied papers were published on the topic over the years and much debate about breath analysis[v].

In 1976 another measurement technique[17] proved equally problematic with wide variation in results and a clash with Wright (Clinical Research Centre, Harrow, of 'Wright's Respirometer' fame) where he said "not only are their results much worse than those obtained in recent years by a number of reputable observers, but they give a picture of the present understanding of the subject that is very out of date". [vi]

An interesting final foray into the effects of alcohol was an investigation of alcohol related flushing of the face and neck, a possible genetic trait, that occurs in situations where the alcohol in the blood exceeds 20-35 mg/dL[18]. Alcohol and aldehyde dehydrogenase enzyme deficiency cause the blood acetaldehyde to become abnormally high. Absorption of alcohol could be retarded by a combination of H1 and H2 antagonists and thus reducing the peak concentration.

Neuromuscular blockade

'Jimmy' Payne worked on the pharmacology of neuromuscular blockade (NMB) from 1958 through to 1987. He demonstrated in 1958 that d-tubocurare (dtc) was

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[v] B M Wright. Breath, alcohol, and the law. *Br Med J.* 1977 July 9; 2(6079): 121
[vi] B M Wright Breath, alcohol, and the law. Br Med J. 1977 May 7; 1(6070): 1216–1217.

different to the other relaxants available at the time in being enhanced by carbon dioxide, K^+ had no effect [19]. Dtc was different from suxamethonium, decamethonium and gallamine by having a pKa value 8.1 – 9.1, as compared to 13. This phenomenon was examined further [20-22]; was it due to a change in the ionic state? Probably not; was it a specific effect on dtc – a chemical union where the hydroxyl groups were neutralised and potency increased?

In 1959 he described suxamethonium tachyphylaxis [23], a progressively diminishing effect of successive doses could then be explained by the residual competition block antagonising the depolarising effect of succeeding doses of either decamethonium or suxamethonium. He hypothesised that all NMB agents may pass through a depolarising phase which is later followed by a competitive block. In 1962 the duration of a variety of agents was tested in the presence of the serum from jaundiced patients [24], the decrease in effect was considered to be due to increased protein binding and the increased effects of the tropeine derivative by the low serum cholinesterase.

A ten year gap separates this work from his later publications; his co-workers were N.Sugai until 1976 and R.Hughes until 1986. Hughes was with Burroughs Wellcome and oversaw the introduction of atracurium.

At the Anaesthetic Research Society (ARS) in Liverpool (April 14th 1973) 'Jimmy' Payne presented one of his most obsessive subjects, that of the value of tetanic contractions vs. single twitches in the assessment of NMB[25 26]. Tetanic contraction was considered a more sensitive measurement of neuromuscular function by Gissen and Katz (1969)[vii]. It was shown that the tetanic response disappeared more quickly than single twitches. At the ARS at the Hammersmith (London), November 1973, Payne's team reiterated the importance of using both single twitch and tetanic stimulation for the study of suxamethonium [26]. It was suggested by Sugai and Payne[27 28] that the response to tetanic and single twitch stimulation represented different aspects of response of the neuromuscular

[vii] Gissen AJ, Katz RL. Twitch, tetanus and posttetanic potentiation as indices of nerve-muscle block in man. *Anesthesiology* 1969;30(5):481-7.

junction, that there may be enough acetylcholine (ACh) for repeated single twitches but not for sustained tetanic contractions.

Payne used the tetanic tension ratio (TTR) [29], which was the height at the end of the stimulation period divided by the height at the beginning, and single twitch technique to elucidate neuromuscular function in parallel with combinations of nondepolarising agents, depolarising agents and anticholinesterases. Using combinations of suxamethonium and edrophonium[30] he came to the conclusion that that tachyphylaxis and change in character of the NMB were parts of the same phenomenon. Amongst the drugs studied were suxamethonium, decamethonium [31], dimethyltubocurarine [32 33], tubocurarine, and gallamine [34]. One pharmacological goal was the 'clean' muscle relaxant without cardiovascular effects – dimethyltubocurarine had some of the required properties.

The interaction between halothane and NMB was also studied[35]. It was thought that the then effect was a pre-synaptic one because the enhanced block was only evident when tetanic stimuli were applied, this could be caused by impairment of Ach release.

A significant paper was published in the B.J.A. in 1980. This was a study of the NMB properties of neostigmine[36]. It was shown that repeated doses of neostigmine caused an increase in NMB as documented by the TTR and the severe fade lasted twenty minutes. This blockade was potentiated by suxamethonium and antagonised by gallamine. The big difference between the effects of the frequency of stimulation was clear. Tetany in the presence of inhibition of acetylcholinesterase and an increase in Ach caused prolonged end-plate potentials (EPPs) to summate and cause persistent depolarisation of the post synaptic membrane and the mobilised Ach reserves became exhausted leading to fade.

The single twitch method of neuromuscular assessment showed an increase in twitch height with neostigmine, this was said to be due to the increased amounts of ACh available and augmented and prolonged the EPPs but in insufficient quantities to produce NMB. Payne advocated the use of tetanic stimulation for assessment of NMB because it was more physiological. This work supported the

early work done by Briscoe in 1936/7.[viii] It would appear that the 'neostigmine resistant block' was in fact a neostigmine induced NMB.

There were many papers investigating the technological aspects of electromyography (emg)[29 37-40]. As new neuromuscular blocking agents came available for study Payne's department was at the ready; fazadinium was assessed in 1976[41]. Fazadinium did not stay in clinical use very long; it was suggested that with repeat doses it might produce a profound NMB. Atracurium was evaluated between 1981 and 1986[42-47], and vecuronium in 1987[48]. Much work was done on atracurium with R Hughes, atracurium being a Wellcome product and worked in the Pharmacology Department of the Wellcome Research Laboratories in Beckenham in Kent. The work with atracurium covered most aspects of its anaesthetic usage. The mathematical mechanism for producing a rate constant for recovery was described[45].

Technology

Payne summarised anaesthetic monitoring requirements in a non-specialist journal in 1970[49], the differing requirements between intra-operative and postoperative requirements being highlighted. ECG monitoring was not routine at this time but it was advocated, citing the work of Johnstone in 1948 on the effects of anaesthetic agents on abnormal heartsix. Real-time computing in patient monitoring 1971, 1972[50 51] was now a real possibility.

Payne's team had an obvious interest in measurement and technology. Payne's team was in at the beginning of the use of the telephone system to transmit data, papers in 1968, 1970, 1972, 1976[52-55] all indicate the potential use for real-time

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[viii] Briscoe, G. (1936). Shift in optimum rate of stimulation due to prostigmine. J. Physiol. (Lond.),88,48P.
----------(1937). Optimum stimulation rates for red and white skeletal mammalian muscles, and shift in rates produced by the eserine group. J. Physiol. (Lond.), 90, 10P.

[ix] Johnstone MW. (1948) MD Thesis. Queens University, Belfast.

analysis away from the clinical environment; three waveforms could be sent at one time. In 1964 Levine and Jossman had published on the same topic. [x]

EEG analysis during anaesthesia was carried out in 1976[56] using a telephone link to an Elliott 903 computer, they did a fast Fourier analysis and then returned the processed information back to the anaesthetic room. This work was part of Perry's PhD thesis and followed on work by Kiersey et al.[xi]

Mass spectrometry, another technical innovation in anaesthesia research, was used in 1976[57] to measure the loss of anaesthetic vapours from breathing circuits during and after anaesthesia. This was an early investigation of the pollution of operating theatre air and hence the requirement for scavenging.

Ethics and Miscellany

Payne had an interest in humanitarian aspects of anaesthesia as well as the scientific, and often they crossed paths.

History: Professor Payne was a great supporter of chloroform, in 1955 he published an article in Medicine Illustrated [10], twenty-six years later he wrote about chloroform again – 1981[58], and in 1998[59]. It was said to be good, "Admittedly, at that time…" the newer agents weren't available "… but it would be surprising if any of these later drugs were shown to be vastly superior to chloroform." There was said to be a great demand worldwide for a safe, cheap, potent, non-explosive and easily stored agent.

He wrote on anaesthetic deaths in 1983[60], stating that anaesthesia cannot be blamed for all surgical deaths and complaining that surgeons are unwilling to study the problem. He emphasised the need for audit to be accepted – (Medicine Digest 1982. 8 p35) and dental anaesthesia in 2000 [61].

He wrote on the resuscitation of the apparently dead[62], the quality (the degree of excellence) of measurement 1970[63] and on the ethics of the use of

[x] Levine IM. Jossmann PB. Tursky B. Meister M. Deangelis V. Telephone telemetry of bioelectric information. *JAMA. 188:794-8, 1964 Jun 1.*

[xi] Kiersey DK. Faulconer A Jr. Bickford RG. Automatic electroencephalographic control of thiopental anesthesia. Anesthesiology. 15(4):356-64, 1954 Jul.

halothane 1974,1986 [64 65]. The 'Quality of Measurement' article was a report on his presentation of the Clover lecture on 18th March 1970 at the Royal College of Surgeons and was a progress report of his seventeen years as Head of Department.

Ethics and Halothane:

Prof. Payne wrote several editorials and they always had a lively, brusque style. The Committee of Safety of Medicines (CSM) made comments about the safety of halothane without referral to clinical anaesthetists and this caused great concern as it was considered to be a clinical decision as alternatives to halothane may have their own risks which could be greater. Quoting Professor Parkhouse...is it the CSM's place to "enter the arena of clinical judgement?"[xii]

In 1975 he wrote on responsibility and accountability [66]. He took the Committee on Safety of Medicines to task on their controversial letter about halothane. He compared the responsibility of individuals for their actions with the apparent freedom of responsibility that committees enjoy, committee members passing the buck to the chair, the chair reciprocating in a similar manner.

In 1978 animal experimentation was the focus [67] and was stimulated by Smyth's monograph Alternatives to Animal Experiments[xiii]. He was commenting on the activists' against animal experimentation need for replacement techniques, which is laudable, but is quite different to whether it is possible. Public concern over the Thalidomide tragedy had stimulated a need for legislation requiring more testing; so the public's view on animal experimentation was in fact not unanimous.

In the same year another editorial tackled the problem of research in intensive care units[68]. He castigated medical staff for avoiding issues and praised a nurse for a paper on the topic in the same journal[xiv]. He took the Royal college of

[xii] Parkhouse J. Letter: Halothane and liver damage. British Medical Journal. 3(5934):807, 1974 Sep 28.
[xiii] Smyth, D. H. (1978). Alternatives to Animal Experiments. London: The Scolar Press Ltd in Association with the Research Defence Society.
[xiv] Bishop VA. A nurse's view of ethical problems in intensive care and clinical research

Nursing to task for providing nurses with "Girl Guide" type guidelines on ethical issues. He advocated greater communication between clinicians and patients and/or patients' relatives, "Patient's diseases are not the personal property of the clinician".

The place of arterial cannulation in research had been hotly debated because there was great fear of real damage to the radial artery (large rigid cannulae being used) and in 1990 the ethics of arterial cannulation in research, in volunteers, was discussed[69]. An audit of the sequelae of arterial cannulation was carried out and the bottom-line was that the risk was low, that small bore cannulae were just as good as large ones and that only those experienced in the art of arterial cannulation should do them on volunteers.

Other topics were medical education 1960[70] 1980[71], the place of alternative therapies 1987 [72] and 1992, pay parity for academics, 1992[73]. There had been an agreement on pay parity but this was all offset by deterioration of work conditions and a decline in academic morale – an all time low - and that the outlook was bleak.

Thirty years on:

The anniversary of the founding of the Anaesthetic Research Society, 1988[74]; in 1952 the Faculty of Anaesthesia (Royal College of Surgeons) asked for the setting up of a research department, this happened in 1957. A research meeting was proposed but not universally condoned (it might set up an elite group of anaesthetists) but John Gillies[xv] said "Get it going and they will all be there." It was an opportunity for those engaged in anaesthesia research to discuss their objectives openly and frankly with similar minded colleagues, the organisation had no permanent officers, no fees and all decisions were made at a dinner after the meeting.

Br. J. Anaesth. 1978 50: 515-518

[xv] Post WWII Reader in Anaesthetics, Edinburgh.

Awareness [75]:

Payne considered awareness to be totally avoidable and that the aftermath for a patient was severe, that it was a form of post traumatic stress disorder and that the patients should receive appropriate support and therapy.

Multiple authorship 2000[76]:

This communication is really a moan about short articles that have six or seven authors names attached, he called these co-authors citation seekers and that it should be the responsibility of the reviewers and editors to curb the activity.

Jimmy Payne's career was certainly vibrant, as was his manner at research meetings, a brusque but able team leader.

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