JAW (Tony) Wildsmith

MD FRCA FRCPEd FRCSEd FDSRCSEng

JAWW was a consultant and senior lecturer in Edinburgh from 1977 until 1995 when he became professor in an independent department of anaesthesia in Dundee[i]. JAW Wildsmith is internationally known for his work on the use of local anaesthetics.


[ii]

His published work has been categorised as follows, some will inevitably overlap:

Comparisons of local anaesthetic agents, opiates and sedatives
Treatises on particular agents
Spinal, epidural and neuraxial anaesthesia
including baricity and spread of local anaesthetics
Axillary, brachial and other nerve blocks
Toxicity and allergy
Dental anaesthesia
Sedation

And then there were other subjects.

Carotid surgery
Intensive care
History

His publication history covers 39 years and started in 1972[iii]. The first four years, 1972-1975 inclusive, covered the subjects of serum

[i] J F Nunn. British Journal of Anaesthesia. 1999; 83(6): 916
[ii] Supplied by JAWW
[iii] Italicised text in quotes are either directly from JAWW or from publications

cholinesterase, pregnancy and suxamethonium [1], results of resuscitation from cardiac arrest [2], the effect of posture on the measurement of oesophageal pressure [3], haemodynamic effects of sodium nitroprusside [4] and sustained handgrip in patients with diabetes mellitus [5]. Another 1974 paper was on the subject of maintaining pulmonary nitrogenation during anaesthesia [6] and followed by blood-gas changes during nitroprusside induced hypotension [7, 8]. This was a staggering amount of research for a 'trainee', and of a very varied nature.

"My career in anaesthesia, including the academic component, was off to a prompt start for two reasons: first, I decided the specialty I wanted to pursue while still an undergraduate so I went straight to the RIE [Royal Infirmary of Edinburgh] *department from my house jobs; second, having passed the primary fellowship examination at the earliest opportunity, it was pointed out to me that I could not sit the final for another two years so that I had a year (assuming that it took a year to prepare – which it did!) to broaden my experience in some way. My earliest mentor, AHB Masson, suggested research training as an option, and that appealed so what followed is as much his fault as anyone's! The University of Edinburgh was kind enough to award me a research fellowship for this training, but I quickly realised that the best way to learn about any method is to use it, this perhaps influencing the wide range of the research topics..." "A key study was one on the haemodynamic effects of induced hypotension with sodium nitroprusside, this involving collaboration with both DB Scott (who had the necessary equipment) and WR MacRae (who used the agent clinically). Both became long-term supporters, and SNP was the subject of my MD thesis."*

"A year on I became a registrar, widening my clinical experience and passing the final fellowship examination ASAP. While gaining this wider experience I came to the view that regional anaesthesia was a much underused solution to many anaesthetic problems, a decision not without longer term significance! A full time clinical training post did not allow much research opportunity, but a lectureship in anaesthesia, specifically dental anaesthesia, became available. Contact with dentists sparked my interest in the early history of anaesthesia, and obviously the post offered time for research. During this period BG Covino, an American friend and regional anaesthesia collaborator of DB Scott, spent some time in Edinburgh, and between the three of us we devised a study of systemic concentrations of local anaesthetics after brachial plexus block, my first venture into regional anaesthetic research."

He became a consultant in 1977 and his first local anaesthetic related publication was in 1977.

"Appointed a consultant, I felt that it was high time someone was examining the routine use of spinal anaesthesia; then a little used technique in the UK. It was used in Edinburgh, but only for very major abdominal surgery and primarily for its hypotensive effect. Scott and Covino encouraged me not only to use the technique clinically, but also to study it, and study it as if it were an entirely new method. So began a series of studies which really only ended when I retired. I continued with studies of induced hypotension to complete my MD, but my interest in regional anaesthesia matters widened and came to dominate. A visit to the USA for the 1979 ASRA annual meeting made me feel that the Old World needed a similar society, and I helped to organise ESRA's first meeting in Edinburgh in 1982. Contact there with two people had particularly significant consequences. First, I met EN Armitage and, finding a like mind; I had a ready and complementary collaborator for Principles and Practice of Regional Anaesthesia, now in its fourth edition. The second person was Covino, by then chairman of department at the Brigham & Women's Hospital in Boston where he had started to build an unequalled regional anaesthetic research group. An invitation to join it was a huge opportunity, allowing me to extend my expertise into laboratory work, pursue my interest in the history of anaesthesia, meet many more like minded individuals than were then to be found in the UK, and show that I could work successfully in another setting."

"Back in Edinburgh AA Spence had followed JD Robertson as professor and, taking a much more pro-active approach to research, was very supportive of my adding laboratory studies to the existing programme of clinical research. My return coincided with a reorganisation of surgical services and I elected to join the newly formed (the first such in the UK) specialist vascular surgical unit which included the very research orientated surgeon, CV Ruckley. The other consultant anaesthetist in the unit was JH McClure with whom I had already collaborated on regional anaesthetic studies, and we settled down to a very productive period of clinical development and research activity. Scott had, for many years, obtained funding for a clinical research fellowship from the Swedish company Astra, and I was able to continue this after he retired, a major focus being studies of their new drug, ropivacaine, on which I wrote the Expert Clinical Report for the European Regulatory process. During this period I served as an elected member (1986-90)

of Council of the Association of Anaesthetists. Chairing the Education & Research committee gave me experience of organising postgraduate meetings and assessing research funding applications from others. Being asked to lead a working party on high dependency care stemmed very much from my involvement with vascular surgery."

One of the 'easier', if one should ever consider clinical research easy, forms of research is the comparative study. Over 25 years there were 13 studies; JAWW was, as the leader, the raiser of the money (grants), advisor to the primary author and editor/sub-editor of the paper.

Comparisons of local anaesthetic agents, opiates and sedatives

1983 midazolam vs. diazepam [29]. Sedation during spinal anesthesia with midazolam and diazepam was studied. The average doses required were 12 mg and 27 mg respectively for surgery of about one hour duration. Drowsiness post-operation was greater with diazepam and amnesia was greater after midazolam.

1991 ropivacaine vs. Bupivacaine (extradural) [67]. Various concentrations of ropivacaine (new at the time) were compared with 0.5 or 0.75% bupivacaine. There was little difference but ropivacaine had a slower onset, shorter duration and less intense motor block when compared with the same concentration of bupivacaine.

1991 extradural bupivacaine + diamorphine (either i.v. or extradural) [68]. Diamorphine 0.5 mg/hour was given either extradurally with bupivacaine 0.125% or as a supplement intravenously. The intravenous diamorphine group had inadequate analgesia but the patients in the extradural group had better analgesia but were drowsier.

1994 ropivacaine vs. bupivacaine [86], efficacy and kinetics. Three sets of patients received 1% ropivacaine, 0.5% ropivacaine or 0.5% bupivacaine extradurally. The groups' blocks were similar; however, the motor block using 0.5% ropivacaine was less dense and wore off more quickly than with bupivacaine. Cardiovascular changes were similar in all three groups. Ropivacaine's half life was shorter and the peak plasma concentration higher than bupivacaine.

1996 comparison of spinal needles [95]. A model involving fresh human lumbar dura was used to determine fluid leakage after puncture with

Sprotte, Atraucan, Quincke and Whitacre spinal needles. Unsurprisingly finer-gauge needles and pencil-point designs produce less leakage than traditional bevelled designs. The new Atraucan was considered worthy of further study.

1998 continuous vs. intermittent bupivacaine extradural anaesthesia [106]. Patients received either intermittent 0.375% bupivacaine hourly or as a constant infusion. The intermittent technique provided a more reliable sensory block.

2000 economic comparison of regional vs. general anaesthesia [120]. This was a complex audit. A computer database provided information about all aspects of the procedures. Regional anaesthetics took five minutes longer but recovery time was 10 minutes shorter. Anaesthetic times were five minutes longer for regional. A local field block with sedation *"was considerably cheaper than a general anaesthetic technique*", £67 vs. £102.

2001 ropivacaine vs. bupivacaine sciatic nerve block [128]. There was no difference.

2003 ropivacaine in glucose 5% vs. bupivacaine in glucose 8% [142]. Ropivacaine provided reliable spinal anaesthesia. The effect was shorter and was accompanied by less hypotension.

2005 plain vs. hyperbaric solutions of ropivacaine [149]. The onset time for hyperbaric ropivacaine was shorter than the plain solution; it spread higher and lasted much longer. The block was more reliable in the hyperbaric group and subsequent mobilisation was quicker.

2008 hyperbaric racemic bupivacaine vs. levobupivacaine vs. ropivacaine [168]. There were no differences in onset time, spread or time to maximum spread. However recovery from ropivacaine was quicker.

The study that is most intriguing to the author is the 1996 spinal needle study.

Treatises on particular agents:

Lignocaine [27, 66]

A letter confirming an acute allergy to amide local anaesthetics: [27].

The effects of two plasma concentrations of lignocaine on performance were assessed using a battery of performance tests [66]. Subjects were aware of the effects and it was suggested that patient's reports of effects might be useful.

Prilocaine [47, 61, 65, 100, 102]

Two of these papers involved the use of prilocaine for intravenous anaesthesia. Intravenous regional anaesthesia was performed using either prilocaine with saline or sodium bicarbonate. The bicarbonate increased the speed of onset and full recovery was slower [61]. In the second paper fentanyl was added - there was no change in the speed of onset but the incidence of nausea increased after tourniquet release; so no benefit [65].

Bupivacaine [18, 20, 23, 39, 45, 52, 54, 62, 67, 68, 77, 78, 86, 98, 106, 127-129, 142, 168, 176] Two have been selected -

A letter: Intravenous regional anaesthesia using bupivacaine has a higher risk of systemic toxicity than prilocaine and so prilocaine must remain the drug of choice [18].

The second letter reiterates the dangers of leakage past a tourniquet and subsequent toxicity – there is also a short piece of doggerel in response to a spelling error. I leave you to discover it! [23]

Ropivacaine [67, 86, 126-128, 131, 142, 149, 153, 155, 168, 176]

Hypersensitivity due to ropivacaine [153]: Another letter about hypersensitivity, this time in response to a report by two Japanese clinicians who were treating pain associated with herpes zoster with epidural ropivacaine over a two week period. The discussion was around whether it was hyper-sensitivity or toxicity or due to plasticisers in the containers.

Intermittent vs. continuous administration of epidural ropivacaine with fentanyl for analgesia during labour [155]. In this randomized, double-blind study of primigravid patients it was determined that the intermittent group, which required fewer additional injections, had better analgesia and therefore the technique represented a superior mode of analgesia.

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Neuraxial anaesthesia

KEYWORDS	
Spinal anaesthesia	[19, 26, 28, 29, 32, 39, 49, 56, 58, 60, 69, 77, 87, 97, 101, 109, 123, 124, 129, 131, 142, 149, 152, 158, 165, 168, 173, 175, 176]
Extradural/Epidural anaesthesia:	[42, 45, 54-56, 67, 68, 78, 79, 83, 84, 86, 90, 98, 106, 109, 112, 123, 126, 135, 153, 155-157, 162, 171, 178]
'Neuraxial'	[167, 172, 177]

Spinal anaesthesia:
The first 'spinal' was given by either Corning, in 1885, or Bier in 1898.

It is fascinating to see certain subjects of study remaining popular over decades. The subject of baricity of solutions for spinal anaesthesia in JAWW's work covers three [19, 26, 28, 32, 149, 168].

Baricity - Glucose, and Posture, and spread of local anaesthetics:

Baricity - Glucose [26, 62, 88, 129, 131, 142]

1982. 1, 2 and 4 ml of isobaric amethocaine was injected at two rates of injection (1 ml per 5 s and per 10 s). Larger volumes had little effect on the height of the blockade and the extent of block was less predictable [26].

1990. The spread of intrathecal injections of bupivacaine containing glucose was measured. The greatest spread was with 8% glucose compared with 0.83% and 0.33%. The lowest concentration produced greater variability in spread, 8% the fastest onset of sensory block [62].

In 1994 another similar study produced similar results [88]. In 2001, however, it was shown that "... the *spread of spinal solutions in the pregnant patient at term is not dependent on density*". CSF density decreases during pregnancy and this study used glucose 8 mg ml^{-1} and glucose 80 mg ml^{-1}. Speed of onset and patient satisfaction were similar [129]. Also in 2001, it was the turn of ropivacaine plus glucose [131]. The onset of sensory block was

significantly faster with the higher concentration of glucose but the maximum cephalad spread was similar. There was no significant difference between the motor block and the time to complete regression. The final publication in this group [142] was a comparison of ropivacaine with bupivacaine, both with glucose). Ropivacaine took, on average, three minutes longer to get a sensory block to T10; the average duration of sensory was twice as long with bupivacaine and they required more treatment for hypotension. As might be expected the ropivacaine patients mobilized sooner. An erratum was published late as the points on a graph were incorrectly labelled.

Posture [3, 24, 35]

1981. Effects of posture on the spread of isobaric and hyperbaric amethocaine [24]. The spread of isobaric solutions was not affected by gravity; the spread of hyperbaric solutions was affected but posture was ineffectual in controlling the spread. Dose affected duration rather than spread.

1983. Barbotage and spinal anaesthesia [32]. This was a letter in response to an article by PJ Nightingale. Wildsmith was stressing that volume does not affect spread and that 'plain' 0.5% is mildly hypobaric at body temperature.

[35] This publication in 1985 was a letter in disagreement about what was stated in a paper by I F Russell[iv]. Russell was of the view that "... *the spread of analgesia against the effects of gravity implies that postural changes independent of gravity are responsible for the extension of analgesia.*"

2002. Head-up tilt and subarachnoid block [140]. "*I have concerns.*" Another letter in response to another article, this time by Loke et al.[v]. JAWW questioned the idea that although the difference of the height of the block by one dermatome may have been statistically significant, was it clinically significant? It was similar with the changes in blood pressure. The response of Loke et al. maintained their viewpoint and they said that "*the possible impact of the collective data should not be dismissed so lightly*".

Predicting the spread of local anaesthetics in the spinal canal was another theme. [26, 58, 77].

Predicting the spread of spinal anaesthesia [58] 1989.

[iv] Anaesthesia 1984; 39: 865-7

[v] Loke et al. Anaesthesia 2002; 57: 169–72

An editorial: This was an overview of the problem of predicting block height with special reference to a research paper in the journal. He discussed the hypothesis that low lumbar injection of local anaesthetic goes into a "*lumbar CSF collection*" and that this "buffers" the spread. This hypothesis was supported by other previous work[vi].

Prediction of the spread of repeated spinal anaesthesia with bupivacaine [77]1992. Another letter: This time it was about a paper by Tuominen et al.[vii]. They reported a study of patients having repeat spinal anaesthetics and said that "*Individual anatomical properties*" affect spread more than expected which suggested to JAWW that baricity was said to be "*relatively unimportant*" and he argued against this. There was a slight barb in the tail of the authors' response – "*We do appreciate the vast literature on regional anaesthesia ... However, we are also prepared to accept new opinions...*".

Spinal anaesthesia was conventionally a single shot technique. However this was to change [69, 87].

A study of 20 patients using a 24g catheter – two patients required a general anaesthetic; one because of inadequate spread of local anaesthetic and the other because of kinking of the catheter preventing a second dose being given [69].

And…

The management of blood pressure: A review article on the topic of prevention and management of hypotension (due to central neural blockade) was published in 1993 [80]. In 2000, [124], a letter about the use of vasopressor vs. the use of fluids – arguments on both sides. The second, another letter, about the complex clinical scenario of management of hypotension, that resulted in a myocardial infarction, in a young patient; strongly argued positions presented. [175]

And…

[High] segmental spinal for cholecystectomy [158, 165]. The need for a laparoscopic cholecystectomy under regional anaesthesia was because of the patient's severe chronic pulmonary disease. The needle was inserted at the 10th

[vi] Foelschow et al. Regional Anaesthesia 1982;7:79
[vii] Tuominen M et al. Br J Anaesth 1992;68:136-8

thoracic interspace and bupivacaine and sufentanil was injected into the CSF, an epidural catheter was also inserted; the resulting block was between T3 and L2. Ephedrine and fluid were given and the circulation was stable following initial hypotension [158]. Following this case report a formal study was carried out where 20 patients had high spinals for laparoscopic surgery. None had to be converted to general anaesthesia. However, it was stressed that spinal anaesthesia above the level at which the spinal cord terminated has to be done with extra care [165].

Extradural/Epidural anaesthesia:

Epidural abscess [84, 90, 156]

The first is an editorial, the second also – about the risk of epidural abscess formation. Causal factors include poor asepsis, direct contamination from nearby bacteria, haematogenous bacteria and the presence of an epidural catheter. It is quite critical of the clinical practice described in an accompanying paper[viii] in which patients with infections had epidurals. However, at least the clinicians did not use the technique in patients on steroids and the blocks were at lumbar level – steroid therapy and thoracic blocks being other risk factors. Carson and JAWW made the point that "no risk is acceptable unless there is very clear benefit.

The third is a review article of 144 references.

Haematoma [79, 112, 162]

The second publication is one of two letters in the same journal – the Wildsmith team were obviously keen letter writers. They were commenting on the use of anticoagulants during epidural anaesthesia [ix]. The sort of dose they would recommend was in the range of 3–5000 units. A much higher dose was considered "*incautious*". Continuing the epidural into the second post-op day may also have hidden the diagnosis. Skilton and Justice strongly defended their position. The third publication is a similar situation seven years later, this time with the use of other agents with anticoagulant effects.

[viii] Jakobsen KB et al. Br J Anaesth 1995;75:536-540

[ix] Skilton & Justice. Anaesthesia 1998; 53: 691–701

Other complications [56, 78, 104, 172]

The first report was a case report: a spinal anaesthetic, following a failed epidural, with 1.6 ml of 0.5% heavy bupivacaine produced a sensory blockade to T2. Because of dyspnoea and distress general anaesthesia was necessary; intravenous ephedrine and fluids were given in response to severe hypotension. Eventually a healthy infant was delivered Caesarean section.

The third – an editorial on accidental intravenous injection of local anaesthetic when doing epidurals – an avoidable event

The fourth publication was the report of an audit carried out on behalf of The Royal College of Anaesthetists. A national audit over two weeks was carried out to determine the rate of all major complications. Symptoms lasting for more than six months were defined as permanent. The census produced a denominator of over 700,000 central neuraxial blocks. There were eighty-four major complications; "*Two-thirds of initially disabling injuries resolved fully*." The incidence of permanent injury was estimated 'pessimistically' as 4.2 (2.9-6.1) per 100,000 and 'optimistically' at 2.0 (1.1- 3.3).

Who might benefit from, or be harmed by, epidural anaesthesia and analgesia? [171]

This is obviously a crucial question. Although complications from the use of spinal/epidural blocks are rare they can be devastating and the risks have to be compatible with the benefits. This was another letter in response to a previous editorial and subsequent discussion. There is a lot of detail in the letter about the pros and cons and relative risks of epidural analgesia/anaesthesia. From the UK point of view it was thought that "*elderly patients undergoing emergency laparotomy are a group with the potential to gain most from epidural anaesthesia*" and it was suggested that if a cost benefit analysis of a suitable sized population was undertaken, the elderly might be the clinical population to study.

'Neuraxial':

The first two papers with this keyword were audits and the third about risk definition [167, 172, 177].

The first was the report of a two-week national audit of the use of neuraxial block. It involved 304 National Health Service hospitals, 90% of the responses were judged to be 'accurate'. The number of procedures reported was 27,533 equivalent to about 700,000 major blocks annually. These data

were to be used as denominators for the calculation of the incidences of complications.

The second determined the complication rate. There were 84 major complications and 52 met the inclusion criteria (see above). The data were considered reassuring central neural blockade had a low incidence of major complications.

The third publication was in response to a letter by Kirkham, Payne and Cooper[x]. Their audit results suggested that the incidences of complications were considered "*clinically useful by 64% of the anaesthetists surveyed and 38% had altered the statistics provided to patients in line with the audit findings*". However, further clarification was required. Cook, Counsell and Wildsmith responded by explaining the necessity for the complex nature of the presentation of the numbers. They also thought that the major problem was the dissemination of the message despite a multi-pronged effort. They thought that they had "*... done quite well compared with others (well, we would wouldn't we!)*". They did agree that a national survey of the impact of the audit was necessary and said that it was underway.

Brachial plexus block:

Axillary: "Axillary brachial plexus block: method of choice?" A review article [63].

Interscalene: Plasma concentrations of local anaesthetics after interscalene brachial plexus block [9].

Three methods: Plasma prilocaine concentrations after three techniques of brachial plexus blockade [47]. Axillary, perivascular subclavian and interscalene blocks were done with 35 ml of 1.5% prilocaine. There was no significant difference in the prilocaine concentrations.

Prolonged block: This was a supraclavicular brachial plexus block with 0.42% bupivacaine. The motor and sensory block lasted 26 hours but there was full recovery at 40 hours [52].

Nerve blocks: [40, 43, 59, 71, 146]

These papers are mainly about nerve conduction, the first about peripheral nerves and local anaesthetic drugs in a symposium on local

[x] Kirkham L, Payne S and Cooper R. British Journal of Anaesthesia 104 (5): 656–66 (2010)

anaesthesia in the Br. J. Anaesth. The second about differential nerve blockade: esters v. amides and the influence of pKa and the third about structure-activity relationships in differential nerve block at high and low frequency stimulation.

Differential nerve blockade is about how pharmacological agents affect the different types of nerve fibres, A B and C. This last study was in rabbits to determine their sensitivity to local anaesthetics. The A fibres were the most sensitive and the C fibres the least. Low pKa and high lipid solubility was best at blocking A fibres; high pKa and low solubility, C fibres. Local anaesthetics of the amide type have a high pKa and low lipid solubility and can produce differential C fibre block.

Only the last reference discusses a technique for a nerve block; in this case a sciatic nerve block

Toxicity of local anaesthetic agents:

This subject is addressed in three letters -

2000 [125]– natural killer cells are a type of cytotoxic lymphocyte that are known to kill tumour cells. This letter is in response to a research paper that suggested that local anaesthetics, by abolishing pain, abolished this activity. This could lead to wound infection and tumour spread. JAWW said that "*The battle to improve the quality of surgical pain relief is difficult enough without such unqualified statements.*" And "*Perhaps they [the authors] would revert to using no anaesthesia or analgesia at all!*" I like the following quotation –"*Observation is easy. Considering the relevance of the observation is altogether more difficult.*"

2006 [159]– local anaesthetic toxicity – prevention or cure.

This was all about the use of lipid for the management of bupivacaine cardiotoxicity and is a response to an editorial. There were some concerns about the "*somewhat over-enthusiastic advocacy*" of untested proposals. The main concern was the emphasis on 'curing' rather than 'preventing' the problem'. Care with dosage was of great importance.

2008 [170]– local anaesthetic toxicity – this is a further treatise on the management of local anaesthetic toxicity and JAWW emphasises first principles in the management of patient resuscitation before specific therapy. Responses by the Honorary Secretary, AAGBI, on behalf of the AAGBI Working Party on

Local Anaesthetic Toxicity ("*How refreshing to have to reply to a letter of support*!") and the author of the original case report are printed.

Allergy and anaphylaxis:

The first is a case report (1981). A patient with allergy to lignocaine was challenged with an intradermal injection of bupivacaine. This resulted in a systemic reaction. There was a decrease in complement C4 suggesting an immunological cause. It was reported as the first of documented by concurrent immunological changes [21].

1993: A letter disputing the cause of an anaphylactoid reaction [83].

1997: Another article disputing causes of allergic reactions during local anaesthetic procedures – this time the description of a reaction that may have been caused by latex rather than the local anaesthetic. Careful assessment is necessary [98].

In 1998 [110] there was a review of 25 patients diagnosed as having local anaesthetic allergy, the review included intradermal testing. Only one patient was genuinely allergic to an amide local anaesthetic. Reactions should be carefully assessed.

2009: JAWW was part of a team that had produced a set of guidelines from the Association of Anaesthetists of Great Britain and Ireland about Suspected Anaphylactic Reactions Associated with Anaesthesia [174]. In brief – morbidity may be reduced if diagnosis is early; initial management should use the ABC approach with the use of adrenaline given as early as possible. The patient should be investigated further - tryptase levels may help and specialist (allergy) referral advised. All cases should be reported to the national databases and all departments should identify a consultant anaesthetist with specific responsibility for anaphylaxis.

Local anaesthesia is associated with dentistry – no 1940s child could forget. Between 1998 and 2005 there were a series of communications on the subject of dental anaesthesia – however this was about sedation and general anaesthesia for dental surgery. Death in the dental chair was a catastrophe and had been highlighted and addressed since 1969, and was addressed again [108]. Conscious dental sedation in 1999 [118] (in the British Dental Journal) and a comment about another sedation technique in 2005 [154], and another comment in the European Journal of Anaesthesiology in 2002 [137]. The 2000 paper was an audit of paediatric dental anaesthesia in Scotland [121].

There were other publications on sedation: 1983 (midazolam vs. diazepam during spinal anaesthesia [29]), 2007 (an update on dental sedation [163]), 2008 (monitoring during sedation [169]) and 2011 (radiologists and sedation – do they follow their guidelines? [180]).

Before leaving the subject of local anaesthesia completely we must deal with the subject of carotid endarterectomy. The last publication first; "*Regional anaesthesia for carotid endarterectomy*" [117]. This is a letter about the difficulties associated with studies comparing general anaesthesia with local anaesthesia. JAWW's preference was for a combined approach – GA +LA. All the other papers were about carotid blood flow:

1991 Transcranial Doppler monitoring [70]
1992 Carotid endarterectomy: future perspectives [76]
1993 Middle cerebral artery blood flow after clamp release [81]
1993 Hyperaemic response after carotid endarterectomy [82]
1994 Extracranial Doppler ultrasonographic flowmeter [89]
1995 Cerebral oximetry [92]

Something different: Induced hypotension

Induced hypotension was a major facet of anaesthesia in the 1970s, the most non-specific, and less controllable, technique being a combination of curare and halothane; ganglion blocking agents were also used.

Nitroprusside became the short acting agent of choice that could be titrated to effect.

It was noted in this first paper [4] that there were "... *marked falls in arterial pressure, peripheral resistance and central venous pressure. Heart rate and cardiac output rose while stroke volume was little changed. All parameters returned quickly to control values on discontinuation of sodium nitroprusside administration.*" The perfect agent?

In 1975 the blood gas changes during induced hypotension with nitroprusside was presented at the Manchester ARS meeting [8], later published in full [7]. PaO_2 declined but returned to the previous values on cessation of the nitroprusside infusion. It was thought to be due to altered ventilation/perfusion ratios.

Nitroprusside did have the potential for toxic side effects and so a technique that could reduce the dosage was a good idea – the combination with trimetaphan was studied and seen to be satisfactory [17, 22, 33].

A letter in 1979 discusses the safe administration of nitroprusside – amongst other details it reminded the writer that during profound hypotension the cerebral function monitor that we used was the rate and rhythm of respiration – so no IPPV [16][xi].

In 1987 a study was performed in a further attempt to reduce doses by giving beta-blockers preoperatively – it was a step too far – too profound bradycardia – not recommended [41].

"*In 1994 my interest was sought in the new academic department of anaesthesia being established in Dundee. All three of our children were by then at University so the timing was good and the opportunity to widen my activities appealing. The members of the Dundee department were regional anaesthesia enthusiasts, but there were other research opportunities as well. Collaboration with the pharmacologist, JJ Lambert, on mechanisms of anaesthetic action was anticipated, but studies with a nursing lecturer, JE Rattray, on quality of life after intensive care, were not! A revision of the undergraduate medical curriculum allowed me to introduce the concept of Acute Care, duties in the Dental Hospital brought me back into contact with that specialty, and a pan-Scotland collaboration between academic departments led to the development of a simulation centre."*

Intensive care: [107, 147, 150, 164]
Janice Rattray and Marie Johnston were, apart from JAWW, common authors for these four papers that spanned a decade, 1998-2007. The first was about the quality of life of survivors of intensive care. According to this study quality of life did not change much (assessed retrospectively). It appeared that the main factors in quality of life were people, family and leisure activities.

In 2004 they attempted to assess the perceptions of patients' experiences in intensive care by the development of an intensive care experience (ICE) questionnaire. It was determined that there were four components: **'awareness of surroundings'**, **'frightening experiences'**, **'recall of experience'** and **'satisfaction with care'**. A year later another publication,

[xi] This monitoring concept shocked trainees in the 1990s and 2000s!

this time on predictors of emotional outcome. It would appear that the severity of the illness was not a factor but there were both objective (*length of stay*) and subjective (*patient characteristics*) factors that helped..

In 2007 a randomised study of post-discharge care was planned (multi-centre with a big team) to assess the emotional impact of the intensive care experience and to see what benefit a special set of clinic attendances might have on the final physical and psychological health of the patient. A pragmatic, randomised, controlled trial of intensive care follow up programmes in improving longer-term outcomes from critical illness[xii] .

Now to something more retrospective - History:

"On retirement (2007) I decided that someone who is 'history' should only pursue that aspect of his former discipline! I have been (2008-10) President of the History of Anaesthesia Society and am currently (2012-15) Honorary Archivist to the Royal College of Anaesthetists."

JAWW was interested in this retrospective aspect of anaesthesia from early on.

1984: Local anaesthetic drugs--an historical perspective [34]
1985: Horace Wells [36]
1985: Origins of local anaesthesia [37]
1987/88: A British footnote to the life of Horace Wells [44, 51]
1997: So just who was James "Young" Simpson? [99]
1999: Donald Bruce Scott, M.D., F.R.C.A., F.R.D.P.Ed. 1925-1998 [115]

2001: **No sceptic me, but the long day's task is not yet done**: the 2002 Gaston Labat lecture [138]. This was an eponymous lecture given to the American Society of Regional Anesthesia. As he says "... *the purposes of an eponymous lecture, an obvious one is to honor the subject*[xiii]*. A secondary purpose might be to honor the lecturer, and Dr. Winnie's introduction was most kind.* "

[xii] BMJ 2009;339:b3723: Not effective or cost effective in improving patients' quality of life.

[xiii] Gaston Labat 1876-1934

The following is included as it gives more of a personal view of JAWW:

"*I certainly wish that my parents had been alive to hear it. My father would have enjoyed it immensely, and my mother, bless her, would have believed every word. However, my wife, Fay, did no more than tolerate his kind words. If I do not keep my ego under control, she will certainly do it for me, so I must deny that this lecture is to honor me*."

There are 231 publications listed below; 65 are letters/comments (his "Disgusted of Dundee" publications[xiv]), 16 are editorials, 9 classified as historical articles and 14 as reviews. This still leaves 132 study reports.

The summaries above are an incomplete description of all the work undertaken but it will have given the reader an insight into the nature of the breadth of the studies. Here are some publications that may be of interest to the reader – you may wish to chase them for yourself [10, 15, 25, 75, 94, 101, 114, 133, 136, 141, 151, 161]!

JAW (Tony) Wildsmith has influenced the safety of local anaesthesia, addressed the problems associated with sedation for procedures and had forays into cerebral blood flow, intensive care and history; a significant body of work.

Addendum:
Searches of computer databases are never foolproof and many of JAWW's publications were missed. Tony Wildsmith has been kind enough to add further detail to the above description of his work. The main purpose of this book has been to highlight themes in an academic's life's work and to try and shed some light on the personality behind the publications and so his own words will help.

The text below is that supplied by JAW himself. A few paragraphs have been included above.

[xiv] Personal communication!

"My career can be divided into five, very different phases:

1. *1970-1977 - Training in Edinburgh;*
2. *1977-1984 - Early consultant posts in Edinburgh, ending with a year in Boston;*
3. *1984-1995 - Consultant to the Edinburgh vascular surgery unit;*
4. *1995-2007 - Foundation Professor in Dundee; and*
5. *2007-???? - Retirement.*

Each phase had its influence on my clinical and academic interests, with the latter developing early through the combination of two factors. First, I was attracted to anaesthesia as a career while an undergraduate, and a four-week 'elective' attachment to the RIE department confirmed my choice of specialty. Thus, I went straight from pre-registration house jobs to training in anaesthesia and was off to a prompt start. Second, having passed the primary fellowship examination at the earliest opportunity, I was advised that I could not sit the final for another two years so that I had a year (assuming that it took a year to prepare) to broaden my experience in some way. My earliest mentor, AHB Masson, suggested research training as one option; it appealed, so what followed is as much his fault as anyone's! The University of Edinburgh was kind enough to award me a research fellowship for this training, but I quickly realised that the best way to learn about any method is to use it, this resulting in the range of topics of my early publications [4, 6]. A key study was on the haemodynamic effects of induced hypotension with sodium nitroprusside, this involving collaboration with DB Scott (who had the necessary equipment) and WR MacRae (who used the agent clinically)[4]. They both joined Alastair Masson as long-term supporters, and SNP was the subject of my MD thesis. Also important during that year was spending one day per week in theatre with HWC Griffiths, pioneer of induced hypotension with high spinal anaesthesia.

A year later I became a registrar, widening my clinical experience and passing the final fellowship at the earliest opportunity. While gaining this wider experience I came to the view that regional anaesthesia was a much-underused solution to many anaesthetic problems, a decision not without longer-term significance! A full time clinical training post did not allow much research opportunity, but a lectureship (specifically in dental anaesthesia) became available in the University Department (head, Prof JD Robertson) in 1975. Contact with dentists triggered my interest in the history of anaesthesia, and the post offered time for more

research [7, 11, 13] as well as teaching dental students. During this period BG Covino, an American friend and collaborator of Bruce Scott, spent some time in Edinburgh, and they encouraged me in my enthusiasm for Alon Winnie's interscalene brachial plexus technique. Importantly, they also suggested using it for research so we devised a study of systemic concentrations of local anaesthetics after brachial plexus block, my first venture into regional anaesthetic research [9].

At that time spinal anaesthesia was a technique used rarely in the UK, and even in Edinburgh considered only for very major abdominal surgery, primarily for its hypotensive effect. However, discussions with Griffiths, Scott and others (especially those who had done locums in Sweden where spinals were used widely) had convinced me that the reasons for its unpopularity in the UK (fear of neurological sequelae) were invalid. Thus, appointment as a consultant in 1977 gave me the clinical freedom to pursue this view, with Scott and Covino again being supportive. They not only encouraged me to use spinal anaesthesia clinically, but also to study it as if it were an entirely new method, and so began a series of publications[19, 24, 26, 29] which ended only once I had retired [172]. I continued the research into induced hypotension to complete my MD [17, 22, 33, 47], and later co-edited one of the very few texts on this subject [Induced Hypotension. MacRae WR, Wildsmith JAW (Eds). Amsterdam: Elsevier, 1991]. However, my interest in regional anaesthesia came to dominate, and widened to include early forays into alleged local anaesthetic allergy [21, 27] and the correct technique for intravenous regional anaesthesia [18], both becoming long term interests. Enthused to encourage others to use regional anaesthesia by the example seen at the 1979 ASRA meeting I instigated an annual local anaesthesia demonstration course in Edinburgh in 1980, and from that realized that a British book on regional anaesthesia was needed to guide its use in British conditions.

In 1982 I helped organize ESRA's original meeting [Regional Anaesthesia: 1884-1984. Scott DB, McClure JH, Wildsmith JAW (Eds). Sodertalje: ICM AB, 1984 (Proceedings of the first ESRA meeting)] also in Edinburgh, and this involvement had three important personal consequences. First, I undertook my first historical research project for a presentation at the meeting [185]. Second, in meeting EN Armitage I found both a like mind and a complementary collaborator for a British book on regional anaesthesia; Principles and Practice of Regional Anaesthesia is now in its fourth edition. Third, Ben Covino was at the meeting and, by then,

chairman of department at the Brigham & Women's Hospital in Boston where he had built an unequalled regional anaesthetic research group. He invited me to join him for a while, a huge opportunity which allowed me to extend my experience into laboratory work, learn much about the pharmacology of local anaesthetic drugs, pursue my interest in the history of anaesthesia, meet many more like minded individuals than were then to be found in the UK, and show that I could work successfully in another setting. The year was very enjoyable, immediately productive [36, 38, 39, 41, 43, 186-188] and had a long-lasting significance for my career. Several subsequent publications stemmed directly from the knowledge or expertise which I acquired during that year [37, 40, 41, 191] and the background influence lasted much longer.

Back in Edinburgh, AA Spence had succeeded JD Robertson as professor and, taking a much more pro-active approach to research, was very supportive, with expanded departmental accommodation allowing for a wider range of studies, including the technique I had used in Boston [61, 66]. My return also coincided with reorganisation of surgical services, and I elected to join the newly formed specialist vascular surgical unit, the first such in the UK. Staff included the research-orientated surgeon, CV Ruckley, and another consultant anaesthetist, JH McClure, with whom I had worked previously. The group settled down to a productive period of vascular related research [75, 76, 82, 86, 88, 92, 121], edited a book on postoperative analgesia [Conduction Blockade for Postoperative Analgesia] and published other related reviews [41, 42, 53, 69, 87]. The regional block studies also continued [39, 45, 46, 48, 106, 189, 190, 196], many supported by funding (originally started by Scott) from Astra for a clinical research fellowship, and a programme of epidural (single-use for surgery [54, 59] and continuous for post-operative analgesia [87, 157] research was added, leading to work on the new drug, ropivacaine [see below]. As an elected member (1986-90) of Council of the Association of Anaesthetists I chaired two groups: first, the Education & Research committee, organising larger postgraduate meetings and assessing research funding applications; and second, a working party on high dependency care, this stemming very much from my involvement with vascular surgery [Chairman of the Working Party: The High Dependency Unit - Acute Care in the Future. London: Association of Anaesthetists of Great Britain and Ireland, 1991]

In 1994 my interest was sought in the new academic department being established in Dundee. All three of our children were by then at University so the timing was good and the opportunity to widen my activities appealing. The members of the Dundee department were regional anaesthesia enthusiasts, but there were other research opportunities as well. Collaboration with the pharmacologist, JJ Lambert [148, 204, 205], on mechanisms of general anaesthesia was anticipated, but studies with a nursing lecturer, JE Rattray (she just appeared one day and asked for my help!), and a psychologist, M Johnston, on quality of life after intensive care, were not [107, 147, 150, 164]. My regional block research took longer to re-establish, but I was busy enough starting a new department, developing other research, and co-editing Anaesthesia for Vascular Surgery! When regional studies restarted they were along similar lines [128], but with some new topics and a focus on ropivacaine [67, 86, 126-128, 131, 142, 149, 153, 155, 168, 176], again with generous support from (by then) AstraZeneca. This collaboration with industry also involved writing the original Expert Clinical Report (and some later addenda) on ropivacaine for the European Regulatory process [Clinical Expert Report: Ropivacaine, 1995; Addendum to Clinical Expert Report: Ropivacaine, 1998 and Clinical Documentation on Ropivacaine: Intra-articular Administration, 2001]. As noted above, I had become interested in local anaesthetic allergy in Edinburgh, and this continued and broadened in Dundee [98, 110, 122], primarily by investigating (and usually refuting the diagnosis in) individual patients, but also contributing to a national guideline [174]. An unusual collaboration was advising some Dutch colleagues who were using intrathecal injection in the thoracic region in high risk patients [158, 165].

Research activity predominates in academic circles today, but I have always felt that the teaching of others is vital, if only to encourage the next generation of researchers. Most of my first-named co-authors were trainees and I hope that they gained more from the experience than just CV development! My duties in Dundee included contributions to the undergraduate medical and dental curricula, [Dental local anaesthesia: Course workbook and "Skilled task teaching and assessment" [210] and at postgraduate level I was involved in the establishment of the Scottish Computerized Simulation Centre ['A beginning, not an end' A Report from the Royal College of Anaesthetists Simulation Working Group, 2005]. The emphasis placed on research (especially grant income) during the 1990s led many to take a very pessimistic view of the future for academic anaesthesia, a view which I found disappointing. Indeed, one of the reasons I took the chair in

Dundee was to show that the future is not totally bleak, but it is challenging, and the specialty has to recognize and meet that challenge. Thus, while a member of Council (1998-2008) of the Royal College of Anaesthetists and chair of its Academic Committee, I persuaded the College to undertake a review of academic anaesthesia, [A National Strategy for Academic Anaesthesia. London: Royal College of Anaesthetists, 2005 and "A national strategy for academic anaesthesia: an overview" [224] this resulting in the formation of the National Institute of Academic Anaesthesia to provide a focus for all those interested in the issues.

My other major area of activity as a member of College Council started at my very first meeting when I disagreed with a motion relating to the teaching of dental students, this resulting in an immediate invitation to chair the dental anaesthesia committee! General anaesthesia was still used in many 'high street' dental practices in the UK then, but not always to modern standards in spite of previous attempts to improve matters, so a report making recommendations agreed by all the relevant organisations (i.e. dental and anaesthetic) was produced [Standards & Guidelines for General Anaesthesia for Dentistry. London: Royal College of Anaesthetists, 1999]. Unfortunately, individuals do not always adhere to published standards, with those in working with general anaesthesia in dentistry seemingly prone to ignore such advice [121]. Almost inevitably, disasters (usually the death of a healthy young child) continued to occur, and these attracted increasing media attention until the Department of Health decreed that general anaesthesia should be restricted to the 'hospital' setting. Only techniques of 'conscious sedation' were to be used in dental practices, a sensible recommendation in itself, but these methods are used widely across medicine, as well as dentistry and are not without their own risks if not used correctly (too often they aren't). Thus I chaired another group, formed under the auspices of the Academy of Medical Royal Colleges, to look at these techniques in all settings [Implementing and Ensuring Safe Sedation Practice for Healthcare Procedures in Adults. London: U.K. Academy of Medical Royal Colleges, 2001], and was involved in the production of a number of related publications [8. Conscious sedation in termination of pregnancy: Report of the Department of Health Expert Group. London: Department of Health, 2002, 10. Guidelines for Conscious Sedation in the Provision of Dental Care. National Dental Advisory Committee. London: Department of Health, 2003, 12. Standards for Conscious Sedation in Dentistry: Alternative Techniques. Joint Working Group, Faculties of Dental Surgery & General Dental Practice, RCSEng and Royal College of Anaesthetists, 2007 and

"Sedation in a radiology department - do radiologists follow their own guidelines?" [180].

From my earliest involvement in regional anaesthesia research I was keen to promote the view that the techniques used should be both safe and effective. That was the specific reason for the study of systemic concentrations of local anaesthetics produced by the very large dose needed (in the pre-ultrasound era) for brachial plexus block, and the insistence on an exact technique for IVRA mentioned earlier. When regional anaesthesia was used rarely in the UK, those of us prepared to do so recognised that there were colleagues prepared to cry 'foul' if something went wrong so we were very aware of complications and how to avoid them. As the benefits became known and usage increased, knowledge of complications perhaps lagged behind, yet the introduction of anti-thrombotic therapy into surgical practice added another risk factor, particularly for central neuraxial blocks. There are perhaps too many to reference specifically here, but a large proportion of my editorials and review papers address the issues surrounding complications, but some examples are worth highlighting [60, 73, 80, 138, 156]. I also feel that persuading those who produced the earliest authoritative UK guideline on antithrombotic therapy that they should include 'anaesthetic' issues was important [Prophylaxis of Venous Thromboembolism. Edinburgh: Scottish Intercollegiate Guideline Network, 2002]

My final contribution in this area was to persuade the Royal College of Anaesthetists to devote one of its national audits to assessment of the exact scale of the problem after central neuraxial blocks in the UK [Major Neuraxial Complications of Central Nerve Block in the United Kingdom: The 3rd National Audit Project of the Royal College of Anaesthetists. London: Royal College of Anaesthetists, 2009, [225], However, these were not the end of written contributions on this subject; against my better judgement (I believe that retired clinicians should stop speaking about their former interests) I was persuaded to give a presentation to a College meeting (and write an editorial), essentially on the relative indications for postoperative epidural analgesia [227]. My strictures did stir up a member of the audience to object, and I am glad to be out of it now, but I will never accept that performing an epidural on an anaesthetized yet healthy child was an appropriate technique for analgesia after laparoscopic surgery. That the child was rendered paraplegic does, sadly, rather prove my point.

After my year in Boston I had little time for researching the history of anaesthesia, but a number of subsequent publications were based on my existing knowledge -

[History and development of local anaesthesia, The history and development of induced hypotension, Recognition from Britain: The Horace Wells Testimonial Fund, History of Pain Relief in Childbirth, Memorabilia of Wells and Morton in New England: The Relics of Injustice and So just who was James 'Young' Simpson?]

As retirement approached, and my other activities lessened or were assumed by others (see below), I had time once again to go searching ancient publications for forgotten people and events, with the focus on local anaesthetic drugs and those who use them -

[So just who was James 'Young' Simpson?, British Pioneers of Regional Anaesthesia, Lidocaine: a more complicated story than simple chemistry suggests [226] Some (mostly Scottish) local anaesthetic heroes [228], European Society of Regional Anaesthesia and Pain Medicine (1982 2012): Thirty years strong [229], From cocaine to lidocaine: great progress with a tragic ending [231]].

Such historical activity continues, and for the last three years I have been Honorary Archivist to the College in London, with the launch of a biographical project on our early fellows (of the then Faculty of Anaesthetists) due later this year.

In the above I have focused on publications related to my major interests, and named my significant mentors and collaborators. I have made reference to encouraging trainees, and they are mostly the first named authors in many of my papers, with me last as leader of the research group, not as head of department! The joy of that role is bringing others up to the status of independent workers, and I am delighted to have supervised the higher degrees of Janice Rattray (quality of life after intensive care), Cameron Weir (now the primary anaesthetic collaborator with Jerry Lambert) and Graeme MacLeod (a growing authority in regional anaesthesia). My successor is a pharmacologist, but at least there is still a department (not the case everywhere in the UK), with Cameron, Graeme and Paul Fettes (who took on supervision of the undergraduate teaching) as integral parts of it.

Books and chapters *(list supplied by JAWW)*

a. *Scott DB, McClure JH, Wildsmith JAW (Eds). Regional Anaesthesia: 1884-1984. Sodertalje: ICM AB, 1984 (Proceedings of the first ESRA meeting)*

b. *Wildsmith JAW, Armitage EN (Eds). Principles and Practice of Regional Anaesthesia. Edinburgh: Churchill Livingstone: First Edition 1987; German Translation 1991; Second Edition 1993*

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w. *Morton CPJ, Wildsmith JAW. Crises in regional anaesthesia. In: Fisher MMcD (Ed). The Anaesthetic Crisis. London: Bailliere 1993: 357-376*

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