

Felicity Reynolds

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Felicity Reynolds was a senior lecturer and head of Department at St Thomas' Hospital from 1978 until 1984 when she became Reader in pharmacology applied to anaesthesia. From 1989 – 1994 she was the Chairperson of Guy's and Thomas' Division of Anaesthetics



and became professor of Obstetric anaesthesia in 1992; she retired in 1996ⁱⁱ. Information from the Obstetric Anaesthetists' Association's website has provided further details of her career. She qualified in medicine in 1960 and trained in anaesthesia in Southampton gaining her Fellowship in 1963. After a year in Uganda she spent eleven years in pharmacology. Her MD in 1971 was titled '*The systemic toxicity of local anaesthetic drugs, with special reference to bupivacaine.*' The OAA website details her many awards.

She wrote many reviews [1-12], editorials [9, 13-23] and letters [24-41], [42-50], [51-56] and [57-68]. Some of these will be addressed later.

The vast majority of her publications are about anaesthesia/ analgesia for maternal and foetal health. In 1968 her first publication, with AH Beckett, was on the topic of the measurement of bupivacaine, lignocaine and mepivacaine in blood using gas-liquid chromatography [69]. This was obviously a key paper as it provided a tool for many investigations. Concentrations as low as 0.04 µg/ml of local anaesthetic could be measured. This publication was followed a year later by '*Blood levels of bupivacaine in obstetric analgesia*' [70]. For almost thirty years this was a topic of investigation [71-76], these citations being just the tip of the iceberg.

In the second paper, with Taylor, Rouss and Beazley, a study of bupivacaine for paracervical blocks with or without epinenephine, bupivacaine was shown to provide effective analgesia without high maternal or

ⁱ Photograph courtesy of FR

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

foetal blood levels, or foetal bradycardia. However the delivery did not take place within an hour of injection. A follow up paper in 1972 [77] was not so positive. The same technique was used in patients due to undergo Caesarean section. The babies were delivered between 5 and 30 minutes after the injection. Under these conditions plasma concentrations of bupivacaine alone were double those where epinephrine was also used. However, concentrations of bupivacaine were not high in the neonates. The authors' bottom line was that because local anaesthetic toxicity was considered a possible cause of foetal bradycardia and intrauterine death, there was reasonable evidence to restrict the concentration of bupivacaine to no more than 0.25% for paracervical block in obstetrics.

Paracervical blocks went 'out of fashion' about this time and continuous epidural (extradural) blocks became a routine method of providing analgesia for delivery. Subsequent work on blood levels of bupivacaine was therefore related to epidural analgesia. Not only was the efficacy of the epidural examined but the effects on the neonate.

It is impossible to have a strict demarcation between studies but the structure of this chapter is along the lines of a) obstetric related pharmacokinetics, b) bupivacaine and fentanyl for epidurals, c) effects of agents on the foetus/neonate d) Caesarean Section and then e) maternal health. This will be followed by some miscellany.

a) Obstetric related pharmacokinetics

After the first two papers in 1968/69 we move on to 1970 [71] "*Maternal and neonatal blood concentrations of bupivacaine: a comparison with lignocaine during continuous extradural analgesia*". Lumbar extradural analgesia was carried out in 29 patients during labour. A set of patients had either bupivacaine or lignocaine for extradural analgesia. The concentrations of the agents in maternal blood and umbilical blood were measured. The results suggested that bupivacaine was a more practical drug and had less potential toxicity.

1971 [72] "*Plasma concentrations of bupivacaine during continuous epidural analgesia in labour: the effect of adrenaline*". This was important to do because of the almost routine use of adrenaline in local anaesthetic solutions to prolong the duration of the nerve block. It was a double-blind study; the duration of action with adrenaline was not significantly increased but maternal plasma concentrations were low in both groups but lower with adrenaline at

the end of labour when higher doses of bupivacaine were used. Neonatal concentrations were always low but a marked difference between the two groups of the maternal/umbilical vein concentration ratio was inexplicable.

Also in 1971 were two studies using mepivacaine. Mepivacaine is a homologue of bupivacaine. Its metabolism and excretion were tested in male volunteers [78]. Sixteen per cent of the mepivacaine and 6% of bupivacaine was excreted unchanged. However, the blood concentrations of bupivacaine fell more rapidly initially than those of mepivacaine. The other study [79] was a comparison of the potential toxicity of bupivacaine, lignocaine and mepivacaine. The methodology was complex but the conclusion was that bupivacaine produced lower blood concentrations and mepivacaine the highest, bupivacaine was therefore considered to have the higher safety margin. [In the author's experience mepivacaine has not been seen to be used in clinical practice.]

1972: The placental transfer of bupivacaine after paracervical block has been described above in the introductory paragraphs [77]. A letter to the *Lancet* in this year pointed out some errors in an editorial on the use of vasoconstrictor agents in local-anaesthetic preparations [80].

1973: [73] Another paper on "*Maternal and foetal plasma concentrations of bupivacaine after epidural block*". In brief, the concentration of bupivacaine in those mothers who had bupivacaine and adrenaline was 0.36 ± 0.03 $\mu\text{g/ml}$ (SE), without adrenaline 0.54 ± 0.05 $\mu\text{g/ml}$, significant at $p < 0.001$. The umbilical vein concentration was also lower with adrenaline. It was suggested that without adrenaline the data showed that 5% of mothers could exhibit systemic toxicity if the bupivacaine dose exceeded 320mg.

Over the next 13 years vasoactivity was studied in detail. The method of choice was the intradermal injection of the test drugs.

1976: [74] "*The effect of concentration on vasoactivity of bupivacaine and lignocaine*". Doses of the drugs were given intradermally to volunteers and it was observed that vasoconstriction occurred at low concentrations and vasodilatation at high concentrations. Only bupivacaine 0.5%, had a longer lasting effect. The next paper was another intradermal study, this time of aptocaine [81]. Aptocaine was more active and long-lasting than lignocaine and prilocaine and appeared longer-lasting than bupivacaine. It had marked vasoconstrictor activity and it was suggested that aptocaine merited clinical trials for use in dentistry.

1978 [82] "*An intradermal study of the local anaesthetic and vascular effects of the isomers of bupivacaine*". A vasoconstrictor effect was only seen with the L(-)-bupivacaine and it had a longer duration of analgesic action than the (D+)-isomer

1981 [83] "*An intradermal study of the local anaesthetic and vascular effects of the isomers of mepivacaine*". Both were vasoconstrictor but the L isomer produced more vasodilation (and haemorrhagic change). The L isomer lasted significantly longer.

1985 [84] "*Comparison of the vasoactivity of amide and ester local anaesthetics. An intradermal study*". The two types of local anaesthetic were tested for their vasoactivity, the ester-linked local anaesthetics, procaine and amethocaine and the amide-linked - cinchocaine, lignocaine, mepivacaine and prilocaine. The ester-linked agents produced vasodilatation, mepivacaine (amide) caused vasoconstriction but the other three amides had more variable effects.

1988 [75] "*Effect of adrenaline on extradural anaesthesia and plasma bupivacaine concentrations during caesarean section*". In a randomised study, patients having elective CS received either 0.5% bupivacaine with or without adrenaline. The plain group needed more additional analgesia and plasma bupivacaine concentrations were higher. Did bupivacaine concentrations remain higher in the extradural space with adrenaline [Author]? In the emergency CS group there were no significant differences. It was concluded that "... *extradural adrenaline does not usefully reduce systemic absorption of 0.5% bupivacaine, but may improve its efficacy in extradural anaesthesia for elective Caesarean section.*"

1988: [85] "*H2 antagonists and bupivacaine clearance*". H2 receptor antagonists, cimetidine and ranitidine, were given to women undergoing elective Caesarean section under epidural anaesthesia. No significant difference was found between the control, cimetidine and ranitidine groups. Neither did the H2 receptor antagonists alter other pharmacokinetic parameters.

1989: [86] "*Effect of time and adrenaline on the feto-maternal distribution of bupivacaine*". This was a study of 80 women, half for elective and half for emergency CS. They were allocated to receive either plain bupivacaine or bupivacaine with adrenaline. The concentrations of bupivacaine in maternal veins, umbilical vein and artery were not affected by adrenaline, but were correlated with the first dose to delivery interval. Foetal accumulation of

bupivacaine did not occur beyond a first dose to delivery interval of 30-40 minutes.

1992 [76] "*Plasma total and free concentrations of bupivacaine and lignocaine in mother and fetus following epidural administration, singly or together*". Forty six women for elective CS were given various combinations of bupivacaine, adrenaline and lignocaine. Protein binding occurred more in the mother than the baby. Plasma concentrations were considered to be below toxic levels; adrenaline did not reduce the maximum levels of free bupivacaine in the mother but did appear to increase foetal uptake of bupivacaine.

2000 [87] "*Chemical stability of bupivacaine, lidocaine and epinephrine in pH-adjusted solutions*". In some centres epinephrine and sodium bicarbonate may be premixed with local anaesthetic solutions in order to facilitate the quality of epidural anaesthesia for emergency Caesarean sections. The study was to assess the stability of such a pre-mixed solution. The alkalinised solutions reduced the epinephrine concentrations significantly over 24 hours but bupivacaine and lidocaine concentrations were unaffected. The authors did not recommend the practice.

The unique aspect of obstetric pharmacokinetics is the placenta and the transfer of drugs across its membranes; not easy to study in patients. From 1984 to 1992 a series of studies were carried out on the placental transfer of drugs in the rabbit, bupivacaine, lignocaine, pethidine and anticonvulsants.

In 1984 doe rabbits were given an intravenous infusion of pethidine, lignocaine, bupivacaine and antipyrine concurrently and the umbilical 'blood' flow rate was varied [88]. Concentrations of the drugs in maternal plasma and umbilical effluent were measured. The results showed the same pattern as is observed in humans. It was determined that placental clearance of drugs increased with flow rate, the transfer rate was reduced by maternal protein binding and was flow-dependent at low flows and permeability-dependent at high flows for the less lipid-soluble compounds.

In a very similar study reported in 1985 [89] the flow rate and protein content of the placental perfusate were varied. The clearance of unbound antipyrine was unchanged with perfusate protein, the 20-30% protein bound lignocaine and pethidine clearance increased slightly and bupivacaine (80% bound) increased 'markedly', but was one-tenth to one-fifth that of the other drugs. From this work it was concluded that the foetal dose would "*be greatest in healthy babies with good placental blood flows and high plasma proteins*". However, bupivacaine did have the lowest transfer rate.

In 1989 Laishley, Carson, and Reynolds wrote two papers on the effect of adrenaline on the distribution and transfer of bupivacaine to the rabbit foetus [90, 91]. Adrenaline may influence transplacental distribution of drugs by decreasing uterine blood flow. In this study it was associated with higher concentrations of bupivacaine in the placenta but there was no other significant effect on foetal bupivacaine concentrations. In the other complex study it was concluded that neither adrenaline nor minor alterations in maternal placental flow affect placental transfer of bupivacaine.

In 1992 [92] in another rabbit pharmacokinetic study pethidine was eliminated more rapidly than bupivacaine and the elimination rates were ranked as maternal plasma > placenta > amniotic fluid > foetal brain > foetal plasma. Analysis showed that the “... *maternal plasma half-lives for pethidine and bupivacaine were 1.0 and 2.0 h, and placental half-lives 1.9 and 2.5 h, respectively. The apparent fetal plasma half-life of pethidine was 9.9 h while there was apparently no net elimination of bupivacaine from fetal plasma*”. These latter half-lives are significantly long.

Anticonvulsants are used in obstetric practice for the management of eclampsia. In 1976 Reynolds demonstrated that salivary drug concentrations correlated with the amount of free phenytoin in plasma and that it was a convenient way to monitor patients [93]. There are eight further communications about anticonvulsant monitoring [63, 94-100]. Of importance is the kinetics of phenytoin during pregnancy and the puerperium. They measured phenytoin levels in 11 pregnant epileptics and in non-pregnant women. The saliva:plasma ratio increased to maximal values at delivery and returned to non-pregnant values within 2-8 weeks. Dose increments had to be changed to maintain therapeutic levels and after delivery doses were reduced to avoid toxicity.

The other drug added to the mix was fentanyl – a combination of fentanyl and bupivacaine in epidurals became commonplace. The word combination ‘epidural fentanyl’ first appeared in 1981 (Google Ngram viewer). The pharmacokinetics was examined [101, 102] but there were many more clinically orientated studies (see below) – of pain in labour [103-107], of the effect on the neonate and of the effect on gastric emptying.

In the 1983 study [101] patients in labour were given either epidural or intramuscular fentanyl, together with an epidural test dose of bupivacaine. Pain relief was quicker and more effective for those given epidural fentanyl; they couldn’t rule out a systemic effect by the fentanyl. A study in rabbits three

years later [102] showed that the transfer of fentanyl from the maternal circulation across the placenta was intermediate between that of pethidine and bupivacaine. The clearance of unbound fentanyl was higher than the others and was not affected by them.

These pharmacokinetic studies entailed an enormous amount of both clinical and laboratory work.

b) Clinical use of bupivacaine and fentanyl for epidurals

In 1982 (one year after the Ngram's detection of the phrase 'epidural fentanyl') a study of the effect of extradural fentanyl was published [103]. This was a double-blind trial in the first stage of labour where either fentanyl or saline was mixed with the test dose of extradural bupivacaine. Within one hour additional bupivacaine was required in about $\frac{1}{4}$ of patients in the fentanyl group and in about $\frac{3}{4}$ of patients in the saline group; a significant finding. There were no serious side-effects although some patients in the fentanyl group had mild itching.

Further work in 1985 [104] reinforced the efficacy of extradural fentanyl and that the presence of fentanyl in the systemic circulation makes a negligible contribution to analgesia; similarly with perineal pain in labour (1989) [105]. A comparison of epidural fentanyl and sufentanil was carried out in 1993; it showed very little difference [106].

Motor blockade limiting the movement and expulsive efforts of the mother can be a clinical problem and was investigated in 1995 [107]. Women in labour were given either epidural plain bupivacaine (0.125%) or 0.0625% bupivacaine containing 2.5 mcg/ml fentanyl. Those patients having the lower dose bupivacaine took longer to full cervical dilation. In this study fentanyl did not reduce the incidence of perineal pain. More women had motor blockade after 0.125% bupivacaine; however, satisfaction with epidural analgesia was similar in both groups. The following year the relationship between motor blockade and spontaneous delivery rates were addressed [108]. A motor block was less common in the fentanyl/low-dose-bupivacaine combination group but this did not produce an increase in spontaneous deliveries. This scenario was re-addressed seven years later producing a similar bottom-line – there was no increase in normal delivery rate with the low dose, reduced motor blockade, drug combination [109].

c) Effects on the foetus/neonate

1984: [40] A letter to *Anesthesia and Analgesia* about neonatal neurobehavioral responses after epidural anaesthesia destroys the conclusions reached by Kileff ME et al. that 2% lidocaine was a suitable substitute for bupivacaine. Reynolds “... *emphasize[s] that 0.5% bupivacaine correctly placed in the epidural space in a dose of 2 mg/kg or that less is outstandingly safe and unlikely to produce central nervous, or still less, cardiovascular, side effects.*”

Ten years later in another letter she addressed “*The effects of maternal epidural anaesthesia on neonatal behaviour during the first month*” again [59]. She destroys the methodology of another set of researchers – Sepkoski et al. Their approach certainly seems crude.

Moving on - in 1995 the effect of maternal hypoxaemia (the influence of analgesia) on neonatal outcome was studied [110]. In retrospect, 51 parturients were recorded as having either “... *no analgesia, pethidine with intermittent Entonox, extradural bupivacaine ... “or a bupivacaine/fentanyl mixture.* The lowest incidence of hypoxaemia, SpO₂ < 94%, was in the extradural bupivacaine group. No correlation was found between maternal hypoxaemia and measures of neonatal outcome.

Again, in 1997 [111] it was found that the dose of fentanyl used in combined spinal/epidural analgesia appeared to have a negligible effect on neonatal condition.

Another study in 1998 reinforced this [112], an erratum appears in *Anesthesiology* 1998 Dec;89(6):1615, it is a correction to a table.

In 2002 Reynolds et al. assessed the effect of epidural versus systemic labour analgesia on foetal/neonatal acid-base status at birth by a systematic review of trials, from five countries, comparing epidural with systemic opioid analgesia [113]. They included both published and unpublished results. Epidural analgesia was associated with improved acid-base status even though epidural analgesia can cause maternal hypo-tension, fever, longer labour and more instrumental deliveries. They suggested that these potentially adverse factors outweighed the benefits of neonatal acid-base status.

In 2005 another meta-analysis indicated that the foetal welfare following Caesarean Section was uninfluenced by the type of neuraxial block or general anaesthesia [114].

In 2010 a review of “*The effects of maternal labour analgesia on the foetus*” was published [11]. In brief, it said that foetal metabolic acidosis is caused by labour pain and stress, and systemic opioids cause foetal/ neonatal

depression and affect breast feeding, doing little to help the stress and pain. Meta-analysis of studies showed that the Apgar scoreⁱⁱⁱ is better after epidural analgesia and neonatal acid-base balance is improved. A similar review came out in 2011 [12], "*Labour analgesia and the baby: good news is no news.*" Reynolds again said that "*Widespread ignorance of the benefit to the newborn of neuraxial labour analgesia in the UK among non-anaesthetists needs to be combated*" and that the only promising alternative was remifentanyl.

d) Caesarean Section (CS)

The CS rate has increased over the last few decades in western obstetric practice. The anaesthetic technique used changing from routine general anaesthesia to neuraxial block – spinal or epidural analgesia, or a combination of both.

Posture during labour, and Caesarean Section, has been a point of discussion for many. The supine pregnant woman at term runs the risk of reduced blood pressure because of inferior vena cava compression. Reynolds investigated the effect of posture post-epidural insertion on the effect of the neuraxial block 1983 [115]. The patients were either turned to the right lateral position or the supine position. The only significant difference was that motor block occurred more in the lateral position ($p < 0.02$). The circulatory disadvantage of the supine position was still considered a significant drawback.

Another aspect of the technique used was studied in 1984 [116]; there were many confounding factors but it was suggested that there was a small subset of patients where epidurals may have increased the likelihood of instrumental delivery.

Changing from epidural analgesia to anaesthesia for CS can be required at any time and in 1991 [117] the effect of 20 ml of 2% lignocaine + 1/200,000 adrenaline was assessed; it produced quick onset blocks that provided adequate anaesthesia for surgery in all patients.

Postoperative CS pain was also addressed – either epidural diamorphine or intramuscular papavaretum (Omnopon) [118]. Epidural diamorphine was best and patients liked the pain free mobility. In '93 a similar study [119], comparing on-demand epidural diamorphine with intravenous

ⁱⁱⁱ A scoring system for the assessment of babies immediately after birth devised by Virginia Apgar

patient-controlled diamorphine the magnitude of side effects were similar in the two groups and overall satisfaction was high, but the patient-controlled analgesia group scored higher.

It has always been easy to get a higher epidural block but to get a lower one can be a problem. In a '94 study epidurals were either inserted with the patient either in a 25° head up position or in the horizontal position. The head up position resulted in more sacral sensory blocks [120]. Later, in 2001, the effect of the supine wedged position, compared with the lateral position, on spread of spinal anaesthesia was investigated [121]. There was no significant difference between the groups in fall of blood pressure or requirement for a vasopressor but the spinal block in supine patients rose more rapidly and was more predictable.

No procedure is without its hazards; general anaesthesia - airway problems, local anaesthetic techniques - nerve damage. Dural puncture is another potential complication which, with an epidural needle, can lead to loss of CSF and result in a debilitating headache. It is commonly associated with the novice 'epiduralist' but it can happen even when done by the experienced practitioner. An audit of 257 (191 responded) obstetric units in the UK determined the rate of accidental dural punctures in the years 1991-1995. The highest rates were in smaller units (3.6%) and lowest (0.19%) in a larger high throughput unit. The rate using saline to detect the epidural space was 0.69%, air 1.11% ($P < 0.001$) [122].

Finding the dural space is obviously of prime importance and the intricacy of how this is achieved comes down to both intellectual and practical skills. A letter in 2005 discusses the various ways of holding the needle and syringe (Son of Doughty technique) [33]! An accompanying letter by JA Wildsmith reports a conversation with Andrew Doughty where he replies that 'his' so-called technique is based on "*You are ashamed of yourself if you puncture the dura*".

Both spinal anaesthesia and epidural anaesthesia have advantages; combined spinal-epidural anaesthesia entered the scene in the 1990s; a more complex procedure and the possibility of increased risk of damage. In 2000 an audit of 222 departments reported a total of 56 patients with prolonged neurological problems [123]. Eighteen problems were attributed to the regional technique but there was no obvious difference in incidence between the techniques. The numbers were too small.

Seven cases of neurological damage were reported a year later and were of significant clinical effect [124]; the needles were thought to be inserted at the L2-3 interspace. The tip of the lower end of the spinal cord is usually at the L1-2 level but the surface anatomy makes it difficult to be absolutely sure of the level used. Because of this, it was recommended that anaesthetists should not insert the needle above L3.

In the last three decades informed consent, about risks associated with procedures, has become mandatory. A survey in 1995 [125] of 523 members of the Obstetric Anaesthetists' Association showed that the majority (63%) would recommend regional anaesthesia, 5% general anaesthesia and the remainder would allow the patient, after full discussion, to decide. This was followed by a letter [42], stating that doctors in Britain and Ireland tailor information on risks to what the mother needs and wants to know; "*... it should not be regarded as paternalistic*" A General Medical Council directive about full disclosure, with the danger of scaring patients, needed to be revisited.

In 2005 Wee, Brown and Reynolds wrote an article on the anaesthetics aspects of the National Institute for Clinical Excellence (NICE) report guidelines for CS [126]. It is a comprehensive practical guide.

We will now have a brief diversion and go to Malawi. An audit of 8070 caesarean sections; 94% were emergencies and eighty five women died, almost two thirds died postoperatively on the wards. Amongst many major causes of morbidity/mortality were the lack of adequate anaesthetic training and the use of general as opposed to spinal anaesthesia. The perinatal mortality was 11.2% in the first 72 hours. It was recommended that improved training in anaesthetics, wider use of spinal anaesthesia and improved postoperative care might reduce mortality [127].

e) Maternal health

Reynolds interests were not only for the drugs, the techniques and the baby. Maternal health was always important.

1993 [128]: The factors associated with long term backache after childbirth were studied. Thirty percent reported long term backache; 15% said they had had no previous back pain. Younger women, unmarried women, those reporting other antenatal symptoms and those who had epidural analgesia reported more. *...“There were no differences in the nature of the backache between those who had or had not received epidural analgesia in labour.”*

1995: “*Maternal sequelae of childbirth*”, an editorial in the British Journal of Anaesthesia, amongst many other things, addressed the problem of blaming all sequelae on the epidural [9]. This was a common theme.

The next, in 1996, [10] was a case-report-based review discussing the problem of sciatic nerve palsy after delivery by Caesarean section. To determine the cause was very important as the cause might be unrelated to the epidural and it was suggested that opioids in epidurals reduced the amount of local anaesthetic used and therefore the nerve block/motor block was reduced. Weakness or flaccidity could then be seen as caused by something other than the epidural and nurses should be educated to recognise the importance of this sign of neuropathic problems.

Backache was a perennial topic of dispute and in 1996 [129], in a prospective randomised study, the relationship between motor block and long term backache was reported. The conclusion was that ... “*There were no significant differences between the treatment groups in the incidence of postnatal backache overall or of new backache or any symptoms after childbirth.*” Backache prior to, or during, pregnancy were associated with backache after childbirth.

Other reports in 1997, 1998, 1998 [18, 41, 48] were supplements to this work. Backache usually resolves soon after delivery and epidurals were often blamed. Similar backaches were reported by 40% of mothers who did not have regional anaesthesia and the backache may be related to hormonal changes, ligamentous laxity and the expulsive forces associated with labour. There was some criticism of the work of others^{iv} in this article which stimulated another letter, Reynolds reinforced her view. A correction was published later but it is a typographical error rather than scientific.

[45] “*Epidurals and backache: again?*” 2002: It is worthwhile reading this in full. She takes the BMJ to task because “... *it took more persuading [the BMJ] to publish prospective studies with negative results, and it flatly refused to publish one showing epidurals were good for babies—good news is no news.*”

^{iv} MacArthur C, Lewis M, Knox. BMJ 1997;315:679

Letters

Felicity Reynolds was a multi-letter writer. To the journal *Anaesthesia* she wrote 16 [24-39] , to the BMJ nine [42-50], to the BJA, *The Lancet* and the *International Journal of Obstetric Anesthesia* four each [51-54] [63-66] [57, 60-62]. There were other letters to other journals.

Not all the publications regarding aspects of obstetric anaesthesia have been noted but there is a sufficient amount to show the depth of her investigations.

And now for something completely different, but not completely non-obstetric:

Upper oesophageal sphincter pressure

One of the major risks of anaesthesia, at any time but particularly during pregnancy, is the risk of regurgitated stomach contents entering the airway and flooding the lungs. With RG Vanner as the lead author there are three papers in 1992 on the subject of upper oesophageal sphincter pressure – the effect of cricoid pressure, inhalational induction and intravenous induction of anaesthesia [130-132]. Cricoid pressure is the application of force to the cricoid cartilage in such a way as to occlude the lumen of the oesophagus and therefore impede the passive flow of gastric contents into the pharynx.

Firstly the effect of cricoid pressure: The upper oesophageal sphincter pressure was measured in 24 patients and the median pressure prior to anaesthesia was 38 mmHg; after anaesthesia and paralysis it was 6 mmHg. Using a special yoke providing cricoid pressure with a force of 40 N the sphincter pressure increased to above 38 mmHg. However, the application of cricoid pressure by operating department assistants (anaesthetic technicians/assistant) achieved this level in only half of the patients. Laryngoscopy made little difference.

Inhalational induction (not common in adults but can be necessary). It was shown that halothane did not have a dose-related effect on sphincter pressure and, unlike thiopentone or suxamethonium, maintained a degree of upper oesophageal sphincter tone. Three patients in the study of 30 did have sphincter pressures of less than 10 mmHg and therefore gastric reflux into the pharynx was a possibility.

Intravenous Induction: It was shown that midazolam and thiopentone both reduced the sphincter pressure to low levels and that the rapid fall with thiopentone was such that it occurred before loss of consciousness. From this it

was suggested that cricoid pressure should be applied very early during the induction process. Ketamine had no effect on the oesophageal sphincter pressure.

The bottom line was, apply cricoid pressure early and the anaesthetic technicians have to apply more pressure to achieve oesophageal occlusion.

Odds and ends

A title that caught my attention was “*A book that informed my practice*”. It turned out to be a book review [133] – “*What a Blessing She Had Chloroform*” by Donald Caton, Yale University Press, 1999. A small quotation from the review “*Historically, women have demanded analgesia in labor when the medical profession approached it with caution, yet now that it has become vastly safer, many women reject it as dangerous.*”



So true, with the passage of time safety has increased a thousand fold but expectations that nothing should go wrong have increased in a similar manner.

Another eye-catcher “*Logic in the safe practice of spinal anaesthesia*” [20]. The word logic is not often seen alongside a clinical technique. She is once again addressing the danger of using the L2-3 interspace – it’s too high – the risk of conus damage too great and identifying the space by surface land-marks prone to inaccuracy.

Finally, again in 2008, “*Obstetric problems? Blame the epidural!*” [134]. I don’t need to comment; she has said it all!

^vPhotograph courtesy of Obstetric Anaesthetists Association. <http://www.oaa-anaes.ac.uk/content.asp?ContentID=39>

Books

Anesthesia and the Fetus by Yehuda Ginosar, Felicity Reynolds, Stephen H. Halpern and Carl Weiner (Nov 20, 2012) Wiley Blackwell

Pain Relief in Labour by Felicity Reynolds, Robin Russell, Jackie Porter and Mark Scrutton (Oct 22, 1997) BMJ

Regional Analgesia in Obstetrics: A Millennium Update by Felicity Reynolds (Jun 23, 2000) Springer

Epidural and Spinal Blockade in Obstetrics by Felicity Reynolds (Nov 1990)
Obstetric Anaesthetists' Association

Effects on the Baby of Maternal Analgesia & Anaesthesia by Reynolds and Felicity Reynolds (Jan 1993)

Lectures

She has delivered numerous eponymous lectures, has had many honorary awards and became the founding editor (now editor emeritus) of the International Journal of Obstetric Anesthesia. See the <http://www.oaa-anaes.ac.uk/> website for details.

A truly productive academic life!

An anecdote

At a research meeting I attended the opening remark by the chairman was "Good morning gentlemen". Felicity Reynolds was the first speaker. "Good morning ladies" she said. The audience laughed. The chairman was bemused.

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