

Alastair Spence CBE MD FRCA

In 1965/6 Alastair Spence was a Research Fellow in Leeds, becoming Head of Department at the Western Infirmary in Glasgow in 1969. In 1975 he became a Reader and was Professor between 1978 and 1984. He moved to Edinburgh as Head of Department and



as Professor in April 1984. He was President of the Royal College of Anaesthetists (1991-94) and retired in July 1998ⁱ.

Twenty percent of Alastair Spence's published output was on the topic of theatre pollution and its effects on staff, and the adverse effects on patients of inhalational agents. Another large section was on the subject of postoperative hypoxaemia, both the physiology and clinical ramifications and management. The remaining miscellaneous publications included matters pertaining to the College, to teaching/training and to aspects of worldwide anaesthetic practice

His first publication, 1967, was in the journal *Anaesthesia*, observations on intragastric pressure [1] with Moir and Finlay. In non-pregnant patients the intragastric pressure was 5-11 mmHg during anaesthesia, in term pregnant patients it was 5-13mmHg; these observations were confirmed by La Cour (1970) and Hartsilver et al (1999).ⁱⁱⁱ In the same year an Anaesthetic Research Society abstract was published in the *British Journal of Anaesthesia* and was titled "The influence on renal function of chloroform and halothane anaesthesia in man" [2] with Linton and Patel. The author was surprised to read that chloroform was still being used, presumably, regularly in 1967. Sixty patients were randomly allocated to

ⁱ Photo courtesy of the Wood Library-Museum of Anesthesiology, Park Ridge, IL.

ⁱⁱ Nunn JF. *B.J.A.* 1999;83:916

ⁱⁱⁱ La Cour D. *Acta Anesthesiol Scand.* 1970;14:5-15 and Hartsilver EL et al *B.J.A.* 1999;82:752-4

chloroform or a halothane anaesthetic. It was shown that, using a urea excretion test, that chloroform patients had greater renal dysfunction.

Postoperative hypoxaemia

Lung function in the postoperative period became an early subject. In 1968 Spence, together with G Smith (later to become professor of anaesthesia at Leicester), with Harris wrote on “the influence of continuous extradural analgesia on lung function in the postoperative period”[3]. This was followed in 1970 by a comparison of morphine with extradural nerve block on postoperative lung function [4], and similar papers in 1971 [5], 1973 [6] and 1975 [7]. This latter paper compared patients receiving either continuous extradural analgesia or narcotic analgesia following upper abdominal surgery; there were no statistically insignificant improvements in PaO₂ in the extradural group. A conclusion they drew from this was that abdominal muscle spasm was not an important factor in postoperative arterial hypoxaemia, a hypothesis that they had put forward in 1972 [8].

A paper in 1973 reported the “apparent” improvement in lung function when using Entonox [9]...the use of the word “apparent” is interesting, almost as if they did not believe their results. Entonox was used for post herniorrhaphy pain and was associated with an increase in functional residual capacity, the morphine group showed a decrease.

In 1977 the difference in lung function between those patients whose lungs were ventilated with oxygen and nitrogen were compared with those ventilated with oxygen and nitrous oxide... no difference was found [10]. Postoperative hypoxaemia and its relation to age was also studied and reported in a letter to Anesthesiology [11]; the PaO₂ when breathing air could be calculated using the regression equation ... $81 - (0.285 \times \text{age})$.

The subject of the mechanism of postoperative hypoxaemia was the subject of several ‘overview’ type publications [8, 12].

Over the next three years there was an intense effort with Smith, Dewar, Alexander, Davis and others on the physiology of postoperative oxygenation.

In 1970 another ARS presentation, “the effect of air at 1 and 2 atmospheres absolute” on respiratory mechanics, ‘normal’ volunteers were studied as well as a group of patients with chronic bronchitis. With increasing pressure the flow resistance increased but in the ‘normal’ volunteers compliance and lung volumes did not. Some of the bronchitic patients, three, out of twelve had large increases in expiratory resistance and expiratory reserve volume. The possible use of the hyperbaric air was broached as it might achieve more uniform emptying of alveolar units [13].

Airway closure (the closing of lower airways during exhalation at low lung volumes) was studied as a possible cause of postoperative hypoxaemia [14-17]. It is the author’s view that Alexander, the principal author in all papers, drove these studies. Lung function studies after elective surgery showed that functional residual capacity was reduced and was worse in those patients having upper abdominal surgery. This was related to an increase in alveolar/arterial pO_2 difference and to the amount of closure of small airways. The concept of airway closure during part of the respiratory cycle helped to explain the changes in ventilation/perfusion and intra-pulmonary shunting. These papers involved a considerable amount of work.

Apart from these clinical studies there was a series of more basic physiological research projects. In 1970 there were two publications on the topic of a nitrogen rebreathing method for the estimation of PvO_2 , both with Ellis (of malignant hyperpyrexia fame – Leeds)[18, 19]. This was a mathematical and experimental evaluation of the technique as PvO_2 is a useful index of either whole body tissue oxygenation or as a component of the Fick equation for determining cardiac output. The final result involves many assumptions and, after equilibration of the bag gas with lung gas, the implication is that the gas has come into equilibrium with mixed venous gas. It would appear to be a technique with many possible confounding facets.

The effects of hyperoxia were also studied [20, 21]. Breathing 100% oxygen at 2 Ata for 5 hours caused an increase in airways resistance of 30%, thoracic gas volume increased by a quarter and specific airways conductance by 41%. Those volunteers breathing an air equivalent at 2Ata had no significant change. Excess oxygen is not good.

General clinical studies:

As in all clinical academic departments there is the usual collection of drug comparison studies and case reports, Spence and his co-workers were no different. Tubocurarine and pancuronium [22], metoclopramide [23], pentazocine [24], glycopyrrolate [25], etomidate [26], alfentanil[27] and ketorolac[28]. The alfentanil study, a double blinded study, is interesting in that the intra-operatively the patients had smaller minute volumes, lower respiratory rates and obtunded increases in heart rate (not surprising) but that they required more analgesia postoperatively because, it was postulated, they did not take up so much volatile agent...this was considered an improvement in the anesthetic technique.

Other studies include the effect on wound healing of nitrous oxide (in rats) [29]; no effect. Does thiopentone affect the speed of onset on previously injected non-depolarising agents [30]? Not really! There were also four studies on the new agent – propofol [31-34], the first a dose finding study compared with Althesin, the second a study of the new formulation designed to avoid Cremophor-related reactions, the third and fourth a comparison with Althesin and methohexitone respectively.

Occupational hazards:

Pollution of the operating theatre atmosphere and its affects on staff was the subject of another major block of work. It started in 1972/3 [35, 36] and continued through to 1991 [37-44].

Knill-Jones was involved in many of these publications... the first study was a comparison between married women anaesthetists with non-anaesthetist

married women doctors. It was reported that spontaneous abortion was significantly greater in those associated with anaesthesia, there were more congenital abnormalities and the involuntary infertility rate was higher.

An article in the *Annals of the Royal College of Surgeons of England* Spence describes the nature and determinants of air pollution in the operating theatre and referring to a study by Pfaffli et al.^{iv} he stated that the average levels of halothane and nitrous oxide contamination were 15ppm and 170ppm respectively. A study by Vaiseman^v had reported spontaneous abortion by 18 out of 35 pregnancies in nursing staff. In this article he is careful to point out that there was no direct evidence to indicate that the polluted theatre atmosphere was the cause of these obstetric mishaps but suggested that there was some urgency to remove contaminant gases.

In 1975 a survey of almost 8000 male doctors in the UK was reported [39], this showed that paternal exposure to anaesthetic contaminants did not affect the abortion rate, congenital abnormality rate or involuntary infertility. However maternal exposure was associated with an increase of 1.6 – 2.7 times the risk of non-exposed pregnancies.

A combined analysis of USA and UK surveys [40] reaffirmed the increased risks and also showed an increased frequency of hepatic disease amongst anaesthetists. In a review in 1978 these findings were reiterated and there was also a call for the audit of causes of death in relation to the type of work undertaken as there were some anxieties about the possibility of leukaemia and lymphoma [41].

In 1987 we move onto environmental pollution by inhalational anaesthetic agents [45]. However the title is deceptive as the 'environment' is still the operating theatre environment, but it is a very important milestone in that it reviews all the recent epidemiological analyses regarding the effects of theatre pollution and comes to the conclusion that the risks may have been overstated. Retrospective postal

^{iv} Pfaffli P, Nikki P and Ahlman K. *B.J.A.* 1972;44:230

^v Vaisman AI. *Eksperimental'naya Khirurgiya i Anesteziologiya* 1967;3:44

surveys may well have produced biased results. Important publications were those from Scandinavia, two from Sweden and one from Finland with 'hard facts'^{vi}.

Other adverse effects of anaesthesia:

The adverse effects of anaesthesia on patients was also studied, Gillies et al. in 1979[46], the bottom line of this report of 'anaesthetic deaths' to the procurators-fiscal (equivalent to coroner) was that many of the postmortem examinations were unnecessary and that more attention should be applied to those unexpected deaths.

Halothane and nitrous oxide continued to be battered in the late 80s and early 90s, halothane [47, 48] and nitrous oxide [49]. In the latter study formiminoglutamic acid excretion was used as a measure of folate metabolism...fifteen control subjects excreted normal amounts for six days, of fifty patients who received nitrous oxide 40% had increased excretion for the first two days...ten anaesthetists had normal excretion rates.

And finally, a cross-sectional study of complications in 16,995 patients receiving an inhalational anaesthetic [50]. The overall incidence of complications was about 14%; complications were more common in the obese and the elderly. There was a correlation with isoflurane but it was thought that this might have been due to its recent introduction and the learning curve that this involves.

Other

Over an academic career of 30 years there are going to be a number of chance, opportunistic, publications and some that are of a non-clinical nature. Alastair Spence was involved in the College of Anaesthetists and teaching, and the wider world of anaesthesia.

^{vi} Ericson A and Kallen B. *Anesth. Analg.* 1979;58:302 and 1985;64:981

Hemminki K, Kyronrn P and Lindbohm ML. *J. Epidemiol. Community Health* 1985;39:141

The following references include College / teaching type publications [51-64]. Of these the following have been selected for comment.

The paper by Mowbray [58] was an interesting example of a cascade form of teaching; ten medical students were taught basic cardiopulmonary resuscitation and then they in turn went out and taught 40 secondary school pupils...all formally assessed... a very novel idea especially in the light of the present view that early intervention, resuscitation, by the public is good in the non-hospital environment.

The comparison of candidates who passed or failed the final (Part Three) examination for the F.F.A.R.C.S. (Eng.) in 1988 by David et al. was an attempt to isolate the factors that influenced pass or failure [61]. Successful candidate thought they had better departmental support, better systematic preparation and their workload was not particularly different to those who failed (47% vs. 55%). Unsuccessful candidates were more likely to have some personal upset during preparation for the exam.

And a final foray into education with a comment on the use of simulators for management of risk [65].

Some authors have a sustained interest in the history of anaesthesia; others seem to reflect on it at the end of their careers. Three publications by Alastair Spence in 1996/7 [66-68] are about ether and chloroform, the latter turning full circle to 1967 [2] where chloroform was part of a study into the renal effects of chloroform.

“The clinical evidence for delayed chloroform poisoning” [67] is a detailed analysis of the clinical data associated with case reports of adverse events (delayed chloroform poisoning, DCP) from 1847 to 1970. It is a detailed review with 69 references. In the discussion it is made clear that many of the reports do not describe hepatic failure and in those that do the link with chloroform is tenuous. At the time when chloroform was in common use hypoxia and hypercarbia were common and, together with other factors such as semi starvation and alcohol and barbiturate use, liver damage was more likely. Thorpe and Spence concluded that, in agreement with others, that chloroform had had “a rough deal” and that by at the

end of the 20th century chloroform did not fulfill the criteria to be a hepatotoxin^{vii}. It compared very favourably with halothane.

Miscellaneous:

Interspersed amongst the papers on the themes described above were a miscellaneous group of articles...not all these have been seen by the author [69-84].

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^{vii} Davison MHA, Wynne NA. in *Anaesthesia and the Liver*, 8th Edition. Boston: Little Brown, 1970 and Strunin L. *The Liver and Anaesthesia*. London: WB Saunders, 1977.

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