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Alan Aitkenhead was a research fellow in Oxford in 1978-79 and then became senior lecturer in Leicester. He remained there until 1988 when he was appointed professor of anaesthesia in Nottingham.

At least a third of his publications were from his time at Leicester and a specific interest was that of colon blood flow.

This is rather a niche topic but the rationale is the answer to the question “Can anaesthesia affect blood flow to the bowel and therefore influence the healing of anastomoses?”

Colon blood flow

The first paper (1977) highlighted by the Medline Database was “The effect of changes in arterial pCO₂ on colonic blood flow in the dog” [1] which was published in the Scottish Medical Journal. Following this was “The effects of subarachnoid spinal block on colonic blood flow in the dog” [2]. This was presented at an ARS meeting in London in October 1977. The blood flow was measured by injecting Xe-133 into the superior mesenteric artery and determining its clearance through the main marginal vein of the colon. Blood flow increased by 22% and this was associated with a marked decrease in colonic vascular resistance, 44%.

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1 J F Nunn. British Journal of Anaesthesia. 1999; 83(6): 916
2 http://www.thestar.com/news/crime/2013/05/02/dr_george_doodnaught_trial_hospital_engaged_in_coverup_prosecution_alleges.html
This was followed by a full paper on “High spinal nerve block for bowel anastomosis...” [3]; a retrospective clinical study of neuraxial block vs. general anaesthesia. There was a threefold incidence of dehiscence in the general anaesthesia group compared with the neuraxial block group. The size of the study was such that this was not statistically significant. Morphine also appeared to have an increased risk of anastomotic failure.

A further dog study ensued – “Effects of subarachnoid spinal nerve block and arterial pCO\textsubscript{2} on colon blood flow in the dog” [6]. Spinal nerve block caused a significant increase in colonic blood flow irrespective of pCO\textsubscript{2}. It was considered to be of some clinical importance.

Highlighting the ‘niche’, another paper in 1980 was published in the British Journal of Surgery [8]; the subject was certainly of surgical significance. This dog study showed that a 10% loss in blood (over 20 minutes), with no change in blood pressure and very little in heart rate, was associated with a 26% fall in cardiac output. Colonic blood flow and oxygen availability fell by a quarter; all these changes were significant. Retransfusion of shed blood resulted in a slow and incomplete return to pre-bleed status. It was advised that the “slightest degree of hypovolaemia should be avoided” and that systemic blood is inadequate indicator of need for transfusion.

The third publication on this topic was reported in ‘Cardiovascular Research’, “Colon blood flow in the dog: effects of changes in arterial carbon dioxide tension” [9]. It describes the use of 133Xenon for the determination of colonic blood flow. Hypercapnia increased the blood flow by 50% and hypocapnia a 25% fall; “there was a straight line relationship between colon blood flow and arterial pCO\textsubscript{2}.” However, hypercapnia also caused a significant increase in colon oxygen consumption. This again may be significant in anaesthetic practice.

This work covered the two major anaesthetic ‘side effects’; hypotension and hypocapnia; the latter very popular at the time as it was considered to be useful in reducing the need for analgesic supplements.

Two review articles were written in 1984 on the subject of “Anaesthesia and/or bowel surgery” [12, 13].
In 1988 there was a prospective study of “High spinal nerve block for large bowel anastomosis” [29]. Dehiscence of the anastomosis was of equal incidence in both the spinal and general anaesthesia groups but the transfusion rate in the general anaesthesia group was twice that in the spinal group.

Moving from the colon to the oesophagus: “Lower oesophageal contractility as an indicator of brain death in paralysed and mechanically ventilated patients with head injury” 1987 [24]. Oesophageal sphincter pressure had been studied as an indicator of depth of anaesthesia. All patients with no spontaneous lower oesophageal contractility were invariably diagnosed as brain dead. Non-propulsive oesophageal activity requires an intact connection between the brain and oesophagus. It was suggested that oesophageal contractility might be useful in identifying brain death.

Another review on the wide topic of “Anaesthesia and the gastro-intestinal system” [26] was published in 1988.

The final GIT related paper was in 1990, "Relationship between lower oesophageal contractility and type of surgical stimulation" [36]. The two stimuli were those of hysterectomy and those of varicose vein surgery. Spontaneous oesophageal contractions and provoked contractions were greater during hysterectomy. The use of surrogate measures of depths of anaesthesia faded when the bispectral index (BIS) and evoked brain stem potentials became mainstream.

**Awareness**

Awareness and depth of anaesthesia was a constant food for thought during the 1970s and 80s. In 1983 Aitkenhead wrote a review on awareness in the Annals of the Royal College of Surgeons of England [10]. It was a general overview. What should a patient be told after experiencing awareness and, preoperatively, about the risk? He addressed this problem in an editorial in 1990 [34]. It was a comprehensive advisory. The anaesthetist should always be informed of the possibility of awareness; lack of communication would be overcome if anaesthetists talked to their patients after recovery and the patient’s account should be believed. Awareness can occur without fault (this should be explained to the patient) and account should be recorded in the hospital notes, alerting future anaesthetists to the
problem. If an error had occurred it was Aitkenhead’s view the error should be admitted “…as this may serve to reduce the patient’s fears about awareness during subsequent operations” [the author concurs with this]. He believed that awareness should be discussed at the pre-operative visit especially for the patient at high risk, such as before Caesarean section.

A year later an audit involving 1000 patients was reported [40]. The patients were interviewed between 20 and 36 hours after surgery. Using a standard set of questions the incidence of recall and dreams were 0.2% and 0.9% respectively. This was much lower than similar studies.

An editorial in 1996, “Awareness during anaesthesia: when is an anaesthetic not an anaesthetic?” appeared in the Canadian Journal of Anaesthesia [61] and finally, in 2014, the “Personal and medicolegal implications of awareness” [81], an editorial.

Intensive Care
The earliest foray into this area was with Ledingham et al. at the Western Infirmary, Glasgow, and concerned the “Movement of the critically ill within hospital” [4]. It described a “mobile intensive care unit (MICU)”; a patient trolley with appropriate attachments for all the usual monitors and pumps.

The next was with Willis and Barnes, Oxfordshire Area Health Authority (Teaching) Ambulance Service staffing officer and senior technician at the John Radcliffe Hospital respectively. It describes the design of a patient trolley to facilitate the transfer of intensive care patients between hospitals with minimal disruption [7].

An editorial with Graham Smith was published in 1986 on “Aspects of intensive care” [22]. It is an introduction to the Postgraduate Issue devoted to advances in intensive care.

In 1989 there was paper, “Comparison of propofol and midazolam for sedation in critically ill patients” [32] in the Lancet, followed by a letter about propofol and intensive care [30]. This was a short letter about the quick time of recovery from propofol compared with midazolam and was answering a comment about the use and

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Standing, V. And Leach, F. Lancet 1989;2:975
expense of using propofol for long term sedation. There was also a review article in the British Journal of Anaesthesia [31] on the same subject, “Analgesia and sedation in intensive care” [30].

Four years later Aitkenhead was a member of the team developing “International standards for safety in the intensive care unit. Developed by the International Task Force on Safety in the Intensive Care Unit” [45]. This was endorsed by the World Federation of Societies of Intensive and Critical Care Medicine. It covered staffing, design, services, equipment, monitoring, records, drugs and infection control.

Respiration/Simulation

“Quantitative effects of respired helium and oxygen mixtures on gas flow using conventional oxygen masks” [11]. A mixture of 79% helium/21% oxygen was administered via a conventional, disposable face mask to a resuscitation dummy. The greatest flow was at tracheal concentration of 40% helium which, with fresh gas flows in excess of eight litres per minute, all masks delivered.

1994: “Effect of three different surgical prone positions on lung volumes in healthy volunteers” [51]. The knee-chest position was compared with the prone position using an Eschmann frame and a standard prone position. The study was done in awake volunteers – it was shown that the knee-chest position caused the least respiratory restriction.

1998: “A physiology simulator: validation of its respiratory components and its ability to predict the patient’s response to changes in mechanical ventilation” [65]. The computer simulator was supplied with a wide variety of parameters from patients receiving intensive care. Values calculated by the simulator for PaO$_2$, SaO$_2$, PvO$_2$, SvO$_2$, PaCO$_2$, PvCO$_2$ and arterial pH, when compared with measured values, were accurate. Subsequent changes in the patient were simulated in the simulator and the 95% limits of agreement were good. It was suggested that it could be used to predict the effects of change in ventilation in stable intensive care patients.
1999: “Estimation of alveolar deadspace fraction using arterial and end-tidal CO$_2$: a factor analysis using a physiological simulation” [67]. Alveolar dead space can be used to monitor pulmonary disease and predict the ability to wean from mechanical ventilation. As in the previous study the simulator used was the Nottingham Physiology Simulator (NPS). Read the original paper; $\text{VD}_{\text{alv}}/\text{VT}_{\text{alv}} = 1.135 \times (\text{Pa-E'CO}_2)/\text{PaCO}_2-0.005$ during normal physiological conditions. The equation could be used clinically to determine the alveolar dead space.

In 2000 there were two papers published in the same issue of Anesthesia and Analgesia, “Investigating hypoxemia during apnea: validation of a set of physiological models” [69] was the first. It was to further validate the NPS. They reproduced the methodology of previous clinical studies. The results were within 2% in most cases and within 13% of all cases.

The second was “Factors determining the onset and course of hypoxemia during apnea: an investigation using physiological modelling” [68]. Using the NPS, hypoxemia caused by apnoea after pulmonary denitrogenation was studied. A wide range of respiratory parameters were altered to examine their influence on the onset of hypoxaemia. Airway obstruction was bad - reducing the time to 50% oxyhemoglobin saturation to 8 minutes (vs. 11 minutes). One hundred percent oxygen was good; prolonging the time to 66 minutes [that’s why it was used in the testing process for brain death]. This did highlight the value of good simulation as this study could not be performed in volunteers or patients.

2002: “Effect of videotape feedback on anaesthetists’ performance while managing simulated anaesthetic crises: a multicentre study” [71]. “Those trainees exposed to videotape feedback had a shorter median ‘time to solve’ and a smaller decrease in chart error when compared to those not exposed to video feedback”. This type of study is intrinsically difficult and these results, as in other similar studies, were not statistically significant.
2003: “Estimating alveolar dead space from the arterial to end-tidal CO₂ gradient: a modeling analysis” [73]. This seems to be very similar to the 1999 paper in Anaesthesia and Intensive Care.

2003: “Validation of an original mathematical model of CO₂ elimination and dead space ventilation” [74]. Data from previous clinical investigations were used to validate a new mathematical model. The first used low-dead space, the second examined dead space during anaesthesia. This validation against previously published clinical data was such that it could be used in theoretical investigations where data is not available.

A subject not much studied is that of the cough. Aitkenhead did author a few papers on the topiciv. The first three were all in 1994 and obviously the result of having bought a tussometer.

“The tussometer: accuracy and reproducibility” [54]. This was a new technique for measuring laryngeal function. It analysed the airflow waveform during a maximum effort voluntary cough. Cough peak flow rate and peak velocity time “...were found to be reproducible; the within-subject variability for CPFR was found to be 23.9% and for PVT 9%.” There was no inter-observer variation.

“Effect of topical anaesthesia on the motor performance of vocal cords as assessed by tussometry” [52]. The measured variables remained unchanged and it was concluded that topical anaesthesia did not impair the motor performance of the vocal cords.

“Relationship between expired lung volume, peak flow rate and peak velocity time during a voluntary cough manoeuvre” [53]. Tussometry was used during voluntary cough manoeuvres at a variety of lung volumes. Peak velocity time varied with cough peak flow rate which had a direct relationship to expired lung volume. These

iv The subject is of interest to the author as, coincidentally, whilst working in Nottingham he had a project based on the mechanics of the cough. As an aside, the one really pertinent publication happened to be in Russian and the author spent a few interesting hours with a Russian/English dictionary translating it.
relationships should be taken into account when interpreting the results of tussometry.

The final paper using the tussometer was in 1995: “Relationship of peak flow rate and peak velocity time during voluntary coughing” [59]. This was a study of the cough dynamics of men and women. “There was a positive correlation between peak velocity time and cough peak flow rate in both...” Height and sex were determinants of the peak flow rate/peak velocity time relationship. Anatomical differences “may have implications” when interpreting tussometry.

Pharmacokinetics
1984: “Pharmacokinetics of single-dose i.v. morphine in normal volunteers and patients with end-stage renal failure” [14]. There was considerable variation but plasma concentrations in the patients with renal failure were higher for the first quarter of an hour. Pharmacokinetic parameters were significantly different between the two groups apart from the terminal elimination half-life and total body clearance, which were similar.

1984: “Pharmacokinetics and analgesic effect of slow-release oral morphine sulphate in volunteers” [16]. The mean peak plasma concentration occurred at a mean time of 142.5 minutes and analgesia was maximum about 40 minutes later.

1988: “The pharmacokinetics of oral and intravenous nalbuphine in healthy volunteers” [27]. A three compartment model was used to derive pharmacokinetic parameters.


Consent
1999: “Anaesthetists need consent, but not written consent” [66] A letter. This was in response to an article by R Dobson which said that
"...consent from patients specifically for a general anaesthetic is not needed". Aitkenhead was making the point that this was an inaccurate reflection of what the AAGBI guidelines advised. A signed consent did not guarantee that the patient understood the risks and that it was more important to accurately record what was discussed about the procedure and to what the patient had agreed.

2006: "Informing and consenting for anaesthesia" [78]. The paternalistic actions of doctors were no longer acceptable and this had been highlighted in the courts. Aitkenhead made it clear that anaesthetists should be aware of current requirements regarding patients' need for information and obtaining appropriate consent for anaesthetic procedures.

Other miscellaneous publications of interest
1986: "Does anaesthetics research need training?"[21] The first line reads "Anaesthetics research in the UK appears to be thriving." This was because of the increase in publications and submissions for presentation at the Anaesthetic Research Society meetings. However adverse comments had been made on the quality and the need for education in scientific methodology. It was said that it was important to recruit the academic teachers of the future, to enhance the finances (both personal and for research), and for the consideration of training of non-clinical scientists.

1987: "Clinical investigation - why we must keep control"[23]. The ‘control’ referred to here is the control group of patients in comparative studies and highlights some of the problems of historic ‘controls’ and placebos and such like.

1991: "Comparison of contemporaneous and retrospective assessment of postoperative pain using the visual analogue scale"[39] Although there were significant correlations between contemporaneous scores and the

\[\text{\textsuperscript{v}}\] At about this time the annual capital available to the academic department in Nottingham was of the order of £3000. No wonder it was hard to recruit a new professor.
retrospective scores there was a wide scatter of results. The two sets of measurements were not interchangeable.

1993: “The effect of the anaesthetist’s attire on patient attitudes. The influence of dress on patient perception of the anaesthetist’s prestige”[48]. This was the difference between formal or casual wear. In brief the mode of dress did not make a difference. The preferred preference was for name tags, white coats and short hair.

1994: “The pattern of litigation against anaesthetists”[50]. This appeared in a Postgraduate Educational issue following a Symposium on Mishap or Negligence. The symposium actually occurred about eighteen months earlier. The ‘pattern’ of litigation described covers all the usual suspects – airway management, hypoxaemia, cerebral damage, drug errors, anaesthetist’s failures, etc.

1997: “Anaesthetic disasters: handling the aftermath”[62]. This is a “Special Communication” on the management of the situation following a major adverse event – severe injury or death of a patient.

Books

Clinical Anesthesia, 1996, by J. S. Gravenstein and A. R. Aitkenhead

Quality and Risk Management in Anaesthesia (Bailliere’s Clinical Anaesthesiology), 1996, by A. R. Aitkenhead

Pharmacology of the Critically Ill, 2001, by Gilbert Park, Maire Shelly, Ronald M. Jones and Alan R. Aitkenhead

Fundamentals of Anaesthesia and Acute Medicine, 2001, by Ronald M. Jones and A. R. Aitkenhead
References


