LETTERS TO THE EDITOR

Preference is given to letters commenting on contributions published recently in the *JRSM*. They should not exceed 300 words and should be typed double spaced

Air pollution and death

Dr Maynard's review article (June 2000 *JRSM*, pp. 288–291) was both interesting and relevant. I was in London during the 1952 smog and can remember trying to feel my way around Clapham Common during what seemed to be perpetual dusk. The accepted explanation for the 4000 or so excess deaths in the 1952 smog was that they were caused by air pollutants compromising lung function. My next two and a half years were spent in Sheffield in junior hospital posts. In those days, between October and March, Sheffield seemed to be perpetually smog-ridden with Hadfields and Steel, Peach and Tozer incessantly spewing fumes into the air. Every winter there was a constant flow of 'blue bloaters' with cor pulmonale requiring admission to the Royal Infirmary and City General Hospital. Many died 'despite' supplemental oxygen, bronchodilators and excellent nursing care. At about the same time, Dr Charles Davies who was a reader in Professor Charles Stuart-Harris's Department of Medicine was measuring the cerebrospinal fluid (CSF) pressure in such individuals on and off additional oxygen. He observed that a substantial number of those with chronic bronchitis and emphysema became drowsy and hypoventilated, while their CSF pressure went way up and some developed papilloedema. It was, however, left to Moran Campbell and Keith Westlake to demonstrate that administration of excess oxygen could cause severe hypoventilation and apnoea. In retrospect one wonders how many deaths in the 1952 smog were iatrogenic, precipitated by the administration of excess oxygen. Unfortunately, many recent medical graduates seem unaware that they need to be careful when prescribing oxygen in chronic obstructive respiratory failure lest they precipitate hypoventilation and severe carbon dioxide retention. It would seem that Moran Campbell's superb 1967 Amberson Lecture to the American Thoracic Society is forgotten or at least passé, no doubt partly because we see far fewer blue bloaters these days.

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Roses red and white

Describing the creation of the coat of arms for our new College, Bernard Valman records that some Lancastrians objected to the inclusion of a white rose (July 2000 *JRSM*, p. 389) and explains that the white rose had been chosen 'because it represented the City of York . . . as well as the rose of England'. This perpetuates the original mistake. The rose of England is red not white, as a glance at an England rugby player's jersey or the emblem of the Labour Party will reveal. The white rose is the rose of the House of York . . . who, yes, did lose the Battle of the Roses. Alongside the daffodil, the thistle and the shamrock should be the red rose.

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The emperor with the shaking head

On the matter of Dr Rice's article (April 2000 *JRSM*, pp. 198–202), Levy and I published an article fifteen years ago[1] which provides some additional references, including a 1920 American PhD thesis on Claudius.

Like Dr Pearce (June 2000 *JRSM*, p. 335–336) I suspect that he had cerebral palsy. In daily rehabilitation practice we know that even patients with 'stable non-deteriorating' disabilities can worsen slowly over the years—for example, those with spinal cord injury, amputees, post-polio patients. This is true of patients with cerebral palsy. Claudius experienced bouts of 'meteoria'—the petit mal seizures or absences typical of brain damage. He was described as drooling, which is not a feature of dystonia (Dr Rice's proposal).

For me, as a rehabilitation physician, Claudius' historic achievement was to decree freedom for wounded or disabled soldier-slaves or gladiators, who were treated at the Valetudinaria on the small island of St Bartholomew in the River Tiber.

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Drawings of changes in scrofulous glands by Frederick Treves

Sir Frederick Treves's forgotten portfolio of drawings, which Professor Om P Sharma has generously donated to the RSM Library (July 2000 *JRSM*, p. 340), adds to understanding of an early stage of his remarkable career. For a few months after he qualified in 1874 Frederick Treves held junior posts at the London Hospital; Sir Jonathan Hutchinson was his mentor and became his lifelong hero. There followed a period of three years when, probably largely because of financial constraints, the direction of his career was uncertain. His eldest brother Dr W Knight Treves, who was established in practice in Margate and who had been supporting him financially while he was a medical student, encouraged him to take the post of resident assistant at the Royal Sea Bathing Infirmary in Margate (sometimes then referred to as the National

Hospital for Scrofula), following which he entered general practice for two years in Wirksworth in Derbyshire. The main evidence for his continuing surgical aspirations during these years was his success in becoming FRCS while in general practice. Though his first book, *Scrofula and its Gland Diseases*, was not published until 1882, it was based on the experience he gained in Margate some six years earlier. But the scrupulously accurate pencil drawings in this album were probably made by Treves in 1880 or 1881, by which time he had returned to the London Hospital and was surgical registrar. In the preface he wrote for the London edition of *Scrofula and its Gland Diseases* Treves refers to 'drawings expressly prepared by me for my article on scrofula in the forthcoming edition of Holmes' *System of Surgery*. By the kind liberality of the publishers . . . I am enabled in the present book to anticipate the publication of those plates'. Thus the London edition was published with lithographic plates made from drawings in this album, whereas no illustrations are included in the New York edition. Could the album have been sent to New York to provide illustrations for the American edition and subsequently remained in the USA, even though no drawings were used? Whatever the truth of the album's travels, its presentation by Professor Sharma as a gift to the RSM Library gives it an ideal home, as well as offering opportunities for further study.

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Continuing legacy of the Third Reich

I should like to draw the attention of your readers to some important issues which I was made aware of when attending the sixth European Conference of the World Organization of General Practice (WONCA) in Vienna from 2 to 6 July. The opening session dealt with 'The Continuing Legacy of Medicine of the Third Reich' and 'Lessons of the Holocaust'. Issues such as apartheid, the death penalty in the USA, and torture were also discussed, and there was unanimous agreement, following my suggestion, that a working group should be set up to study the ethical issues of medical involvement in these horrors.

Perhaps of even greater concern to your readers will be the following facts. There is still continued use of the Pernkopf Atlas by Austrian medical students. The illustrations in this atlas are thought to be based on concentration camp victims, with the original artists using SS symbols in their signatures. There is also the unresolved case of Dr Heinrich Gross, an Austrian doctor accused of killing children in a Nazi euthanasia centre. Many of the brains removed from these children are still standing in an Austrian anatomical museum and cannot be buried until the case of Dr Gross is finally concluded. He recently claimed inability to plead at a court case and subsequently walked free. He then gave a perfectly coherent interview to the media, delighting in the case's outcome.

Finally, it is extremely sad that an Austrian, Professor Wolfgang Neugebauer, who is looking into many of these unresolved 'medical issues', has had a case of defamation of character brought against him by Dr Jorg Haider, supported by the Austrian minister of justice.

I feel that your readers should be aware of some of these issues, on which I would be happy to supply further details.

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Experimental allergic encephalomyelitis and multiple sclerosis

Dr J N Blau makes some very good points in his review of *Books of the Body: Anatomical Ritual and Renaissance Learning* (August 2000 *JRSM*, pp. 446–447). The inertia in accepting new ideas is not confined to medicine. Before the notion of chemical elements could be accepted, the previous ideas of Aristotle (that the earth was composed of four elements) had to be discarded; this took many hundreds of years. New ideas are never very welcome. When Isaac Newton was professor of mathematics at Cambridge, no students attended his lectures.

Dr Blau considers that the production of experimental allergic encephalomyelitis (EAE) in monkeys, and the classification of multiple sclerosis as autoimmune, did not help patients and may even have hindered progress. This is because of gaps in our knowledge of these conditions. American researchers in neuropathology have told me that EAE owes its discovery to the fact that in 1935 many monkeys in American laboratories had tuberculosis. The original experimental results have never been successfully reproduced; since then, Freund's complete adjuvant or a similar agent has been used at some stage in the experiments.

One source of error or misunderstanding is the fact that experimental pathologists and clinicians use the word relapse to mean different things. The relapses in multiple sclerosis occur about once every 15 months; and, since this is an average, the intervals will often be much longer. By contrast, in animals such as rats, with whole myelin used as the antigen, three or four relapses may occur in 2–3 months. This enormous difference indicates that the two types of relapse are completely different phenomena. When a relapse involves the human eye, the condition is called retrobulbar neuritis. I have never seen this described in animals, even monkeys. In humans it is associated with marked swelling of the optic nerve and

pain on eye movement, almost certainly due to stretching of the meningeal cuff around the optic nerve; visual impairment is of very limited duration, with a central scotoma tending to recover in 3–4 weeks. Shahgaldi and I have proposed a new explanation for the relapses in the human disease, based on computer-made models of the myelin sheath—namely, that they result from failure of the mechanism which controls pressure in the myelin sheath[1]. When the pressure becomes too high the myelin sheath bursts, releasing the proteins responsible for the relapses. We also describe a method for measuring this pressure in the sciatic nerves of pigs.

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Cerebral venous thrombosis: the first reported case of adolescent stroke?

The symposium on cerebral venous thrombosis (May 2000 *JRSM*, pp. 230–243) was an erudite summary of a difficult clinical state. Readers may be unaware that the first clear description of this condition (two cases together with post-mortem findings) came from Thomas Willis (1621–1675). The first of these is probably also the earliest record of an adolescent stroke; the second case was due to a cerebral abscess, and I shall not mention this further.

Willis's case 1 was originally recorded in Latin in his *De Anima Brutorum*[1]. Regarding the age of the 'young scholar' I should mention that, according to contemporary sources[2], boys attended Oxford and Cambridge from their early teens.

> '. . . fatal or incurable headaches sometimes arise from a phlegmon or abscess. Once a young scholar complained for two weeks of being incessantly afflicted by the most severe headache. This was eventually aggravated by a fever, and succeeded by insomnia, convulsive movements and delirious talk. At that time a doctor was called in and industriously applied phlebotomy, enemas, plasters, emetics and blistering and also internal remedies to divert the flow of blood and humours from the head. But he could make no progress to prevent the quick onset of death. When the skull was opened the vessels leading towards the meninges were full of blood and greatly distended as if all the arterial blood had flowed together there. When the sinuses were dissected and opened the blood burst out in abundance to the weight of several ounces above half a pint. In addition the membranes themselves, affected throughout by an inflamed swelling, appeared discoloured. When these coverings were removed all the bends of the brain and its ventricles were full of water and its substance, inasmuch as it was excessively watered was soaked and insufficiently firm.'

Hughes[3] commented on this case that the history and post-mortem findings were in keeping with a venous infarction of the brain following a septic thrombosis of the vein of Galen or the straight sinus. Willis's own interpretation was that 'without doubt in this case the headaches and subsequent delirium were caused by the incursion of the effervescent blood into the meninges and its accumulation there which caused a phlegmon'.

The aetiology and epidemiology of childhood and adolescent stroke are completely different from those in adult populations[4] and the condition offers special challenges for the paediatrician and paediatric neurologist[5]. Otitis media as a cause of cerebral venous thrombosis, with 'the infection being carried by the blood vessels'[6], was described over one hundred years ago and was recognized at that time as a cause of childhood/adolescent stroke. The debate as to the actual importance of venous thrombosis as a cause of childhood stroke is equally longstanding. In 1887 Abercrombie[8] was disputing Gowers's opinion that 'thrombosis in arteries is a very rare lesion in childhood, far more rare than combined thrombosis in sinuses and veins', feeling instead that arterial obstruction 'will hold good in the great majority in the other cases'. Time has vindicated Abercrombie's judgment for the present day, but of course it is impossible to obtain a clear answer for his own time.

Until the antibiotic era, otitis media was the most frequent underlying cause of cerebral venous thrombosis in older children. Complications of acute otitis media are now very uncommon and acute mastoiditis is rare in the developed world[8]. The decreased frequency of acute mastoiditis has been ascribed to antibiotics, but improvements in social conditions, in particular housing and nutrition, are key elements whose contributions remain unclear. In the USA there are concerns regarding possible increases in recurrent otitis media[9] and mastoiditis[10]; however, for the Birmingham Children's Hospital (serving a population of about one million children within the West Midlands) the number of cases of mastoiditis remained unchanged between 1993 and 1998 at roughly 6 per annum.

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Plague, rats and the Bible

Dr J P Griffin (August 2000 *JRSM*, p.449) alleges that the plague of the Philistines was bubonic plague, and 'an association with rats established'. This is almost certainly erroneous[1]. The black rat, the vector (via its fleas) of the great European bubonic plague outbreaks, reached the Near East from India some time before the first century AD[2]. But the silence of all ancient authors, including Aristotle, makes it unlikely this happened until late antiquity. I have suggested that the black rat arrived from India in the second or first century BC, when recognition of the monsoon led to massive sea trade between India and the Roman Empire[3].

Rat-borne diseases are therefore unlikely to have occurred in Old Testament times. The plague of the Philistines was examined in great detail by Shrewsbury[4]. In accord with Josephus's ancient interpretation (*Antiquities* 6.1), he showed that both the circumstances of the epidemic and the nature of the symptoms perfectly fitted bacillary dysentery, which can lead to piles—the 'emerods' (haemorrhoids) of the Authorized Version, the best translation of the Hebrew word *opalim*. The biblical concordance leads one to *Psalms* 78.66, where the Lord 'smote his enemies in the hinder parts'. The 'mice' that also afflicted the Philistines were not vectors but crop pests; they were 'mice that mar the land' (1 *Samuel* 6.5). They may have prepared the way for the epidemic by producing food shortage[3].

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How long is full-time?

The editorial 'Goodbye to presenteeism' (June 2000 *JRSM*, pp. 281–282), which argues for an end to the work culture that fosters long hours, left me with a feeling of unease. Perhaps in this new century 'as much off-duty as possible' will be the major aim in life. I was wondering how to answer the article without being labelled a dinosaur. Fortunately Dame Betty Boothroyd did this for me when she told the leisure-seeking young generation of MPs that their parliamentary responsibilities took priority; for parliamentary please read medical.

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Albumin revisited

In his Arthur Hall Memorial Lecture (August 2000 *JRSM*, pp. 402–407) John Swales uses the 'albumin controversy' of 1998 as an example of conflict between two cultures, experimental medicine and the modern extrapolation of the 'numerical method' to evidence-based medicine. Dredging material from the dustbin of clinical investigation, I recall that in the early years of the war it was shown that plasma protein (in various forms) was of benefit in burns shock[1], but not in haemorrhagic shock[2]. These are both causes of 'critical illness' and there are of course many others. But, to keep things simple, it is clear that in a group containing only patients with these two types of shock, the 'benefit' or 'harm' of giving albumin would depend on the proportion of each type of shock within the group; nor would the question be resolved by having ten such groups instead of one.

There are of course countless successes to attest the value of the numerical method and its descendants, appropriately applied. But the first touchstone of appropriateness must be clinical homogeneity of the study group. As a determinant for inclusion, 'critical illness' is worse than useless—whence the grumbling persistence of the controversy.

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Heparin for colorectal cancer

In their commendable review of chemotherapy for colorectal cancer (August 2000 *JRSM*, pp. 416–419) Dr Nicum and colleagues refer to the thrombotic complications of certain regimens, both in the hepatic artery and in association with catheter lines, along with their toxicity and high cost. However, they do not mention an inexpensive chemotherapeutic agent with low toxicity that also reduces venous thromboembolic complications. There is growing evidence that heparin[1], and low-molecular-weight heparins in particular[2], should be regarded as having antitumour properties. A prospective trial[3] of chemotherapy with and without intravenous heparin in patients undergoing colorectal cancer surgery has shown a trend to increased survival in the heparin group. Three retrospective analyses[4–6] also collectively point to lower mortality in colon cancer patients receiving subcutaneous heparin perioperatively, not explainable by differences in venous thromboembolism. In view of the cost and complications of cytotoxic chemotherapy, which offers only a 5% survival advantage, confined to patients with Dukes' C cancer, more attention should be given to the role of heparin in this setting.

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Provision for parish paupers: able-bodied or sick?

The members of the local Board of Guardians conveyed their sense of authority and projected their confidence in the system they administered under the Poor Law, when they chose the design for a new workhouse in the parish of St Leonard in Shoreditch. But the building was completed only a short time before the Metropolitan Poor Act of 1867 led to the creation of hospitals for the indigent sick of London, under the control of the Metropolitan Asylums Board rather than local Boards of Guardians. St Leonard's Workhouse was incorporated into an infirmary which in due course became St Leonard's Hospital, Hackney.

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