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

Insulin coma therapy for schizophrenia

Dr Jones (March 2000 *JRSM*, pp. 147–149) depicts deep insulin coma therapy (DICT), which was an approved internationally practised treatment for schizophrenia from 1933 to about 1960, as a medical craze, a sudden enthusiasm without scientific basis enduring for 25 years and then collapsing. This does no justice to the facts, ignores the thinking of the time, and fails to offer what DICT can teach us about the validation of therapy.

In 1933 schizophrenia was an illness for which there was no treatment—barbiturates and psychoanalysis among many others had been shortly tried and failed—and it often became chronic and permanently disabling. Insulin, available since 1923 for the control of blood sugar in diabetes, had been tried empirically in many medical conditions—in subcoma doses to help body weight gain, as an anxiolytic, in delirium tremens, and by Sakel in suppressing distressing autonomic symptoms during morphine detoxification. For schizophrenic inpatients Sakel used it in a threefold novel way[1], deliberately inducing hypoglycaemic coma, holding it 30 minutes or so and then reviving the patient with oral and intravenous glucose. This coma was repeated almost every morning for six weeks or more. He observed and spoke with the patient both in the three-hour run-up to coma and at the revival later, experienced by some patients as death and rebirth.

The outcome was often remission of symptoms. Of 94 first admissions to the Vienna clinic (already notable for the success of malaria for general paralysis of the insane), pre-Sakel (1931–1933) only 19 (20%) had left hospital and 75 stayed more than three years. Of 104 similar cases treated by Sakel (1933–1935) 71 were discharged (68%) and only 33 remained[2]. Beforehand patients were typically self-absorbed, preoccupied with strange inflexible thoughts, largely unreachable. But in the run to coma, and just after revival, they often showed flashes of lucidity, responding briefly to Sakel with sudden sense and feeling, encouraging him to persist. These flashes impressed observers[3], who came to scoff but left to spread its use, complex, difficult to repeat and even dangerous though it was. The dose of insulin to induce coma varied considerably from individual to individual, and even from day to day in the same person, requiring frequent readjustment. Coma had no sharp point and was hard to gauge in depth. Revival was sometimes delayed, coma prolonged in spite of glucose, and there were deaths, all a constant anxiety to the therapist. After revival, suddenly a few hours later coma might recur spontaneously, but respond to further glucose. The technique required much doctor time, special nurses, a special ward where patients stayed together as an elite group, all matters difficult and costly for medically impoverished mental hospitals.

Depth of coma and electroencephalographic changes did not correlate with measurements of hypoglycaemia, which was assumed to be somehow therapeutic though there was no scientific theory of how it acted on the (unknown) brain pathology of schizophrenia, an illness recognizable only from history and symptoms. There was much research to improve the technique and on the endocrine and autonomic and neurological effects of insulin in normal individuals (compare phenomena in insulinomas[4,5]). During the 1939–1945 war practice and research were suspended for lack of facilities, but resumed in 1946.

There was widespread agreement that DICT was only for schizophrenia and only helpful in those ill for less than three years, and despite the arrival of electroconvulsive therapy it was still the only hope for many. The post-war rise to dominance of American psychiatry by psychoanalysis, with its hostility to physical treatments and change in recognition of schizophrenia, may have fuelled the doubts about the effectiveness of DICT which began to arise. Results seemed less good than formerly, but also DICT had changed in an unremarked way. Interest in lucid flashes had been lost, the doctor was no longer in very personal contact with the patient; the emphasis was on the insulin dosage and group nursing.

Spontaneous remissions had been thought rare, but their frequency might differ between centres as part of the unknown variations between different selections of inpatients at different times and places. In the 1950s the blind controlled clinical trial emerged as a way to neutralize these effects on measured outcome. Ackner *et al.*[6] reported the only such trial of DICT in 1957. Successively, 50 acute schizophrenic patients were assigned randomly to insulin with glucose revival or barbiturate with amphetamine revival, 35–40 comas, and the same nursing and group care. Results: 9/25 on DICT and 10/25 on barbiturate, full remission (18/25 on insulin discharged)—small figures, which show insulin no better than barbiturate used in this way (i.e. non-specific for schizophrenia). But since barbiturate has central nervous actions, it is not a true placebo. Perhaps used as a coma agent both it and insulin act non-specifically to modify the mental state. Chlorpromazine (1953) and reserpine (1954) are not specific, but were found to be safer, cheaper, easier and (partly) effective treatments in schizophrenia. It is no surprise that at this point DICT was simply abandoned on practical grounds, neither proved nor disproved. Important questions remain. Does insulin in high dose have a direct effect on some

neurons and the balance of brain functions (some neurotropic factors are also 'insulin-like')? Does psychological handling influence outcome of treatment, and if so, how?

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Surgical correction of refractive error

We reply to two letters in the May 2000 *JRSM* (pp. 274–275) that respond to our review article in the March issue (pp. 118–123). Mr Jory, of the London Centre for Refractive Surgery, cites Schlote *et al.* (1997), and notes that, in this isolated report, the majority of eyes (67%) treated by photorefractive keratectomy (PRK) failed the night-time driving guidelines set by the German Ophthalmologic Society (DOG). However, he neglects to point out that these were early PRK cases with a small diameter zone and that the German vision standards are particularly stringent; 5% of untreated eyes wearing soft contact lenses and 18% of 'normal' eyes wearing spectacles likewise failed this DOG standard. Furthermore, these tests have not been applied to eyes that have undergone radial keratotomy (RK or corneal microsurgery), so it would be wrong to assume that RK compares favourably. Indeed, when Ghaith *et al.*[1] assessed radial keratotomy and PRK with four different devices to assess contrast sensitivity and glare disability, they noted that (1) RK and PRK produced largely similar reductions in contrast sensitivity and glare disability, (2) the relative effects were highly time-after-surgery dependent (becoming less marked with time) and (3) these measurements did not accurately reflect patients' subjective assessments of daily visual performance.

Mr Jory refers to the hazards of Sato's surgical technique. This is more akin to contemporary radial keratotomy than to excimer laser surgery. Additionally, it was applied to the non-regenerating endothelial (posterior) layer of the cornea rather than the anterior corneal stroma treated by excimer laser techniques. Extensive investigation suggests that after excimer laser surgery there is no significant effect on the endothelium[2]. Although individuals with complications following radial keratotomy, excimer laser, or other refractive procedures may ultimately consider legal action[3], we believe the major class actions in the United States referred to by Mr Jory have been launched by laser companies claiming patient infringement rather than by aggrieved patients.

We did not dismiss radial keratotomy (corneal microsurgery), and acknowledged that it has a role in the management of low to moderate myopia—in our opinion up to about −4.00 D. Although a major review of radial keratotomy concludes that for patients with less than −6.00 D of myopia the percentages of eyes achieving 6/12 unaided vision following RK and PRK were very similar[4], several excimer laser studies report results yet to be routinely achieved by RK, with up to 100% of eyes under −6.00 D achieving 6/6 (20/20) unaided[5]. Nevertheless, Rowsey and Morley judge that, 'because similar results can be obtained more economically with RK than PRK, RK will always retain a place'[4].

With regard to trauma after corneal microsurgery or RK, reports continue to document post-RK eyes with globe rupture following trauma in daily living, sports and assault[6]. Specifically, the only corneal refractive procedure which has been scientifically shown to reduce the amount of energy required to rupture the globe is corneal microsurgery or RK. Although mini-RK uses shorter length incisions, the energy required to rupture the globe is still lessened by around 50%[7]. Steinemann *et al.* warn, 'any patient considering radial keratotomy should be counselled about the risk of greater ocular damage in trauma'[7].

In reply to Professor Choyce's comment on the use of polysulfone inlays, he himself has stated, 'unfenestrated polysulfone appears to be associated with colour change and varying degree of stromal opacity in eyes evaluated 12 years post-operatively and thus cannot be considered clinically acceptable'[8]. With regard to 'permanently dry eye' certainly both PRK and LASIK can cause short-term dry eye symptoms during regeneration of corneal nerve endings. However, we are unaware of any convincing peer-reviewed scientific evidence that LASIK or PRK in themselves can cause permanently dry eye. Of course, severe dry eye is a relative contraindication to refractive surgery.

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Placebo

It is some time since I studied Greek, so I may well be wrong about this, but it seems to me that the phrase quoted by Dr Jacobs (April 2000 *JRSM*, pp. 213–214) means 'I will be pleased in the company of the Lord in the land of the living', rather than 'I will please the Lord in the land of the living'. I don't see what any of this has to do with the use of the term 'placebo' but it does perhaps suggest that the Septuagint was Greek to St Jerome.

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Female beauty

Nigel Drury's erudite dissertation 'Beauty is skin deep' (February 2000 *JRSM*, pp. 89–92) posits that beauty is not in the eye of the beholder but, rather, a biological property with survival value. I challenge the latter part of his supposition. First, he displays a European tunnel vision, over and above his eclectic classicism and even if he chooses to demean it by reducing it to an acronym, WHR (waist to hip ratio). Secondly, what is considered to be beautiful and, according to Drury, attractive in terms of reproductive strategy, differs from place to place. The Inuit, the Maori, the Tibetans and the Bushmen will not consider Drury's chosen lady beautiful, neither will most tribes of Africa. Some like fat bellies and spindly arms, others like stubby noses and slim hips, others again adore pendulous breasts.

Now it could be that in each and every instance there is an evolutionary advantage inherent in these various attributes. Indeed, small eyes may be an advantage in windy deserts, slim long legs in mountainous country, big feet where there is much mud and so forth. These differences in shapes and preferences may deserve intense research. Trouble is, there appears to be change in preference over time even in our own cultural heritage. The *Song of Solomon* is a case to study. Apart from the fact that the comely lady is compared 'to a company of horses', one of her attractive features, her belly, 'is like a heap of wheat'. This does not sound like a favourable WHR. Moreover, in Michelangelo's *Temptation and Expulsion from Eden*, illustrating the soothing proportions of the golden cut to the readership, we see depicted an Eve who, today, would not be considered beautiful, and not just because of her poor WHR. Indeed this lady, if she lived today, would probably make her living as a wrestler in late-night TV shows. Yet Adam, fortunately, has not been repulsed by her proportions. . .

Adam had no choice; one suspects the selfish gene drove him until he overcame his aesthetic reservations. This is precisely what happened to many men throughout the millennia and continues to happen to our contemporary fellow men: they have no choice in the matter. Perhaps extreme cases, such as poor Jacob's, who thought he lay with Rachel only to discover in the morning that he had lain with Leah, are rare, but it seems evolution has cursed the male *Homo* with the urge to reproduce without much regard to ideal proportions.

Beauty may well be real, as Thomas the Aquinate has taught. Yet here on earth reproduction is delinked from beauty. On the other hand, it could be that evolution uses a different trick altogether. The platonic idea of beauty may travel in the DNA, but, as in reality beauty is rare, hormones influence perception. If testosterone warps the vision and makes the WHR look better than it is, well, that would mean that beauty is after all in the eye of the beholder.

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Pain management in herpes zoster ophthalmicus

Dr Goon and colleagues report use of PCR to demonstrate the presence of varicella-zoster virus DNA in nasal and conjunctival samples from a patient with ophthalmic zoster *sine herpete* (April 2000 *JRSM*, p. 191). This technique will be a useful tool when clinical findings are insufficient to establish this diagnosis. From an ophthalmic viewpoint, however, the case report raises questions about the management of the patient, who presented with an extremely painful red eye. The article does not give an aetiological explanation for this pain, which in herpes zoster ophthalmicus (HZO) can be caused by several mechanisms; and treatment needs to be tailored accordingly (Table 1). In most hospitals a dedicated pain clinic led by anaesthetists

Table 1 **Mechanisms and treatment of pain in herpes zoster ophthalmicus**

Mechanism	*Treatment*
Herpetic keratouveitis	Oral or intravenous acyclovir (Refs 2,3)
	Topical steroids
	Topical cycloplegia
Raised intraocular pressure secondary to keratouveitis or trabeculitis	Topical steroids
	Pressure-lowering medications such as topical beta-blockers, topical α_2-receptor agonists or topical or systemic carbonic anhydrase inhibitor (acetazolamide)
Scleritis	Topical steroids
	Systemic NSAIDs
	Systemic corticosteroids in combination with systemic acyclovir
Acute skin pain	Oral or intravenous acyclovir
	Stellate ganglion block (Ref 1)
	Capsaicin skin block (Ref 4)
Postherpetic neuralgia	Stellate ganglion block, given early (Ref 5)
	Capsaicin skin lotion (Ref 4)
	Antidepressants, gabapentin (Ref 6)

NSAID=non-steroidal anti-inflammatory drug

will be in the best position to advise on intractable pain related to HZO. Early referral is warranted, since adequate pain management in the acute inflammatory phase may prevent development of postherpetic neuralgia[1].

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Venous pulsation in the orbit

I would like to add to Dr Cheng's good letter about pulsating veins (April 2000 *JRSM*, p. 213). Sir Thomas Lewis showed that the height to which the jugular veins pulsate, when measured vertically from the sternal angle, was a good guide to right atrial pressure[1]. He wrote that 'in cardiac failure . . . pulsation of the veins is often very prominent and extensive'[2]. In 1940 he described a patient with advanced cardiac failure and atrial fibrillation who had a jugular venous pressure in excess of 11 cm with venous pulsation extending from the angle of the jaw to the front of the ear. With each pulsation the eyeball moved forward several millimetres and he was able to record this movement with an ink-writing polygraph. Lewis said that in the five years after this observation he sometimes saw venous pulsation in the orbit though it was only slight, adding that the observed eye must be brought to a suitable level in relation to the heart[2].

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The emperor with the shaking head

Dr Rice's description of Claudius' disorder (April 2000 *JRSM*, pp. 198–202) is admirable, but I do not think the evidence adduced permits a diagnosis of a secondary dystonia. Admittedly, the retrospectoscope is a poor instrument for diagnosis, especially with so little available accurate description[1].

Her diagnosis is essentially based on a normal early childhood, onset of symptoms in early youth and diurnal variation of symptoms. This may not be accurate, since the Romans would have hidden any suggestion of physical defect or 'imbecility', particularly in a member of a royal family. In Britain we did much the same until the mid-20th century, albeit with perhaps fewer assassinations.

Like Mottershead[2], I suspect that Claudius suffered from the athetoid variant of cerebral palsy from birth. His gait disorder, abnormal movements[3,4] of the head and hands, dysarthria[5], hypertrophy of neck muscles, unseemly laughter and anger are characteristic. Improvement when 'declaiming' is quite consistent and reminds us of the disappearance of organic stammering when the patient starts to sing or shout. Claudius published many works, of which none survives. They included 43 books on Roman history, 21 on Etruscan history, and 8 on Carthaginian history, a book on philology and a rhetorical defence of Cicero. These works and his mastery of government institutions testify to his intelligence[6].

High intelligence is common in this athetoid group. Whilst teaching briefly at Harvard, I encountered a medical student with classic athetoid cerebral palsy, sinuous abnormal movements of head, trunk and limbs, and gross dysarthria who had an IQ of 150+.

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Malaria with a positive 'monospot' test

In his 'survival guide to imported fever' (March 2000 *JRSM*, pp. 124–128) Dr Jacobs warns that malaria can develop even years after exposure.

A woman aged 22 reported intermittent fevers for the past month. She had been evaluated twice by her student health centre, which had diagnosed infectious mononucleosis on the basis of the clinical picture and a positive 'monospot' test. As well as fever every second or third night she had rhinorrhoea, sore throat and a non-productive cough. On examination she was febrile and had slight splenomegaly. Haemoglobin was 10.2 g/dL and repeat 'monospot' test was borderline. Results from her previous evaluation were reviewed, and these included negative serology for Epstein–Barr virus. It then emerged that, a year earlier, she had spent three months in Kenya and on return to the USA had not completed her full course of mefloquine chemoprophylaxis. Blood film was positive for malaria and she recovered with treatment. EBV titres remained negative.

Heterophile antibody tests are a rapid, inexpensive method for diagnosis of recent EBV infection but are limited by their nonspecificity. False positives with malaria have been reported before. Other rare causes include toxoplasmosis, virus infections (cytomegalovirus, hepatitis, HIV-1), bacterial infections (brucellosis, Lyme disease, syphilis, rickettsial disease) and drug reactions[1].

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Ancient records of birth defects

Dr Bates (April 2000 *JRSM*, pp. 202–207) describes various deformities and monstrosities, his earliest reference being 1552. He points out that congenital malformations were 'a subject for popular literature in Elizabethan England'. In 1921, Eugen Hollander published a book of some 373 pages with 202 illustrations entitled *Wunder Geburt und Wunder Gestalt*[1]. This volume brings together cases described in German in many pamphlets that appeared between 1495 and 1670. Also, G-J Witkowski in his large book *Histoire des accouchements chez tous les peuples*[2] describes many monsters from the literature with illustrations of conjoined twins, hermaphrodites, supplementary breasts (including one on the left thigh), and so on. I have not found any pamphlets myself, but the German and the French books are interesting and well illustrated.

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Pulled elbow

In his article last year, Sai Sankar[1] noted that 30% of children with pulled elbow have no clear history of traction but he did not offer supporting data. We too have observed that the 'classic' history of sudden traction is commonly absent. On auditing the notes of 31 patients seen over three months with 32 episodes of pulled elbow, we found that 24 gave a typical history of sudden traction on the affected arm; the remaining 7 gave no such history. Radiographs of the arm were obtained in 2/24 of the former group, 2/7 of the latter.

We suggest that the absence of a clear history in 20% of our patients may be simply explained by adults' reluctance to say they have accidentally harmed the child. Medical staff must be sensitive to this possibility, if unnecessary irradiation is to be avoided.

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The most pressing issue?

Professor Potts (January 2000 *JRSM*, pp. 1–2) is concerned about the future world population and believes it would be a good idea to reduce birth rates by abortion, contraception and sterilization. Although he refrains from using the term population control, this is what we are dealing with.

The population control lobby has always believed in a degree of compulsion in restricting numbers of births, and continues to do so. At the 1969 population conference in Dacca, radical social engineering measures were proposed in order to destabilize the institution of the family. With the full knowledge that birth rates amongst cohabiting couples are much lower than birth rates amongst married couples, the population control movement and its various adherents has waged a long campaign to undermine the institution of marriage. The International Planned Parenthood Federation [IPPF (to which the British Family Planning Association is affiliated)] has advocated the provision of 'fertility control' to children from the age of ten without parental consent[1]. By encouragement of early sexual activity, a risk factor for subsequent marital breakdown is introduced.

Population control lobbyists like to give the impression that there is some kind of unmet need they are trying to meet. This 'need' is often generated by the population controllers themselves. IPPF has published suggestions for disincentives for couples who fail to follow national population control policies[1]. There are numerous examples of coercion around the world where forced abortion, contraception and sterilization are carried out. The United Nations donations to the Chinese programme to buy 600 vans equipped with both abortion suction machines and clamps for women who try to resist, and the brutal sterilization programme in Peru, which to date has caused at least 18 maternal deaths (www.pri.org), are only two current examples.

According to a recent World Bank report, contraceptive use accounts for only 5% of the differences in fertility rates between countries. Parents' own desired family size is the best indicator of their country's total fertility rate.

In the developed world there is a population problem and it is a population *implosion*. According to the United Nations population division, within 25 years the European Union will need up to 159 million immigrants to keep the current ratio between workers and retired people. The millstone of below-replacement fertility is now firmly around China's neck. Family breakdown, increased abortion rates, human rights abuses and longer term economic problems are some of the destructive effects of population control not mentioned in the editorial.

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Chronic fatigue syndrome in mother and child

Dr Rangel and colleagues (March 2000 *JRSM*, pp. 129–34) report 'family factors' in mothers of children with severe chronic fatigue syndrome (CFS) and recommend further investigation of this aspect. Their observations strengthen the likelihood that many such patients suffer from osteoarthralgia. The reason that this explanation has been avoided in the past is in part because there are no blood tests to prompt suspicion. Moreover, it seems that there are many specialists who are unwilling to diagnose osteoarthritis (OA) until there are changes on X-ray. But what were those pains at the same sites for the many years before the X-rays showed changes?

It seems that the tenderness over muscles ('trigger points') giving the impression of a myalgic condition is due to tenderness referred from joints. As such the condition should be regarded as an arthropathy rather than a myopathy.

However, the greatest obstacle to considering this possibility is that OA is thought of as a disease of old age. Herein lies the unexpected explanation. Careful history-taking will often reveal a strong family history of this creaking joint syndrome, often on the maternal side. Even more interestingly there is often an element of genetic anticipation with successive generations starting it a decade or two younger. For example, where the interval, which tends to run true for a given family, is 20 years, great grandma might start it age 70 if she lived that long, grandma age 50, the mother 30, and the child age 10 years. It is this genealogical evidence that can make the diagnosis so credible. The reason for a lot of the so-called psychological overlay is that no plausible diagnosis is available, with the implication that all the suffering is inappropriate.

This insight was gained indirectly through studies of low back pain in which young OA could be recognized through identification of the facet joint syndrome which more often than not is engendered by OA[1,2]. Clearly there can be other causes that would be classified under the umbrella of the symptom complex of CFS including 'genuine' post-infective debility.

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Bias in peer review

We respond to your editorial 'Of knowledge and deception' (April 2000 *JRSM*, p. 163), in which you questioned the propriety of research involving deception of peer reviewers. The current consensus is that peer review is better than any alternative. This needs periodically to be re-examined and the stimulating article[1] which you commendably published in the same issue is part of this process.

Resch *et al.* reported that fewer reviewers recommended publication of invented data relating to a homoeopathic remedy than the same data relating to an allopathic drug, although scoring of individual aspects did not differ. This is clear evidence of bias, which might have been greater had a therapy even less well accepted than homoeopathy been cited—for instance, food intolerance.

If they had known they were being tested, the reviewers would surely have sought to conceal their prejudices; without studies which, like this, blind the study subjects we will never find out. If potential referees declined to be tested would you and other journal editors stop using them?

In our long publishing careers we have produced around 129 papers and during this time have met some very helpful reviewers whose advice (not always favourable) has been of great value. We have also seen the reverse. This includes a study taking two years to complete being dealt with in five lines, a review whose author had clearly not read the paper, reviews from individuals unfamiliar with the subject who were nevertheless happy to express an opinion, and a referee seeing a revised text submitted to a different journal providing a copy of his/her original report, taking no notice of the fact that the criticisms had been met.

Soon all UK doctors will undergo revalidation. It is not unreasonable to expect that the process of peer review should, like other aspects of medical practice, be able to demonstrate its objectivity and value.

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1 Resch KI, Ernst E, Garrow J. A randomized controlled study of reviewer bias against an unconventional therapy. *J R Soc Med* 2000;**93**:164–7

John Cheyne

This month in history

Brainstem haemorrhage was first recognized pathologically by Cheyne in 1812, in his work *Cases of apoplexy and lethargy: With observations upon the comatose diseases*, published in London. One of Cheyne's initial cases was a 35-year-old carpenter who died on the night of 1 June 1808. The symptoms included unusually severe recurring headaches for the past few months. After a long day of work, the patient became breathless and complained of severe headache. He appeared very ill, vomited, and became insensible. 'About an hour and a half after the attack, his breathing was extremely irregular and laborious; inspiration would cease for nearly a quarter of a minute and then go . . . his pulse was slow and irregular . . . . A pound and a half of blood, taken from the arm was the only measure tried for his relief . . .'. The patient died about two and a half hours after his breathing became affected. Postmortem examination revealed dark clotted blood within the pons.

Although born and trained in Scotland, John Cheyne (1777–1836) made his greatest medical contributions in Dublin, as an influential member of the 'Irish School'. The history of 'Cheyne–Stokes respiration' can be traced back to Cheyne's paper in the *Dublin Hospital Reports* in 1818, and the account of Stokes 28 years later.

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