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Res Medica

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CONTENTS

Editorial

"The Society dates from the year 1737, when ten medical students agreed to meet weekly in a tavern to hear one of their number read a dissertation on some medical subject. It is the oldest medical students' society in Great Britain, and the only one to hold a Royal Charter (which was granted by King George III in 1779) The Society has a long and distinguished tradition, and its history is the history of the Edinburgh Medical School. Amongst its past members it boasts the names of Lister, Simpson the chloroform pioneer, Joseph Bell of Palsy fame, Charles Darwin, and Andrew Duncan. Today as in the past the Society's business comprises the reading of dissertations by members, the arranging of talks given by men eminent in the contemporary medical world and the organising of symposia on subjects of current interest."

Since the founding of the Royal Medical Society in 1737 it has always sought to broaden the educational horizons of its members. *Res Medica* was first published in 1957 to augment learning and provide a platform on which members ideas and work could be aired out with the walls of the Society's hall. This edition of *Res Medica* represents its resurrection after a dormancy period of eight years and it is hoped that this tradition will be supported and maintained in future years. Over this eight year period we have witnessed numerous changes in the practice and teaching of medicine and the Royal Medical Society has remained in touch with these new ideas especially with its recent investment in a variety of computer aided learning facilities.

For some time, our national headlines have been preoccupied with reports on how our food is a threat to our health. The BSE crisis has undoubtedly had the greatest immediate impact. It therefore seems highly appropriate that in this edition of *Res Medica* Dr. Jeanne Bell from the Western General has provided us with an overview of the current understanding of BSE and its links with CJD. It appears that this disease is indeed transmissible as well as carrying a genetic predisposition.

Edinburgh is a city rich in history and The Royal Medical Society is extremely fortunate to have ex-presidents such as Professor Kaufman with his enthusiasm about Edinburgh's past and we are very grateful for his splendid account on the history of the RMS buildings.

We are always happy to receive contributions to *Res Medica* and welcome support in any manner. Many thanks must go to all the contributors of this edition as well as to our sponsors and in particular to the Royal Medical Society Trust whose financial support has brought *Res Medica* back to life.

Update On CJD

Dr Jeanne E Bell, CJD Surveillance Unit, Western General Hospital, Edinburgh EH2 4XU

INTRODUCTION

In March 1996 the discovery of a new form of Creutzfeldt-Jakob disease (CJD) was announced to a shocked world. In order to understand why this news took the headlines by storm, it is necessary to trace the history of this rare disease and in particular its links with bovine spongiform encephalopathy (BSE), or "mad cow disease". By 1996, Britain had been in the throes of the BSE epidemic for ten years and it was believed that cows had contracted the disease by consumption of scrapie-contaminated food-stuff, scrapie being a similar but much older disease in sheep. Because this represented a species jump from sheep to cows, it was predicted in many quarters that BSE posed a new threat to human health. Therefore the Government took steps to remove BSE contaminated products from the human food chain and the Department of Health set up a surveillance programme to monitor the incidence of CJD in UK. Although CJD occurred subsequently in several British farmers who had BSE amongst their herds, the numbers were not significant and there was no rise in the incidence of CJD in UK above what might have been expected from heightened awareness of the disease and better ascertainment of cases. The Government continued to reassure the public about the safety of British beef but unease remained widespread and the question of CJD and BSE was never far from the headlines. It was against this background that the sudden appearance in UK of what appeared to be a new form of CJD in 10 young people caused major panic[1]. Immediately, the new variant of CJD (nvCJD) was linked in the press with BSE. In fact, there do appear to be some scientific grounds for concluding that nvCJD does results from transmission of BSE, which carries the implication that because huge numbers of people have been exposed to a similar risk, thousands of people may be smitten by this new plague. So what do we know about CJD which might help to determine the likely future outcome? In fact, the story has all the elements of a fascinating scientific thriller - a very rare disease with a long and silent incubation period, a disease both genetic and transmissible, caused by a unique and elusive agent which is apparently capable of replicating itself in the absence of nucleic acid[2].

CJD IS TRANSMISSIBLE

First described in the 1920s by Creutzfeldt and then Jakob, in a handful of patients (most of

whom turned out subsequently not to have CJD!), the clinical phenotype of CJD was soon defined in terms of a rapidly progressive and fatal dementia. It became clear that the neuropathology of this condition resembled that of scrapie, a disease of sheep that had been known for 200 years. Both were characterised by atrophy of the brain (Fig 1), microscopic vacuolation of the grey matter with accompanying neuronal loss and gliosis (Fig 2) and, in some cases, amyloid plaques. The human and animal diseases were linked under the term, spongiform encephalopathies. In the 1950s, suspicion began to grow that these diseases might be transmissible, especially with the discovery of a similar disease called kuru, in a remote tribe inhabiting Papua New Guinea, who practiced cannibalistic funeral rituals[3]. This prompted some crucial experiments in which affected brain tissue was inoculated into animals and the transmissibility was confirmed when the recipients developed spongiform encephalopathy after a comparatively long incubation period. In kuru also there was often a delay of years between ingestion of the infected material and the onset of symptoms. Once the tribe were persuaded to abandon their particular funeral practices, kuru became far less common and has now virtually disappeared although one or two cases are still occurring, highlighting the fact that the incubation period may be as long as forty years.

Kuru was the first known example of human to human transmission of a spongiform encephalopathy. Experimental evidence from inoculation of affected human brain tissue into

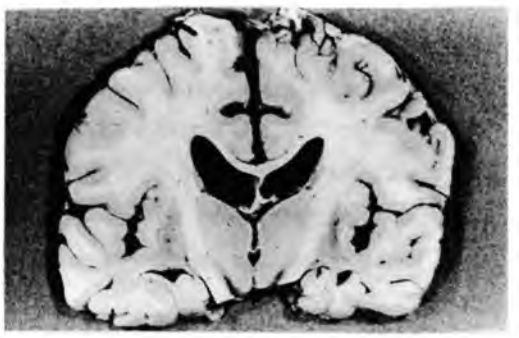

Fig.1 Coronal section of brain from a patient with CJD showing atyrophy and ventricular dilatation.

animals showed that human to animal transmission could also occur. If further evidence was needed that these diseases were transmissible, the occurrence of iatrogenic CJD in the course of medical and surgical treatments certainly confirmed the risk. Iatrogenic transmission of CJD has been reported occasionally following neurosurgical procedures and in patients receiving corneal or dural transplants, but more often in young patients who were treated with cadaver-

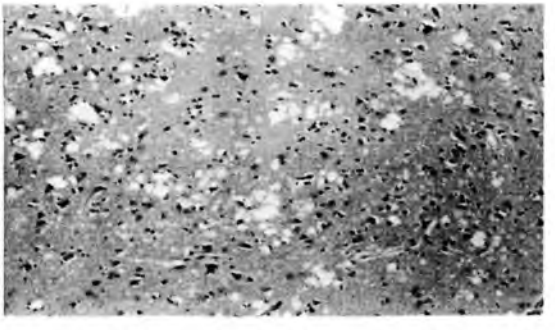

Fig.2 Section of the cerebral cortex from a patient with CJD showing vacuolation of the grey matter. Haematoxylin and easin x200.

derived human growth hormone prior to the 1980s[4], some of whom later developed CJD. Symptoms of CJD were manifested in the recipients between two and thirty years after the therapeutic procedure.

CJD IS GENETIC

While all this evidence indicated that the spongiform encephalopathies were

transmissible diseases, it was also becoming clear that in some cases there was a significant genetic component[5]. Although around 85% of CJD cases occur sporadically and in isolation, the remainder have a history of other affected family members. An even rarer form of human spongiform encephalopathy known as the Gerstmann-Straussler-Scheinker syndrome (GSS) occurred only in several very large kindreds and the disease was clearly inherited. Fatal familial insomnia is a more recently recognised member of this group of heritable CJD-like diseases, but does not display spongiform change, rendering the term spongiform encephalopathy no longer appropriate.

GENETIC AND TRANSMISSIBLE - What is the infective agent?

The fact that these diseases could be both transmissible and inherited made them of unique interest and focused attention on characterising the infective agent in the human diseases. Parallel studies of scrapie were in progress both to isolate the infective agent and to identify the genetic background[6]. The genetic locus was identified and named in Edinburgh's Neuropathogenesis Unit as the sip gene in sheep and the sinc gene in mice. Inoculation experiments in which scrapie brain material was injected into mice demonstrated a range of incubation periods and pathology patterns which were very reproducible and which were attributed to strain variations of the infective agent[7]. This was thought at first to be a "slow" virus (slow because of the long incubation period). However despite intensive study, no nucleic acid was ever identified in the infective material used for transmission experiments, leading to the concept of a virino, this being a very small particle of nucleic acid which was coated with a protein derived from the host animal. While some workers still entertain this concept, current thinking on the nature of the infective agent has swung in favour of the "protein only" hypothesis which has been strongly advocated by Prusiner[2]. In this model, the infective agent is made up of protein (which Prusiner has named prion protein, a term constructed from proteinaceous infective particle). Prion protein, at least in the form which accumulates in diseased brains

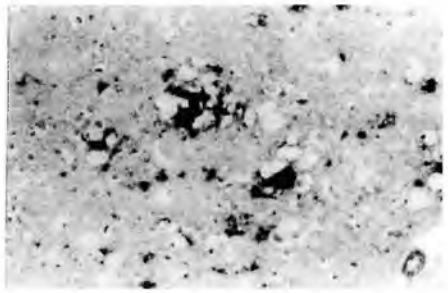

Fig.3 Immunohistochemical evidence of PrP deposition in relation to vacuolation of the cortex in a case of CJD x400.

(Fig 3), is an insoluble, protease resistant protein which is formed by pathological conversion of a normal cell surface protein present in all brains and chiefly on the surfaces of neurones. This protein is of unknown function and is coded from a gene, the PrP gene, which is located on chromosome 20.Work in the last decade has revealed that familial cases of CJD, fatal familial insomnia and all cases of GSS, are associated with mutations in the PrP gene. It also appears that the allelic make up of codon 129 of the PrP gene is peculiarly influential in that homozygosity for valine or methionine at this locus confers susceptibility to the disease both in sporadic and in acquired CJD. To summarise the prion theory briefly, the notion is that in affected individuals the normal soluble form of PrP undergoes a post-

translational modification to an insoluble form which is deposited in different patterns and locations in the central nervous system (Fig 3), giving rise to the characteristic pathology and symptomatic disease. The possession of a PrP mutation makes this change very much more likely to occur so that mutation-bearing individuals have a strong likelihood of developing disease in their lifetime. Cases of sporadic CJD are attributed to spontaneous post-translational modification occurring in the brain of that individual for unknown reasons. In iatrogenic cases, the infective protein may have been introduced directly in contact with the brain (e.g. by dural transplant) and this initiates a relatively rapid onset of translational modification of the host PrP. In patients inoculated peripherally (e.g. injections of infected growth hormone) the agent presumably travels from the periphery to the brain where the pathogenetic change commences somewhat later. The route by which the agent travels from the periphery to the brain is still unknown but may be along peripheral nerves and through the spinal cord, or through lymphatics and involving the lymphoid tissues and the spleen, or by the bloodstream.

Although there is persuasive evidence for the protein-only hypothesis particularly from the spontaneous development of disease in transgenic animals bearing a mutated human PrP gene, there are still some unexplained features such as strain variation[7], which suggest that other factors may play a part in transmissibility. Many workers in the field consider that variations in pathology and incubation period are governed solely by the host genome which controls expression of the PrP gene and that extraneous nucleic acid is not required to explain the phenomena of transmission. So far as function of the normal prion is concerned, it is of interest to note that animals in whom the PrP gene has been deleted progress to old age in an apparently normal manner without neurological deficits. These PrP-null animals also fail to develop any signs of disease when inoculated with infective material, confirming that the presence of normal host protein is required as a basis for subversion in the disease state. Whatever the true function of normal PrP proves to be, it is widely expressed in many different tissues and it is somewhat perplexing that pathology and symptoms are confined to the nervous system.

It should be emphasised that the disease is not contagious in the sense of being transmitted by normal contact with affected individuals but requires inoculation or ingestion of infected material to effect transmission.

DECONTAMINATION OF PRION PROTEIN

In addition to eluding exact identification, the transmissible agent displays some unique properties in resisting many of the usual decontaminating agents such as boiling water, ultraviolet radiation, alcohol, nucleases and even formalin fixation and gluteraldehyde. Subsequent experiments have confirmed that the agent is remarkably persistent in the environment and can remain infective after years in the soil. The only measures which are thought to be effective in degrading infectivity include very strong chlorine solutions, strong sodium hydroxide, formic acid, prolonged autoclaving at high temperature and incineration. The remarkably resistant and persistent properties of the agent only add to its mystique and cause enormous problems in

planning for effective removal from the environment or destruction of infected material.

BSE AND HUMAN HEALTH

The appearance of the new cattle disease, BSE, led to the Department of Health asking Dr. R. Will, who had previous experience of this task, to set up a surveillance project for CJD in the UK[8]. This was established in Edinburgh in 1990 and the neuropathological arm of this surveillance project was instituted by the author to confirm the diagnosis of CJD in clinically ascertained cases. Dr J Ironside joined the project shortly after it was initiated. The CJD Surveillance Unit monitored all cases of suspect CJD in the UK from 1990 onwards and sought to identify the risk factors for development of sporadic disease by comparison with suitable controls. A high autopsy rate was maintained and this allowed validation of the clinical diagnosis. Approximately 40-50 cases of sporadic CJD were identified in UK per annum until 1995 when a number of unusually young patients were referred to the Unit with a diagnosis of suspected CJD. These patients had an atypical clinical presentation with a psychiatric onset followed by progressive ataxia and finally cognitive impairment. As the pathology became available through biopsy and then autopsy of these young patients, it became clear that they did indeed display some features of CJD but with a highly unusual pattern of prion deposition which was remarkably uniform between cases. These features included the very widespread appearance of prion amyloid plaques in the cerebral cortex and particularly heavy deposits of prion protein in the cerebellum. By February 1996, 10 young cases of CJD had been identified with this apparently new pattern of pathology. None of them showed a PrP mutation or had a history suggestive of iatrogenic transmission. They were however all methionine homozygous at codon 129 of the prion gene. Review of previous young cases that had happened occasionally in the past and consultation with European colleagues involved in similar surveillance projects revealed that this cluster of young cases was peculiar to the UK and that the pathology was of a pattern not previously described. Subsequently one similar case has been described in France. This group of young CJD patients was announced in Parliament in March 1996 and published in the Lancet in April 1996[1]. It was widely assumed that this new variant CJD represented the transmission of BSE to humans and the description of similar pathology produced in macaque monkey brains by inoculation of BSE material only strengthened this hypothesis. Since publication of the initial 10 cases, a further seven cases have occurred in UK but nowhere else in the world. In view of what is known of the incubation period of these diseases, it is impossible to predict how many cases of nvCJD will occur in the future particularly in human hosts of different PrP genotype. Ongoing surveillance of CJD in UK will be required for at least the next decade. Meanwhile the efforts to eradicate BSE from British herds continues and there is no optimism that this will be easy considering what is known of the persistent and resistant nature of the infective agent. Research work is still in progress to establish or refute the connection between BSE and nvCJD and to determine the route by which the infective agent reaches the central nervous system. The

need to develop a clinical test for CJD remains paramount and the recent description of detection of PrP in tonsilar tissue is of great interest both from a diagnostic and pathogenetic viewpoint[9].

If it is proven that nvCJD is due to the transmission of CJD, the cost to families who have lost young people to a dread disease, and the cost to the Government of millions of animals slaughtered, lost export markets and lost public confidence has been incalculable. It is a sign of the potency of public concern and condemnation that millions of pounds have now been directed to research in this field. Let us hope that this unique group of diseases with their underlying fascinating scientific conundrum remain very rare in the human population.

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Comment:

Clinical Features

The clinical features of nvCJD are relatively distinct from those of the sporadic form of CJD, however, there is some overlap in the presenting symptoms and disease course: the absolute diagnosis is confirmed only by neuropathological tests.

The earliest clinical features of nvCJD occur at a mean age of 29 years. The median duration of the illness is longer than that of sporadic CJD (14 months for nvCJD compared to 4.5 months). Over this period the most prominent features are the psychiatric disturbances or sensory symptoms or both [1]. The sensory symptoms reported include foot pain, paraesthesia in the legs and persistently cold feet. Most cases where psychiatric diagnoses were suggested were depressed, apathetic and withdrawn. These initial neurological symptoms are followed by more gross neurological dysfunction, mostly ataxia and a rapidly progressive dementia with involuntary limb movements, urinary incontinence and immobility.

These patients are dependent on continuous nursing care and the development of aphagia may indicate the need for I.V. fluids and artificial feeding. Before death patients are increasingly unresponsive, akinetic and mute.

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For the Relief of Pain

Lucy M^cDowall

"*We humans are the most exquisite devices ever made for the experiencing of pain: the richer our inner lives, the greater the varieties of pain there are for us to feel – and the more resources we will have for mitigating pain*"[1] so say the authors of your trusty Oxford Handbook of Clinical Medicine. Hmm. Few people like to experience pain (there is the odd masochist out there) but we endure it knowing it is for our own good. Looking back though my second year neuroscience notes I find Dr. Malcom Wright exhorting us to remember that:

"Pain is an unpleasant sensory experience quite distinct from any other form of sensation. It occurs following noxious (nociciptive) stimuli in normal persons and is the presenting symptom in many disease states. It is a warning that damage has occurred in the body"[2].

Pain is not only useful to the individual, but also to the medical student and doctor. Imagine how much harder our job of diagnosis and management would be if none of our patients could tell us "where it hurts" or when it "feels better". But what if the underlying condition has been treated as far as possible and the pain still remains? Such a pain signal which is no longer "useful" is labelled chronic pain[3] or neuropathic pain. It is estimated that 11% of the UK population currently suffer from this; approximately 6.42 million people[4]. The college of health suggests:

Chronic pain often has a powerful emotional accompaniment: the pain is anxious - they don't know when the pain will come or when it will end. This may lead to depression, requiring psychotropic drugs as well as analgesia. Autonomic accompaniments such as faintness, sickness and palpitations add to the problems and patients often complain that their chronic pain "rules their lives" and "makes life a misery".

"There has been a failure to understand the essential nature of chronic pain, of how it needs to be viewed as different from acute pain, and as an illness in its own right"[5].

There are pain relief clinics being established in hospitals across the country, but demand far outstrips supply and the waiting times can be long – up to 40 weeks[4]. The Pain Association Scotland is a voluntary organisation that exists to help such unreached people through patient support groups, pain management courses for sufferers and the dissemination of information on alternative methods of coping with pain such as hypnosis, hydro therapy and acupuncture[6]. This is aimed to back up and in

some cases replace the care offered by anaesthetists, psychotherapists and physiotherapists in the hospital setting.

As current and future medical practitioners we are all expected to use our "medical knowledge ....to benefit peoples' health....and provide the best care(we)....can" [7]. Why and how do we do this in the realm of pain control?

Reurning to the wisdom of Malcom Wright, this is why he suggested to us that we might want to relieve pain[2]: (these points relate especially to post operative patients)

- For humanitarian reasons
- To allow the patient to breathe deeply (shallow breaths can lead to alveolar collapse at the bases of the lungs on expiration, a left to right shunt and a fall in arterial PaO_2).
- To allow the patient to cough and expectorate secretions. Retained secretions lead to blockage of bronchi, alveolar collapse, a right to left shunt and a fall in PaO_2. Following bronchial obstruction, pneumonia may occur.
- To allow mobility. Immobility predisposes to deep venous thrombosis and increases the risk of pulmonary embolism.
- To reduce sympathetic stimulation (which causes a rise in BP)
- To reduce the tendancy to paralytic ileus.
- To reduce the metabolic response to trauma.

As a house officer on the ward, pain control most frequently takes the form of adminiseting analgesic drugs. Below are guidelines to help in this[1]:

- Pain is affected by mood, morale and meaning. Explain its origin to both patient and relatives, as explaintion and reassurance can lessen the amount of analgesia required.
- Identify and treat the underlying pathology wherever possible.
- Review and chart each pain regularly eg. On a pain score chart so you can monitor how effective your treatment is.
- Assess each pain carefully- different types of pain respond to different approaches and analgesics eg. Amitriptyline in nerve conduction pain, and NSAIDs in bone pain.
- Choose the best route: oral if possible, or PR, IM, epidural, sub-cut, inhalation or infusion
- Give regular doses with the aim of preventing the pain, rather than analgesiia on an "as required" basis.[1] (Remember pre-emptive analgesia before surgery)

Due to the wind up phenomenon and alterations in the pain pathway which have been produced following chronic pain, these patients are often taking maximum doses of analgesics without relief, and putting themselves at risk of severe side effects. Other methods of pain control have been used in these circulstances:

1. Nerve blocks – an analgesic procedure more appropriate for acute pain.
2. Manipulation methods and exercise eg. Physiotherapy, osteopathy, chiropractice, Alexander Technique, massage and ultrasound.
3. Electrical methods – Transcutaneous electrical nerve stimulation (TENS): a small battery operated pulse generator

used externally with two or more electrodes to relieve localised pain.

4. Behavioural management eg. Psychology, education, biofeedback and relaxation.
5. Complementary therapies eg. acupuncture, reflexology and homeopathy.

Dealing with people in pain is something most of us will be doing every day of our working lives. Remember some of the points outlined above will help us and our patients. Remember, next time you discharge someone suffering with chronic pain, think: hospital follow up and support from the Pain Association Scotland (telephone 0131 312 7955. Head office: Cramond House , Kirk Cramond , Cramond Glebe Road, Edinburgh EH4 6NS).

"Never forget how painful pain is"[1]

Thank you to Dr. Malcolm Wright and The Pain Association Scotland for their help in producing this article.

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Figure 1. The Society's first hall is to the right, being No. 11 Surgeons' Square, next to the equally handsome building with numerous pillars which contained the extra-mural anatomical school of Barclay and subsequently Knox, called Barclay's House, at No.10. This was shortly to become infamous as the school that purchased the often still warm bodies of the victims of the notorious Burke and his accomplice Hare. Barclay died in 1826, and his classes were taken over by Knox. The building to the far left in the engraving is Old Surgeons' Hall, at No.8, and Gordon's class-room, formerly that of John Thompson, at No.9 is just seen between the latter and Barclay's House. This View of Sugeons' Square, published in 1830, was drawn by Thomas H. Shepherd, and engraved by T. Barber.

First and Second Halls of the RMS.

An Early history of the Royal Medical Society, with observations on its first hall (1775-1852) in Surgeons' Square and second hall (1852-1966) at 7, Melbourne Place.

Professor M.H.Kaufman

The Society's First Hall (1775-1852) in Surgeons' Square

Towards the end of 1734, a group of six medical students agreed that they should meet in the evening once a fortnight at their respective lodgings, and that a dissertation on some medical subject, at the choice of the Society, should be composed and read at each of those meetings. This laid the foundations for the Royal Medical Society. The Medical Society was formally constituted in 1734 with ten members, and early meetings were held in a tavern close to the University. A president was appointed to supervise the business of the meeting, and fines were instituted for those that were absent without due cause. A treasurer was appointed to collect these dues, and a secretary appointed to provide a formal record of the proceedings.

Shortly after 1741, when the Infirmary moved from Robertson's Close to its new building in Infirmary Street, the managers permitted the Society to hold its meetings in one of their rooms, and the funds which had previously been spent on hiring rooms in a tavern were now deflected towards the accumulation of a library. With its increasing popularity amongst the students the accommodation at the Infirmary became too small for those who wished to attend the meetings, and the library

was particularly cramped and inconvenient to use. In 1771, a committee was established to investigate the possibility of the Society building its own hall, and a subscription list was opened at that time. By 1775, sufficient funds were available to begin the building of a hall on land granted to the society by the College of Surgeons on a site close to their own Hall in Surgeons' Square, adjoining the grounds of the old High School at the foot of Infirmary Street. The foundation stone was laid by Dr. William Cullen, one of the most ardent of the fund-raisers, and an address was given by the Senior President.

The first meeting in the new Hall was held in it on 26th April 1776. The building was a particularly handsome containing three principal rooms each measuring 30 by 20 feet. One served as a hall for the weekly meeting , another acted as a repository for the Societies valuable library, its natural history collection and anatomical preparations, and it was planned that the third room would be set up as a chemistry laboratory. The roof terminated in a cupola, the original intention being that this be used as an observatory. The atmosphere of the Society's Hall and its surroundings is best gained from an analysis of the well-Known engraving of 1830 of the south-west corner of Surgeons' Square (figure 1). An important landmark occurred the early history of the Society when, following Counsel's advice, the members decided to procure a Royal Charter. This was subsequently granted by King George III on the 14th December 1778. The King furthermore indicated that in his view the Society's activities were entirely laudable and deserving of encouragement. The Charter was sealed at Edinburgh in January 1779, and the Charter and Seal are now displayed in the Society's Hall at Bristo Square. Later that year, the Surgeons generously fixed the ground rent at £5 per annum.

In 1786, several small rooms were available in the Hall for the purposes of private dissection and chemical experiments, *although these had to be undertaken at the expense of the members concerned.* And only those experiments that had previously had the approval of the Society's Experimental Committee could be carried out.

During 1818-19, the membership rose to 84 and this posed particular problems in the Hall where the meetings took place, in the library and in the rooms set aside for scientific investigations. The Society could either restrict its membership, alter the building or consider the possibility of moving to new premises with more accommodation. A committee was accordingly set up to estimate the probable cost of a new building. Various options were considered, but none was proceeded with. In 1835, Barclays House became available, as Knox's classes became too small for their continued viability. The rooms were, however, too expensive for the society to afford. In 1837, an architect was engaged and numerous plans drawn up with proposed alterations to the Hall, including the addition of a third floor. No decision was, however, taken at that time and it was not until 1850 that the Society was compelled to find other accommodation.

A considerable number of sites were investigated, but for one reason or another, none proved suitable for the Society's needs. As the Managers of the Royal Infirmary had

bought an adjoining house for £600 it was estimated that the Society's Hall was not worth more than £400, and a subscription fund was established to support the purchase of a New Hall. The value of the Society's property was not enhanced by the fact that the south gable wall of the Society's Hall had been damaged by the pulling down of the adjacent building (Barclay House) during 1850 with a view to extending the Surgical Hospital. The Managers generous offer for the Society's property of £1700 was gratefully accepted. The sum raised, was however, insufficient to cover the £2000 needed to purchase Melbourne Place, and £600 of the Society's shares had to be realised in order to cover the purchase and furnishing of the Society's new premises. The last meeting of the Society held in the Old Hall in Surgeons' Square was on 12th November 1852 (Figure 2), and the first in the extensively refurbished New Hall took place one week later, so that by this means continuity was maintained.

The Society's Second Hall (1852-1966) at 7 Melbourne Place.

Because insufficient funds were available to consider the building of a *new* hall that would be able to accommodate a meeting hall of adequate size for the considerable membership of the society, to house its extensive and valuable library, to provide experimental laboratories and rooms for the officers of the society as well as additional sundry committee rooms, it was accordingly decided to purchase the upper four floors of the tenement at 7 Melbourne Place. Despite the fact that extensive remodelling of the interior of the building was required to satisfy the needs of the society, all the work was completed during the following 6-7 months, and the Society was not only handsomely accommodated but was able to hold its first meeting in the New Hall on 19th November 1852.

Figure 2. Painting on wood by a Mr. Dallas of the Medical Society's Hall in Surgeons Square as it appeared shortly before its demolition. Note that Barclay's House, formerly located to the right of the Hall, is not shown, and that numerous minor differences exist between the depiction of the Hall shown here and in Shepherds version (figure 1). This picture now hangs in the Society's Meeting Hall.

The group of buildings along Melbourne Place was erected in 1835, with the demolition of Old Bank Close the previous year. The first individuals to occupy No. 7 were Messrs. James and John Gray, who started trading there in 1837, and were the proprietors of various weekly and monthly broadsheets.

The view from the Front of the property was particularly impressive and the Society spanned five windows in width. The Fifth storey was ornate and in the Dutch gable style, and surmounted by a large golden eagle with outstretched wings in the Imperial Roman or French style (figure 3). The suggestion that the property had at one time been the Prussian Consulate appears to have been a folk legend associated with the Eagle.

Figure 3. External view of the frontage of No. 7 Melbourne Place, showing the main entrance doorway, flanked on either side by two shops. The width of the Society's premises is seen to include the five windows clearly displayed on the 2nd 3rd and 4th floors. The Eagle, with spread wings, is mounted on the pinnacle above the 5th storey (with permission, Royal Commission on the Ancient Historical Monuments of Scotland).

Unfortunately, no records survive to indicate the extent of the alterations made to the fabric of Melbourne Place, but they were considerable. Substantial changes were made to the third and forth storeys of the building to produce the meeting hall, which fully extended the width of the property. The intervening floor had had to be removed, and the windows on the easterly-directed fourth floor that fronted onto Melbourne Place were always kept shuttered, while those on the third floor were always covered with heavily draped curtains. The roof of the Hall was timbered and supported by substantial wooden beams. A number of large fittings hanging from the ceiling supplied the majority of the lighting, and these were supplemented by small wall fittings, one pair on either side of the president's chair.

The president's enormous chair emblazoned with the society's emblem, was raised on a platform, with its back towards the middle of the eastern wall of the hall, directly in front of which was the president's desk. High on the wall above hung a painting depicting the Royal Arms of King George III. The secretary and two other officers of the Society sat at a table in front of the president to record the proceedings. To the left of the chair was the Society's Royal Charter, and to the right was the bust of Dr. John Gordon (figure 4). At the north end of the Hall (figure 5) was a marble fireplace, above which hung the painting of Andrew Duncan *Snr.* At the opposite end of the hall was a similar fireplace, and on this wall hung paintings of William Cullen and Joseph Black.

Figure 4 (Above) Internal view of the eastern side of the meeting hall, showing the president's chair and desk, both raised on a platform, in front of which is the secretary's table. To the left of the president's chair is the Society's Royal Charter , to the right is Gordon's bust, and above the chair is the Coat of Arms of King George III.Figure 5. (left) View of the south end of the hall, showing the painting of Andrew Duncan Snr. Note in particular, the patterned wall covering with its very ornate borders.

(Both figures with permission. The Royal Commission on the Ancient and Historic Monuments of Scotland).

The wall coverings in a drab olive-green stucco were particularly memorable. The background was floral in design and interspersed in a regular pattern with the logo of the Society - the scrolled letters "RMS" surmounted with a small crown against a plain background. The heavily ornate borders were about a foot wide; on the north and south walls the vertical borders included the medical insignia, a staff associated with an entwined snake. High up in the centre on each wall was a very ornate Royal Coat of Arms, and on either side exotic shields, one of which announced "Society Instituted A.D. 1737", while the other proclaimed "Incorporated by Royal Charted A.D.1778" (Figure 5). The whole impression was that of a very conservative gentleman's club, which is exactly what it was until the last year of the Society's existence at Melbourne Place , in 1964, when female medical students were first admitted to membership of the Society.

Above the meeting hall, the fifth storey was originally fitted out as the librarian's house, while the small rooms on the fourth floor initially served as a museum. On the third floor, the two smaller rooms accommodated some of the Society's older books, while the other room was adapted in 1938 to preserve the most valuable books in the collection. On the second floor were accommodated the north and south libraries, and the secretary's and librarian's rooms.

The main door of the premises opened into a wide entrance passage and the foundation stone which had been recovered by the workmen with the demolition of the Hall in Surgeons' Square in 1853 was, in due course, incorporated into the wall of the staircase. It is now displayed in the Society's present Hall at Bristo Square. The ground floor only consisted the entrance passage, as two shops were situated one on the south and the other on the north side of the society's main door and entrance.

The entire block of properties in Melbourne Place was compulsorily purchased by the Edinburgh City Council in 1965, and demolished shortly afterwards, being replaced by the Council Offices of no architectural merit whatsoever. When the Society's premises were demolished, the original Foundation stone to the first hall and the eagle from the pinnacle of No. 7 were rescued (figure 6) with one or two other smaller momentoes, and these are now displayed in the Society's present premises in Bristo Square. Indeed it was one of the author's first duties as senior president, in October 1966, to accompany the eagle as it flew from Melbourne Place to Hill Square *en route* to its present eyrie at Bristo Square.

The excellent relationship that has always existed between the Society and the Royal College of Surgeons came to the Society's rescue when the College offered the Society Temporary premises at £. Hill Square, close to the site of the Society's first Hall in Surgeons' Square. While far smaller than Melbourne Place , it provided the Society with a breathing space during which a more pleasant site could be found. Unfortunately because of the lack of space, only the bound dissertations and a relatively small proportion of the Society's enormous collection of books could be adequately displayed.

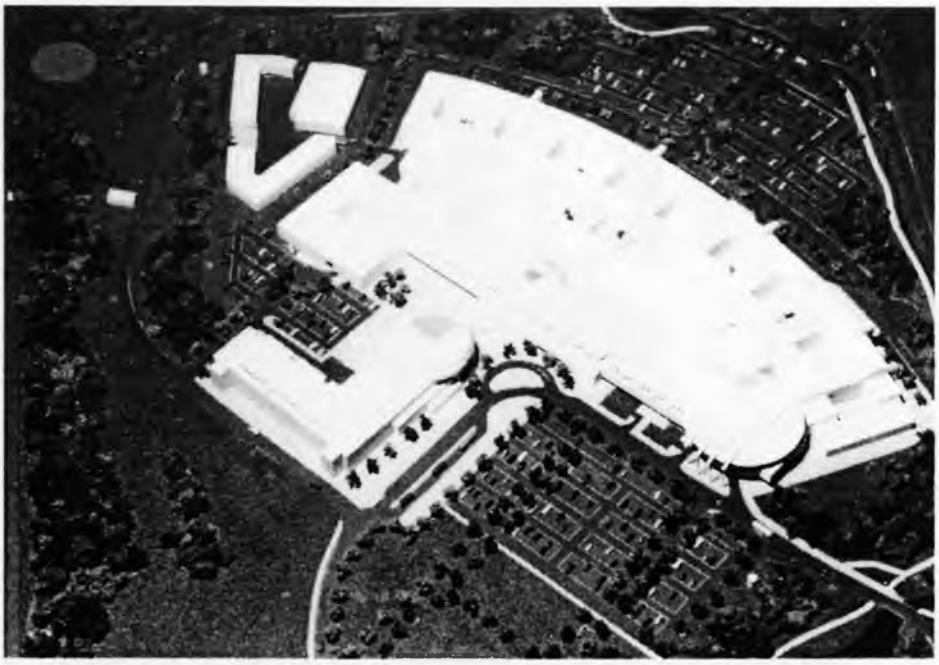
Figure 6 Rescuing the eagle from the ruins of Melbourne Place, On the back of this photograph is a contempoary inscription: *"The eagle leaving 7 Melbourne Place on 23/10/66 for Hill Square attended by the Senior President, Mr. M.H.Kaufman and Miss F.M.Marr and Mr.R.Nixon, Junior Presidents"* (In the author's possession).

An appeal Committee was established and recommended that a major part of the historical collection of books be sold. It was, however, accepted that the Society should keep as the foundations of a continuing library its unique collection of dissertations by members, complete from 1759, together with a selection of books of particular relevance to the Society and to Edinburgh medicine.

Gray in his *History of the Royal Medical Society 1737-1937* (1952) wrote that "The Society is primarily, but not purely, a student society. There is a senior element, never intrusive, but careful to ensure adherence to sound traditions. With the maintenance of these traditions, the Royal Medical Society of Edinburgh will continue to flourish". It is to be hoped that these ideals, and the continuity with the past that they embrace, will be maintained.

Acknowledgements:

The author is grateful to Mrs. Pat Strong and Dr. Jack Cormack for their advice during the preparation of this manuscript, and assistance by the staff of the Special Collections section, University of Edinburgh Library.



R.I.E.

The building of the New Royal Infirmary is now under way. How can this smaller hospital meet the demands of its surrounding population?

The Royal Infirmary of Edinburgh at Lauriston Place is soon to be replaced by a new 869 bed teaching hospital to be built in Little France, $3^1/_2$ miles from the city centre. Currently the 1067 bed hospital provides acute care, rehabilitation and long term care as in-patient services as well as a wide range of out-patient facilities. The new RIE will be adopting a new method of care provision and will be organised in a different way to maintain the existing clinical services at the same time as meeting the demands of teaching and research

Lothian Health's strategy to maximise the efficiency of a high technology acute hospital on a peripheral site is based on the principle of "doing at Little France only those things that can be done at Little France". This goal will be achieved by increasing the proportion of healthcare services in the neighbourhood and at home, supported by modified acute services based on modern facilities at fewer sites. It has been proposed that by spreading services to a small number of out reach facilities (five such locations are planned in Edinburgh) the new Royal infirmary will not only function better but serve its population better too. Out reach clinics, where hospital consultants provide services for patients on GP premises, have been available for a number of years. The most

common service provided is by psychiatrists with more recently an increase in ENT, dermatology and orthopaedics (presently 30% of ENT and dermatology clinics are held off hospital premises). The expansion in out reach out patient clinics has been driven largely by the development of GP fundholding. Many trusts now face pressure from GPs and health authorities to establish out reach clinics in a range of specialties. Moreover, moving towards a primary care led NHS, in which an increasing proportion of care is delivered outside the traditional hospital setting, is now a national NHS priority.

Evaluation of the original concept of out reach, in which a large number of low-tech common clinics should be provided in GP clinics and health centres, has failed to show any benefits except to the immediate local population. Research has also demonstrated that these types of set up have not facilitated the transfer of skills from the specialists to the GPs [1]. A number of basic operational difficulties have also been identified with GP satellite clinics of this kind: a fully equipped clinic requires a large capital expenditure, the administration of patient records poses a problem and travel to outlying clinics is not an efficient use of consultant time [2]. The probable increase in the number of practices that want such set ups make it unlikely that this type of out reach clinic will have a permanent place in the NHS as there are simply not enough consultants.

The new model for out reach services which is to be adopted by the New RIE states that a range of appropriate services could be developed in appropriate volume to a small number of centres, geographically dispersed within Edinburgh and the Lothians and tailored to suit the population needs which will function as devolved locality clinics. These would provide a range of well staffed and well equipped out reach services aiming at high standards of care, efficiency and quality. Locality clinics could serve geographical subsets of the Edinburgh population not only as out posts of the Royal Infirmary but as visible and effective local centres for a range of health related services and activities.

Community Treatment Centres: Types of services to be provided

Day care/day hospital places
Community teams
Pre-admission services
Diagnostic services
Hospital out patient clinics/shared care facilities
Health promotion/information services
GP out of hours services
Complementary medicine
Post acute rehabilitation
Telemedicine ie. Scanned images sent to hospitalspecialists via satellite link.

References

1. Leese B. Health Service Journal December 1996 24-26.

2. Robb P. Health Care Management **11**: 34-35.

Abstract

The following are summaries of student led research projects.

Epidemiology and Outcome in Lateral end Clavical Fractures.

L.A.Khan *Department of Orthopaedic Surgery, Edinburgh University Medical School.*

The clavicle is the most commonly fractured bone in the human body. Fractures of the clavicle can be divided into medial, middle and lateral clavicle fractures. Conservative treatment is widely accepted as the treatment of choice for both medial and meddle clavicle fractures. However, lateral end fractures have previously been shown to have a high incidence of non-union and re-fracture if treated conservatively and a number of studies have recommended operative treatment, open reduction and internal fixation for this type of fracture.

Our results have shown that males were 2-3 times more likely to sustain a lateral end clavicle fracture and the average age at fracture was 42 years for males as opposed to 47 for females. The main causes of fracture were simple falls, road traffic accidents, falls from heights and sporting injuries. The results also showed that in contrast to the earlier recommendations those patients who were treated conservatively had earlier union, less local complications and were able to return to work and activities of daily living earlier than those who underwent operative treatment. In addition those patients who were treated conservatively had better shoulder function, less pain and were more satisfied with the outcome. This leads us to conclude that lateral end clavicle fractures should be treated conservatively.

Study into Oxygen Therapy and Doctors' Knowledge of it.

Adam J. Paul. *Western General Hospital, Edinburgh.*

We looked at the use and knowledge of oxygen therapy. This involved postal questionnaires sent to SHOs and PRHOs from which we received 120 replies, interviewing 45 SHOs and PRHOs at the WGH and visiting all patients on oxygen on 5 occasions over 5 weeks. In total 72 patients were seen. With a response rate of 52.1% only 7.3% of doctors were able to answer 6 basic questions correctly; only 20% of those working at the WGH knew when oxygen should be humidified. 25.5% of interviewees write up oxygen on the Kardex with only 1 in 45 specifying a mask type. However, 94.7% of patients seen were wearing their masks, but only

19.4% had an oxygen prescription written up and 30.6% had had no ABG or pulse oximetry carried out since they had been started on oxygen. Given these findings we felt that, coupled with 26.7% of interviewees who have had no formal oxygen therapy teaching, there is a need for improvement in oxygen prescription and documentation procedures and in basic oxygen therapy knowledge of junior staff.

Diabetes Prevalence and In-Hospital Mortality in Patients with acute Myocardial Infarction.

James East. *Departments of Cardiology and Diabetes, Royal Infirmary of Edinburgh.*

Acute myocardial Infarction (AMI) is a principal cause of death in patients with diabetes. To Quantify both the prevalence of known diabetes and previous undiagnosed diabetes (random plasma glucose > 11.1 mmol l^{-1}) in patients presenting with AMI and in-hospital mortality we retrospectively examined the records of all 409 patients admitted to the RIE CCU during the calendar year 1997 with AMI. 79 (19%) fulfilled the criteria for diabetes with 29 (37%) being previously undiagnosed (PUD). Mean (+/- SD) admission plasma glucose was 14.7 mmol l^{-1}. The in-hospital mortality rate was 14% for non-diabetics and 25% for diabetic patients, (p=0.026), odds ratio 2.04 (95% C.I. 1.13-3.70). The excess mortality was accounted for by the death rate in those with PUD, 41% vs 16% known diabetics, (p=0.026) vs 14% non-diabetics, (p<0.001). Those with PUD were older (71 +/- 9 vs 66 +/- 10 years, p=0.02), more likely to be female (79 vs 47%, p=0.021), and had a higher admission plasma glucose (15.9 +/- 3.4 vs 13.8 +/- 5.0 mmol l^{-1}, p=0.041) than in those with known diabetes. This suggests that patients presenting with AMI without previously diagnosed diabetes and an admission blood glucose level > 11.1mmol l^{-1}, indicating either stress hyperglycaemia and/or undiagnosed diabetes, represents a high risk group. This group of patients may particularly benefit from active glycaemic management.

Urinary Analysis of Luteal Function in Infetile Couples.

Simon Pridgeon. *The Department of Obstetrics and Gynaecology, Royal Infirmary of Edinburgh.*

Introduction. About half of couples who fail to conceive within a year seek infertility advice. Routine investigations fail to determine a cause in a quarter of cases. The measurement of hormonal metabolites in urine is used widely as part

of ovulation assessment, however, only limited information is extracted from these results. Here we are seeking to find the best ways of utilising this data.

Objectives. To establish guidelines for the interpretation of urine tracking results and to investigate whether this test can be used to identify luteal phase defects. We also aim to evaluate its role in the monitoring of ovulation induction.

Methods. A case note survey of infertile couples in whom urinary tracking has been used either to investigate ovulation or to monitor ovulation induction with anti-oestrogen therapy was carried out and a patient questionnaire was used to examine the methods of urine collection.

Results. 528 sets of urinary tracking results were examined from 400 different women. 165 of the cycles were used to monitor ovulation induction. The patients fell into 6 diagnostic categories: male factor infertility (7.3%) ovulatory infertility (28%), tubal infertility (8%), unexplained infertility (19.5%), mixed cause of infertility (16.7%) and unclassified
(20.5%). Menstrual cycle length was shown to describe variations in ovulatory patterns. A normal range was established for the urinary pregnendiol/creatinine ratio to identify ovulation (1.16 +/- 0.5). An unexplained increase in urinary oestrone was detected in avovulatory women. There was no difference in maximal pregnendiol measurements to identify luteal phase defects in women with unexplained infertility (pmax: 1.15 +/- 0.50 in the unexplained group compared with 1.17 +/- 0.51 for all other diagnoses). There was a significant difference in the maximal pregnendiol values between successful ovulation induction and induction that failed to produce an ovulatory event (1.35 +/- 0.71 compared with 0.36 +/- 0.21).

Conclusion. Urinary tracking is a highly practical and user friendly investigation. It is valuable in monitoring women with long menstrual cycles or those being treated with anti-oestrogen induction. We have found no benefit for the use of urine tracking in women with normal menstrual cycles nor have we found any evidence that it can be used to identify women with unexplained infertility who would benefit from clomiphene therapy. The establishment of clear guidelines for interpretation of urinary data may optimise its usage and cut the costs of infertility services.

Cumulative Ultimate Incidence of Diabetes and Impaired Glucose Tolerance Over 6 Years of Follow-up in Patients with Gestational Diabetes in Edinburgh.

P.J. Huggan,*Department of Diabetes, RIE.*

Objectives. To determine the cumulative incidence of abnormal glucose metabolism in women with previous history of GDM.
To identify factors in the index pregnancy that are associated with an increased risk in later life of abnormal glucose metabolism

Methods. Retrospective study of defined cohort.

Subjects. 119 women diagnosed with GDM at the Simpson Memorial Maternity Pavilion (SMMP) between July 1991 and March 1993, reassessed in 1998.

Main Outcome Measures:

Glucose status as defined by diagnosed diabetes, impaired glucose tolerance or normality during the follow-up period. Patients whose glucose status was unknown at follow-up were invited for measurement of fasting plasma glucose

Results. Twelve women had diagnosed diabetes (10%) and 4 had impaired glucose tolerance (IGT; 4%). Thirty women (25%) had definite normal glucose tolerance and four women (4%) could not be classified. Sixty-nine women (61%) were either lost to follow-up or failed to attend for follow-up screening. The prevalence of confirmed abnormal glucose tolerance in the 50 studied women was 32%.Women with post-partum diabetes had a higher BMI in pregnancy and both fasting and 2 hour blood glucose levels were higher during OGTT in pregnancy.

Conclusions. The 5 year cumulative incidence of diabetes after GDM is at least 20% in this cohort. The high rate of patients lost to follow-up or failing to attend for screening makes an accurate level impossible to determine. Those developing post-partum diabetes were heavier and had a more severe disorder of glucose metabolism during pregnancy.

Trends in HIV Testing- A Comparative Analysis Between Homosexual and Heterosexual Men.

K. Dhaliwal, Department of Genito-urinary Medicine, RIE.

Introduction. The development of an enzyme-linked immunosorbent assay for screening blood for antibodies to HIV was a major milestone in the history of AIDS prevention and treatment. With the recent upsurge of public awareness, testing has become more of a health issue for many people. At present, few studies have been conducted to analyse the reasons for testing and to see if the increase in uptake can be attributed to any specific cause. In 1996 alone, the G.U.M. department conducted 2217 HIV antibody tests (same day testing and routine testing).

Aims. 1) to study the reasons why patients undertake HIV testing and in particular to analyse if there are any differences between homosexual men (HO) and Heterosexual (HX) men in the **reasons for testing**.

2) To assess if there has been an increase over the years in the numbers of test carried out ***proactively*** (patients not at past risk of infection, but confirming their negative status to allow a future event to proceed safely, e.g. discarding condoms).

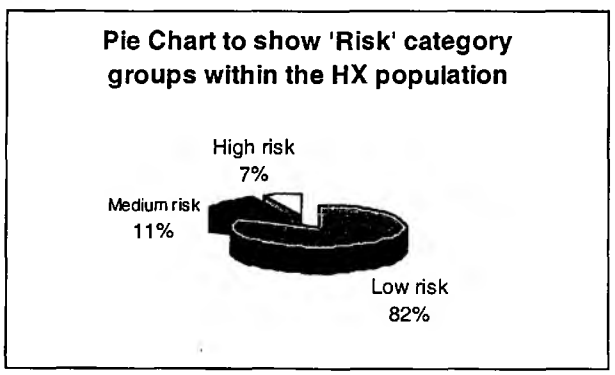
Methods. Database compiled for homosexuals and heterosexuals (matched for age, sex and dates of attendance). Six year quarters from 1994 to 1997 were chosen. Each test episode was assigned a 'reason for testing' category.-past risk, ongoing risk, new relationship, proactive, illness, indeterminate reason.. Results were analysed for quarter studied and age groups.

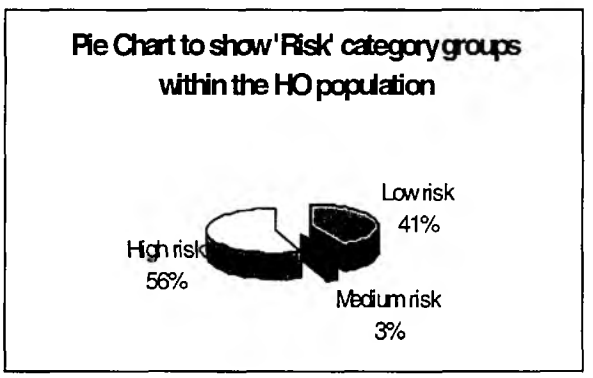
Results. 438 HXs and 427 HOs compared. Past risk- HO= 53% & HX= 52%. 15% HX proactive as opposed to 6% HO (χ^2=18.3, p<0.001). No positive HIV tests in HXs vs 17 in HOs for the periods studied. Risk status; low risk (HO=41%, HX= 82%). Medium risk-(HO=3%, HX= 7%). High risk (HO= 56%, HX= 7%).

Conclusions. **Past risk** and **indeterminate reason** account for the majority of tests taken by both population groups.

The majority of heterosexual males being tested had a 'low risk' of being infected, this was in contrast to homosexual men, the majority of whom had a 'high risk' of being infected (see graphs).

The results of the study do not support the impression held by the clinicians that there had been an increase in the proportion of tests carried out proactively. Further extension of the years analysed may show a trend for increasing tests being taken proactively.





Journal Scan

A review of recent periodicals has revealed some articles of interest.

"Voice of the Castrato". This article by Professor J.S.Jenkins of the department of medical history (The Lancet 1998, **351**:1877-1880) explains the induction of male hypogonadism to preserve the male unbroken voice into adult life and how its poularity was such that in the 18th Century the unique pitch of the castrato dominated opera throughout most of western Europe.

The sound of the castrato voice resulted from the high pitch of a child's vocal chords associated with fully grown resonating chambres produced by the pharynx and oral cavity as well as the adult thoracic cavity. A contemporary critic described the castratro sound as being "as clear and penetrating as that of a choirboy's but a great deal louder with something dry and sour about it yet brilliant, light, full of impact". The author goes on to describe the physical appearance of the castrato and a documentation of the rise and fall of these unique artists.

"The Evlution of the Menopause" appeared in *Nature* **329**: 759-761 (P.W.Sherman 1998). Here, the author explores four theories to explain the menopause. The first idea is that menopause occurs because women live longer now than in the past. Most animals reproduce as long as they live, but zoo species (whose lifespans have been artificially increased) often stop reproducing before they die. Therefore the menopause is a result of medically lengthening the lifespan of a primate with a fixed number of gametes.

Secondly, it may be explained by the deterioration of a physiological process that was once well regulated; like eyesight and memory this process fails with age.

The third theory proposes that the menopause is an adaptive process in that it is an evolutionary mechanism to protect against birth defects which increase with age.

The final explanation involves a concept called the "Good Mother" theory. This proposes that the ability to reproduce later in life may mean that the mother will not be able to care adequately for her offspring as a younger mother and she would also be risking the future of her existing children. The good mother theory is therefore an adaptive theory which allows a mother to devote her attention to her existing young to ensure that more offspring will grow up to reproduce themselves. Supporting this theory are several species in which females live beyond their last pregnancy including Japanese macaques, elephants and killer whales where offspring require extensive maternal care.

"Gastrointestinal Emergencies in Marathon Runners". The New Zealand Medical Journal

(Scobie B.A. June 1998:211-212) presents the cases of two marathon runners who developed gastrointestinal emergencies during a competitive run. The first patient, a 35 year old female collapsed near the end of her race with abdominal cramps. At laparotomy the left half of the greater omentum was found to be infarcted and was resected. Further surgery due to persisting peritonism revealed that the remainder of the greater omentum was infarcted and an acute oedematous pancreatitis was found.

The second athlete was a 27 year old male. After 10 km he collapsed semi-comatose and was found to be hypoglycaemic (blood glucose 2.0 mmol/l). Following intravenous resucitation he regained conciuosness butr registered a temperature of 39.5°C. He developed D.I.C., rhabdomyolysis, renal shutdown and progressive hepatic failure. His condition stabilised following dialysis, however, his hepatic function declinedand died on the 32nd day.

The authors examine the pathological processes in the two cases and suggest that they provide support for ischaemia being a major contributor to GI catastrophies in marathon runners.

Interrupted Fertility

From "A case by Dr. Taylor, 1777".

This case history was resurrected from the societies archives by C .Vaughn Ruckley.

Mrs Buff, wife of Mr Buff, silk weaver in Fashion Street in Spitafields, London, aged twenty-seven years the mother of several childerine, on 27th of July 1774, having gone her usual time of pregnancy, was attended by her midwife several days, but the labour pains ceasing, the midwife left her promising to return soon but did not fulfil her promise. Mrs B. not being delivered of her child, thought she might have a month longer to go and went about her domestic affairs as usual. Christmas following she prov'd with child again and not being delivered of the former one she became uncommonly big and unwieldly, she applied to several physicians for advice and particularly to Dr. Wyman an eminent man midwife in Aldermanbury, who ordered her a variety of purgative medicines but without relief. In June she sent for me, and after hearing the above narrative I assured her of being with child and in October following I laid her of a healthy living child. Having had an easy natural labour, she recovered very well until the tenth day, she was taken of a violent purging, her stools very offensive and of a dark bloody appearance, having taken some astrigent medicines with Diascordium and Diarrhoea ceas'd but was followed by profuse sweats

which weakened her considerably and she was obiged to wean the child I had laid her of, the sweats and purging stools having her for three months she was much reduced in her strength, about this time a thigh bone of a child came away in a purging bloody stool, a few days after half a frontis, two months after she passed half of the under jaw having the sockets of five teeth well marked. All these bones were of a brown darkish colour and were voided with purging bloody stools. March 5th a ragged piece of upper jaw came away, she begins now to recover her appetite and gets strength , the bones always come away with a purging and some coagulated blood with, and after, her stools with a sharp tremus.

During the summer she had passed several small bones , but her appetite and strength is much mended, having gone in a coach to Twitenham 4 miles distant from London she was so ill from the jolting that she was obliged to be brought home in a chair and the day following seventeen bones mostly ribs were extracted, and as most of them lay transversely I was obliged to turn them and bring them out lengthways , this could not be effected without a great effusion of blood and the most excruciating pains. In October the remaining bones of the cranium came away all but one these bones having three edges were always followed a profuse haemorrhage. In November she was troubled by the Whites and a heat in her urine. In December the largest and only remaining bone of the cranium was extracted, the swelling of her belly subsided and she has recovered her strength greatly. In February1776 her courses appeared and the next ensuing period, but both times by the anus, of which she made grievious complaint, I assured her they would soon come to the natural way, which happened the May following, since the above she has been married to a second husband, by whom she had three childrine. The girl which she was delivered in October 1775 is still alive and a fine healthy girl.

RMS Events

The calendar of events for the forthcoming session (1998-1999) runs as follows:

Autumn Term

Friday 9th October: FRESHERS' ADDRESS, Mr. J. Christie, F.R.C.S., followed by a party.

Friday 16th October: INAUGURAL ADDRESS, 7pm Dr. Jack Cormack, *Olim Librorum Custos.*

Tuesday 20th October: PRIVATE BUSINESS ONLY, Election of committees.

Tuesday 27th October: DISSERTATION.

Tuesday 3rd November: VISION 2000, Mr. Philip Evans.

Tuesday 10th November: HOMEOPATHIC MEDICINE, Dr. Sally Tothill.

Tuesday 17th November: DISSERTATION.

Saturday 21st November: ANNUAL DINNER in the Hall of The Royal College of Surgeons, 7pm for 7.30pm. Guest of honour: Dr. Sandy Simpson, F.R.C.S., F.R.C.P.

Tuesday 1st December: NEUROSCIENCE TUTORIAL, Dr. F. Kristmiundsdottir. 8pm.

Tuesday 8th December: PRIVATE BUSINESS ONLY, mulled wine and mince pies.

Spring Term

Tuesday 19th January: DISSERTATION.

Tuesday 26th January: PRIVATE BUSINESS ONLY, Burns Supper.

Tuesday 2nd February: ANNUAL QUIZ, sponsored by the Medical Protection Society, 8pm.

Tuesday 9th February: DEBATE, Medics Vs Lawyers, 8pm.

Tuesday 16th February: PATHOLOGY TUTORIAL, Dr. E. Duvall, *Olim Praeses.*

Tuesday 23rd February: DISSERTATION.

Tuesday 2nd March: PHARMACOLOGY TUTORIAL, Dr. Jamieson Walker.

Summer Term

Tuesday 27th April: PRIVATE BUSINESS ONLY.

Tuesday 4th May: 1st M.B. TUTORIAL: Dr. G. Findlater, Lecture in Anatomy.

Tuesday 11th May: 1st M.B. TUTORIAL: Dr. I. A. Nimmo, Lecture in Biochemistry.

Wednesday 12th May: ANNUAL EXTRAORDINARY GENERAL MEETING, 7pm.

Friday 14th May: PRESIDENT'S VALIDICTORY, Mr. Andrew Sutherland.

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