The Scottish Society of the History of Medicine

(Founded April, 1948)

REPORT OF PROCEEDINGS

SESSION 1998 - 99 and 1999 - 2000

The Scottish Society of the History of Medicine

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THE FIFTIETH ANNUAL GENERAL MEETING

The Fiftieth Annual General Meeting of the Society was held at the Royal College of Physicians and Surgeons, Glasgow on 30 October 1998. The President Dr Harold Swan was in the chair. The minutes of the Forty Ninth Meeting were approved and the Secretary's and Treasurer's reports were accepted. The membership of the Society was noted as 217. Dr Nick Gordon was standing down as auditor. He was thanked for his contribution and Dr Rufus Ross was appointed in his stead. One Council member retired, Mrs Alena Fraser and she was thanked for her help. Four new council members were elected, Mrs Ailsa Blair, Dr E H Jellinek, Dr Rufus Ross and Prof T H Pennington.

Dr David Wright was elected as the new Vice President. The retiring President, Dr Swan then handed over the chain of office to the incoming President, Dr John Forrester.

THE ONE HUNDRED AND FIFTY SECOND ORDINARY MEETING

This meeting directly followed the Fiftieth Annual General Meeting in the Royal College of Physicians and Surgeons in Glasgow. The President, Dr John Forrester, introduced two speakers. The first, Dr Steve Sturdy, Wellcome Award Holder at the Science Studies Unit of Edinburgh University, gave a paper on the Laboratory of the Royal Infirmary of Edinburgh. The second paper was given by Dr Elizabeth Wilson on Family Planning in Sierra Leone.

FAMILY PLANNING IN SIERRA LEONE

My talk today is about family planning in Sierra Leone where I worked in Freetown, the capital, some eight years ago but ongoing contacts lead me to believe that things have not changed much since then, except for the worse.

Firstly, where is Sierra Leone? On the map of Africa it is a small country just below the bulge into the Atlantic, next door to Liberia on the east and surrounded by Guinea to the west and north. Freetown is on the peninsula that projects into the ocean about halfway down the coast. Historically Sierra Leone was the first British colony in Africa. In 1787, 300 freed slaves from the United Kingdom went out to settle on the West coast of Africa in a ship fitted out by public subscription, promoted by a national newspaper, picking up 90 women from Plymouth on the way. Unhappily most of these first colonists died, but those surviving were joined by 1100 more from Nova Scotia. In 1808 the Freetown peninsula was made a British colony, largely to prevent the French from gaining access to the superb deep-water port, and in 1896 the hinterland was declared a British Protectorate.

Independence was granted in 1961 and the newly liberated colony succumbed to the advances of Moscow and in 1971 declared itself a republic. Inevitably this became a one-party state in 1978. After several years of increasing unrest there was a military coup in 1992, which overthrew the totally corrupt civilian government of President Momoh. Eventually, in March 1997 Kabbah was democratically elected President, but not for long. In May he was bloodily replaced by a junta, which remained in power until March of this year and the country continued to suffer the atrocities and destruction of civil war. A year later Kabbah was reinstated amid a great deal of extra-Sierra Leonian publicity concerning Sandline and the British High Commissioner, Peter Penfold, who is nevertheless, regarded as a hero in Freetown.

So much for its history, what about the demography? In 1990 the population was about 4.5 million and growing at a rate of 2.5% p.a. Child mortality was 366 per 1000: nearly half the population was under fifteen, which was the average age at first pregnancy. The expectation of life for men

was 46 years and considerably less for women at 38 years, largely due to the very high maternal mortality rate. No accurate figures are available but haemorrhage, sepsis, obstructed labour and septic abortion are common. Three out of the six teachers at a large primary school beside one of our outreach clinics died in childbirth during the year I was there – these were educated women within reach of the maternity hospital. Female genital mutilation is widely practised. Clitoridectomy is the norm for Muslims and Animists and even for poorly educated Christians but infibulation is practised in the North. It is culturally promoted by women through their secret societies, or Bundu.

I was invited by Marie-Stopes International to go as a clinical medical officer to their project in Sierra Leone. MSI's policy is primarily to promote family planning and better health care for women and it aims to use local personnel. Each project should be self-financing within two years and the Main Clinic in Freetown achieved this while I was there. Unfortunately several attempts had been made to employ local doctors but after sending them on special training courses abroad each had left Marie-Stopes and gone back into private practice, as all doctors did, whether in government employ or not.

General health services were found to be so poor that polyclinics were set up which included family planning. The first was opened with a grant from the EC in 1988. By the time I arrived in 1990, nine clinics had been established, seven were employment based in police premises and the Port compound. All were in Freetown or its environs. In 1991, two up-country centres were established but one at Segbwema in the Methodist Hospital 150 miles away, was only open a month before rebel activities (three Lebanese were decapitated in a nearby town) forced the staff to flee through the bush. The other clinic closed for similar reasons a year later. However a maternity unit was established in Freetown, which has been successfully functioning ever since.

I found my role initially was as a GP in the polyclinics. I had no experience in tropical diseases and had never done paediatrics but I had an excellent textbook of therapeutics and, in addition to eighteen years in general practice, I had six children of my own. I was also trained as a medical student in the use of my five senses (although I never used the fifth to taste the urine). This was a great advantage as, apart from Xrays, used very sparingly because the people could not afford them, we had virtually no modern aids to diagnosis.

Family planning was carried out by nurses far more effectively than I could do it but it did not take long to realise that their basic training was not only inadequate but also factually incorrect. They had a touching faith in 'foam tablets' (vaginal spermicides), they knew about the diaphragm but neither they nor their clients found it acceptable; condoms were used almost exclusively by men to protect themselves from venereal diseases and AIDS. The combined oral contraceptive pill(COC) was popular and well promoted, sensibly packaged to include seven iron containing pills during the 'pill-free' week; progestogen only pills were unheard of ,while intrauterine contraception was more popular with the nurses than the women. The injectable, depoprovera, was used very sparingly.

No woman was started on the pill unless she could demonstrate that she was menstruating as it was thought that if she was already pregnant any problems could be blamed on the contraceptive. Consequently there was a high drop out between the first two visits. Many of the patients were illiterate and instructions had to be simple. Few could read a calendar but they always knew how many days or weeks or months had passed since they had had a period. They would count on their fingers and periodically raise their index finger and their eyes heavenward, presumably to the full moon, to indicate the passage of a month. There were many simple clinical situations which were a complete mystery to both the nurses and the Russian doctor who worked part time in the Main clinic. I found to my horror that she treated mid-cycle bleeding on the pill (almost invariably due to forgetting a tablet or taking it late) with ergometrine followed by a D&C if it did not respond!

Depoprovera, a three monthly injection, was only used for women over 35 who had at least four children, by which time many would not need contraception as their husbands would have a younger wife! The medical hierarchy in Freetown was much influenced by American propaganda directed against this product, which is, in fact, remarkably free from life endangering side-effects. It took me a little time to encourage the nurses to offer it to their patients, especially those whose husbands wanted them to conceive again as soon as they stopped breast feeding. The staff would collude with the women explaining their amenorrhoea as "women's troubles"

We were provided with the worst possible type of IUDs, which had to be individually loaded by hand. The family planning nurses all had to be trained midwives and they enjoyed a great deal of job satisfaction from inserting them but they had no idea that a non-pregnant uterus might actually be retroverted! Women rarely presented themselves for ante-natal care before the sixth month and some left it to the eleventh hour. One lusty young man was delivered, amid great rejoicing, on the examination couch and was subsequently named Mario to commemorate his place of birth. The only problem was that our scale of fees did not include childbirth at that time.

Breast-feeding was THE MOST IMPORTANT method of family spacing that was practised. Nearly all babies were suckled for at least 14, often 16 months and intercourse during this time was taboo (or tambu as it is called in Krio). Naturally polygamy was a direct result of this as was noted by Mary Kingsley in the eighteen eighties when she explored West Africa and inveighed against the importation of dried milk from Europe at that time! There were various local devices to prevent pregnancy. One of our nurses from up-country showed me her "rope", a grass string she wore round her waist with several little packets attached to it at intervals. One contained some blood from the umbilical cord of her first child, another some of her husband's semen; the others contained equally efficacious materials. Whatever they were, they worked. When she wanted to conceive for the second time she just threw the rope into the river and she was pregnant the following month!

Sterilisation was almost unknown and certainly not popular in 1990. I was asked to give a paper on depoprovera at the symposium for the AGM of the Sierra-Leonian Medical and Dental Association. During the course of that morning's session a gynaecologist spoke briefly about vasectomy. It was obvious to me that he knew little about it but was prepared to answer questions at the end. "How long is it before it becomes effective!" he was asked by one of the fifty males in the audience (There were only three women present) "Oh, about two-three days". I caught the chairman's eye. He nodded. "Well, I am sure that is true in Sierra Leone but it takes between 18 and 24 ejaculations and in the West that would take about three months" .The audience laughed as only Africans can and it took several minutes to restore order. No doubt my white hair and grandmotherly appearance only served to heighten the joke. I asked how many of those present would consider having a vasectomy. Three hands were raised. "When my youngest wife is past the menopause" "If I was paid a million leones", "When I reach my seventieth birthday". If these are the views of educated professional men, what hope is there of influencing the general population to look on it with favour?

M-SI wished to encourage female sterilisation by mini-lap and sent a doctor on a special course to learn the technique. She enjoyed the overseas experience and the week she was given being entertained in London but she never performed a single operation in a Marie-Stopes clinic in Sierra-Leone. The doctor apart, it was quickly apparent that it was not going to be easy recruiting patients. Three were lined up for a visiting gynaecologist, two defaulted and the third was nearly forty and had not conceived for four years.

One of the ironies of my work was that I had been employed to boost the family planning side of the project and indeed, overall attendance did go up after my arrival in July 1990, as did the use of depoprovera, but in practice, it was the infertile who were most anxious to consult me. Women who did not have children were at the bottom of the pecking order, despised and usually exploited. I came across many tragic cases and a few which, at first, I could hardly credit. A female sergeant in the police was being prescribed clomid to induce ovulation by a gynaecologist who had himself removed her uterus. He also owned the pharmacy from where she was buying the drug. The cost was half her weekly wage. A school-teacher had had a caesarean section for obstructed labour when she was eighteen. The baby was stillborn. The rotten catgut broke, the incision burst open and became infected. She was five months in hospital and was lucky to survive as the wound granulated up from the bottom. She had a grossly puckered abdominal scar the size of a fist, which moved visibly when the cervix, which had survived, was manipulated vaginally. She also was prescribed clomid, by a different gynaecologist, to help her conceive. It was not all doom and gloom. A healthy twenty three year old had not conceived after two years of marriage. It transpired that she had had a termination when she was a school girl. Inquiry by the nurse as to who had performed the operation revealed the name of a woman gynaecologist who always inserted an IUD

postoperatively at the request of the parents but without telling the patient. An ultrasound revealed the hidden device and with the help of a special instrument I had brought out from home, I was able to remove it. She conceived the next month.

My time in Africa helped to define some opinions I had already formulated rather loosely before I went. Firstly, what factors govern the provision of family planning in the third world? Government policy is of over-riding importance but what determines that policy? Obviously religion and culture are extremely important. It will be impossible to introduce contraception to Afghanistan while the Taliban are in power. Some governments want to encourage a high birth rate for political reasons but many do not take a strong line and are willing to let NGAs run clinical services alongside their own, often very poor ones, but the politicians are advised by the medical establishment in their own countries. These doctors are almost invariably male, elderly, out of date and conservative and have vested interests in maintaining the status quo. The acceptability of family planning by the general public is governed by very different considerations. The standard of living is the key. If you are merely subsisting, today's rice is all that interests you, what happens in nine months is irrelevant - In Freetown most acceptors had some regular source of income and most could read and write. The education of women is crucial, especially as in most third world communities they are the ones who do most of the work. Their children must survive, their care in old age depends on having family to support them. The attitude of men both to women and to their own fertility can be an almost impossible barrier. One policeman told me that he would only agree to his wife having the pill when she had given him ten sons!

Attitudes of both men and women only change gradually and FP agencies can only have a marginal effect but they can increase the availability of services and give women a choice of methods, which may encourage them to try one. A friendly atmosphere promotes further visits and the continuation rates rise.

Finally, what is the role of the doctor? I believe their prime function lies in setting standards and leading effective and relevant training. Hands on face to face consultation is done far better by properly trained female nurses who speak the local language and have empathy and understanding with their women clients. In these days of mobile phones each nurse-staffed clinic could be linked to a medical officer for consultation in difficult cases. Family planning nurses or even nursing auxiliaries could be trained to use a check list, take blood pressures (easy now), and give intra-muscular injections. This assumes that IUDs are not part of the programme. These devices are costly to implement and potentially dangerous and I think the third world should do without them. I was interested to see that the uptake of this method in Freetown was somewhere about 5%, very similar to that in Glasgow.

As I stated in the final paragraph of the book I wrote about my experiences, I have no illusions about 'doing good'. I learnt as much about life and humanity as I taught about contraception. I have little faith in the long-term effects of Western involvement in Africa but Marie Slopes Sierra Leone needed a doctor in 1990 and I was available. One can only do the work in front of one on a day-to-day basis, each patient deserves one's whole attention and skill. If this unspoken message is transmitted to the staff with whom one works then, in a sense, one leaves a legacy. But in the chaos of post-colonial Africa, that inheritance is fragile indeed.

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THE ONE HUNDRED AND FIFTY THIRD ORDINARY MEETING

The One Hundred and Fifty Third Ordinary Meeting was held in the Buchanan Suite of the Royal Concert Hall, Glasgow. Two papers were read, one by Dr John Burnett on Poverty, Milk and Tuberculosis in Glasgow 1900-1935 and the other by Professor Ronald Girdwood on Personal Wartime Research Activities in India and Burma. Dr Burnett had stepped in at short notice to cover the absence through illness of the arranged speaker and the Society expressed its gratitude to him.

POVERTY, MILK AND TUBERCULOSIS IN GLASGOW 1900-1935

Introduction

By the last decade of the nineteenth century, the public health movement had made huge advances, and in many areas of activity its challenge was not to find ways of combating disease, but to harness the political will to make the necessary changes in housing, water supply and sanitation. Minds were therefore moving on to problems such as food and diet, and the one major disease with which little progress had been made, tuberculosis. At the same time, bacteriological studies of individual diseases, started in 1876 by Robert Koch's discovery of the anthrax bacillus, were making diagnosis a more useful basis for action. In 1882 Glasgow's Medical Officer of Health had said that he knew of no deaths in an outbreak of anthrax, but some might have been misreported as 'brain fever' or 'acute pneumonia'.(1) Twenty years later this kind of misleading vagueness was less common.

Koch discovered *Mycobacterium tuberculosis* in 1882, and showed that tuberculosis was a contagious disease. Soon it was accepted that several apparently different diseases were caused by the same organism, including consumption (or phthisis), scrofula and lupus. It was becoming clear that TB could be communicated through food, and particularly through milk, and by 1914 TB specialists agreed that most cases of TB in the stomach, neck glands and throat had been transmitted by milk.(2)

In the period under discussion, three notable men were Medical Officer of Health for Glasgow. James Burn Russell (1837-1904) took up the post in 1872.(3) He manoeuvred the Town Council so effectively that Glasgow's public health strategy was admired even in America. After Archibald Kerr Chalmers (1857-1942) was appointed in 1897 he was joined first by a Veterinary Officer and then in 1899 by Robert MacNeil Buchanan, a Glasgow graduate who had studied under Koch, as the city's first bacteriologist. Chalmers was succeeded in 1925 by (Sir) Alexander Macgregor.

Turning to the question of milk supply, the minister of Campsie had written in 1793: "It is of the utmost importance to the state at large, that the children of tradesmen and sedentary people should be healthy: I know of nothing more calculated to promote that end than fresh milk."(4)

Only a century later did the state agree with him. The Boer War (1899-1902) required the largest military recruitment for a century. The army was shocked to find that it had to reject a significant proportion of volunteers because they were unfit, unhealthy or simply too small. In some industrial areas, the proportion reached 40%, and military planners made politicians wonder whether Britain had a sufficient supply of cannon fodder to fight a large war. The need was identified for healthy babies who would grow up into healthy soldiers, and milk was identified as the most important food for children.(5)

Yet dirty milk was a well-recognised carrier of disease. Russell had addressed to farmers a pamphlet *On the Sanitary Requirements of a Dairy Farm* (1889). He said that a dairy byre should be well-ventilated, well-lit, clean, with lime-washed walls, and that it should not be overcrowded. Many small farms had unheated byres in which the beasts huddled together for warmth: Russell pointed out that this made the transmission of TB easier. He told farmers that milk from diseased cattle should not be sold. Wooden barrels were in Scotland the normal means of transporting milk: Russell drew attention to the difficulty of cleaning them, and recommended metal vessels. He asked farmers to separate milk production from any possible source of disease. "Explosions of enteric fever and scarlet fever have been traced to the washing-day, when soiled clothing has been brought to the boiler-house, or washed opposite the milk-house door."(6) Russell was trying to persuade farmers to act differently. Glasgow became in 1890 the first city in Britain to take powers to prohibit the sale of milk "dangerous or injurious to health."(7) Those powers controlled the product, but not bad practice beyond the city boundary. Russell could only try to persuade.

Developments after the First World War

Before the War, the government had been about to take action against bovine tuberculosis passing into the milk supply. The Milk and Dairies (Scotland) Act was passed in 1914, and the Tuberculosis Order which had been circulated that summer was about to come into force, but both were suspended on the outbreak of war: Britain needed all the nutrition it could muster, even if the food was diseased.

The 1914 legislation had been generous in its treatment of the farmer. For example, it required cows to be removed from the milk supply if they had signs of TB in the udder, or if they were suffering from tuberculous emaciation, but milk could be sold from other cows that were known to be tuberculous.

After the end of the War there was a period of economic instability which saw food prices collapse, and agriculture entered a severe depression that lasted until the mid-1930s. Many farmers within reach of the cities took up dairying, which supplied them with a monthly income if not much profit; and the increased supply brought a fall in the prices of dairy products. One consequence of the food crisis was that attempts to improve the quality of food were again delayed. Late in 1918 the Corporation discussed a return to the pre-war practice of seizing all meat infected with tuberculosis and destroying it: they realised this was not possible, and discussion on how to make milk more hygienic was deferred "in view of the present circumstances". A few months later they were able to go back to the pre-war practice of seizing all tuberculous meat and destroying it.(8) Milk remained a problem.

The Second Conference of the International Union Against Tuberculosis was held in London in July 1921. Chalmers and Buchanan saw it as an important step in reawakening interest.(9) Like other large local authorities, Glasgow Corporation put pressure on central government to bring into operation both the 1914 Act and the Tuberculosis Order. In 1923 the Corporation went further and advocated the strengthening of the legislation so that it applied to all cows with TB.(10) The legislation of 1914 was finally brought into action in 1925.

Milk was not equally available to all Glaswegians. In the winter of 1919/20, the Ministry raised the price of milk over the whole country. In the poorest areas of Glasgow such as Mile-end the consumption immediately fell by a third despite there being a surplus of milk on the market in the city. A Welfare Nurse who worked in the Calton said "the very poor here never use milk in the quantity they should, but give the infants tea with toast soaked in it." Porridge made with milk and milk puddings had been given up, and rice with milk at midday was replaced by cocoa. Nurses reported that mothers understood the importance of milk for babies, and tried to make up for lack of cows' milk by breast feeding as much as possible. In contrast, the middle classes could afford the milk they needed, and they were able to pay for higher standards of hygiene. Bottled milk had been sold in Glasgow before 1914, although most milk was still being sold from open cans in 1920.(12) When a system for milk grading was introduced in 1923, the milk from tuberculin tested herds was perceived to be the best and was the most expensive. At this point the effectiveness of pasteurisation equipment was still questioned so the milk which was guaranteed to be free of tuberculosis bacteria was sold only to those who could afford it.

The interested parties

By 1925 a certain amount of progress had been made in developing methods for reducing the number of human and animal sufferers from TB of bovine origin. Yet little progress was being made in practice because the situation was extremely complex and until it was simplified there was little chance of a large, practical problem being solved. In addition, milk producers and public health workers had little interest in understanding one another's points of view.(13) In order to understand the situation more clearly we need to look at the views of the four central groups.

The dairy farmers were concerned with staying in business, not with TB. The two Scottish farming journals, the *Scottish Farmer and the North British Agriculturalist*, were almost completely devoted to events on the farm and to farming life. There was scarcely a mention in either of them of national or local issues of food supply, or of the quality - rather than the quantity - of food sold. Neither was there any discussion of the particular needs of the city, especially the enormous conurbation of Glasgow.

In 1920 an English farmer went on a lecture tour to Ayr, Glasgow and Edinburgh on behalf of the National Clean Milk Supply Society and said that "The danger from dirty milk was much greater than from tubercular milk."(14) He advocated cleanliness in byres and in distribution, but not public health controls. This shows two things concerning the farmers at this period: most accepted the need for cleanliness to fight disease, but did not recognise the dangers of TB, and they deeply resented interference from public health officials. One angry individual said that, If any more rules and regulations on dairying came into force he would stop dairying.(15)

Neither had milk suppliers, the second group, been much concerned with the quality of their product. Typically they had been small operators who had a poor reputation for cleanliness, and they often watered the milk.(16) In the 1920s larger firms were rapidly taking over the milk supply of British cities. Although it has been possible to study the situation in London in detail, there appears to be insufficient evidence to give an adequate account of what happened in Glasgow. It is certain, however, that the larger firms had the capital to introduce pasteurisation. Yet even where there was a commitment to pasteurisation, there was uncertainty as to which of the methods was the most effective. Problems continued to be caused by the use of inadequate techniques of pasteurisation and the mixing by wholesalers of tubercle-free milk with milk of less certain quality. By the late1920s the larger distributors of milk were installing "positive holder" pasteurising plants, which heated the milk to145-150 F for 30 minutes, passing it directly to modern bottling plants which sealed the filled bottles, but smaller retailers had inferior equipment, or none at all.(17)

The situation in Scotland was radically different from that in America. In New York, Public Health Inspectors were in 1902 sent into the surrounding countryside to encourage the farmers to improve the hygiene of milk production, and in 1910 all drinking milk in the city was required to be pasteurised.(18) Such legal powers were not available in Britain, and milk remained an effective transmitter of tuberculosis to children in Glasgow. The life insurance industry encouraged the drinking of milk because it was healthy, and the prohibition of alcoholic drink and the aggressive marketing of milk combined to increase consumption. The *Scottish Farmer* suggested that 'The farmers' slogan for good times might be, "pasteurization of milk and prohibition of liquor."(19) There were also doubts as to whether it might in some way lessen the quality of the milk by destroying some (unspecified) part of its nutritive quality. For this reason, pasteurisation was, according to farmers, "a defensible expedient under existing circumstances."(20)

Glasgow Corporation was concerned about TB for two reasons: first, they were responsible for public health, and second, the isolation of patients, and sending some of them to distant sanatoria, was expensive.(21) The place in which the important discussions took place is significant. The Corporation had a sub-committee on tuberculosis: its main role was not to deal with matters of policy, but to ensure that sanatoria had adequate funds and facilities. The will to solve larger problems and to find ways of making beneficial changes was at a higher level. The Medical Officer of Health, the Bacteriologist and the Veterinary Surgeon all had access to the Committee on Health, and their chief concerns were discussed by the Corporation itself.(22) They strenuously used all the powers they had – but the powers were limited.

Glasgow was able to insist that milk cows that were being tended in the city were slaughtered if they were suffering from tuberculosis. But as in Russell's time the city's powers did not extend beyond its limits. When cases of "sore throat"- diphtheria – in the city were traced to a dairy in Dunbartonshire, Glasgow could take no more action than to say that no more of its milk should be sold in the city.(23) Local authorities in milk-producing areas were reluctant to act against poor farming practice for fear of antagonizing the farmers who were ratepayers and voters. The Public Health Act 1897 had given powers to local authorities, but many chose not to exercise them: Ayrshire was still holding out against dairy inspection, on cost grounds, in 1925.(24)

These were the four groups whose views shaped and hindered action against TB. There were several others that for one reason or another had little impact in this period. National government continued to believe that public health was a local matter. Professional bodies such as the British Medical Association and the Royal College of Veterinary Surgeons could press for action, but all of their recommendations required significant expenditure. A variety of charities, from the local to the national, concerned with tuberculosis and poverty, or child health, could effect only minor amelioration. No one had sufficient power to make real change.

Later events

The move towards a sufficient supply of disease-free milk over the whole of Britain took place between 1930 and 1960.(25) The new factor that made it possible was the creation of the Milk Marketing Boards. The Scottish Milk Marketing Board was established in 1933 (with smaller boards covering the Aberdeen area, and the north of Scotland).(26) In 1934, only 393 herds out of about 8,000 in Scotland were free of tuberculosis, but four years later there were 2,300. The Board bought milk at a guaranteed price that depended on its bacteriological quality. In 1930 USA started to

import cattle only from Tuberculin Tested herds, and this was a major incentive to farmers with Ayrshire herds.(27) By 1946 over 4010 herds in Scotland were TT.(28) The change had been made through the creation of a national structure in which scientific and medical knowledge could be applied. One might see it as a demonstration of one of the reasons why a National Health Service was needed.

Acknowledgements

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PERSONAL WARTIME RESEARCH ACTIVITIES IN INDIA AND BURMA.

Like almost all the 140 male students, some from overseas, who graduated in Medicine in Edinburgh in 1939, I went to an office in Drumsheugh Gardens and signed up for military service before war was declared: the records show that 102 were known to have enlisted in the Services. Sadly seven were killed, while two became prisoners in Japanese camps and one was captured in Germany.

Having by mistake been at the immense Nuremberg Nazi party rally with a fellow student in 1937, (because we did not speak German and did not know before we went that it was taking place), I had little doubt then that war was coming soon. Chamberlain's journey to Munich in 1938 did not provide reassurance. In 1939 I resigned from the posts in surgery and neurosurgery which had been allocated to me and hence my only house post was a medical one.

My early years in the Army were for the most part spent first in Yorkshire and then in Sussex, but in 1943, having applied for an overseas posting, I sailed from Greenock for an unknown destination, there being six of us, all medical officers, crammed into a two berth cabin. We survived two attempted bombing attacks at sea, the ship next to us sustaining a direct hit with 1115 casualties in the first attack, and after a short stay in a tent at Port Tewfik in Egypt, set off again, this time for Bombay. My posting now was to a tented hospital at Deolali beside the holy city of Nasik, the term "Deolali tap" being commonly used to describe a form of madness. This was where the British troops who were being repatriated were assembled, and nearby the 19th Indian Division, which later relieved Mandalay from the Japanese, was forming up. Needless to say we were all kept very fully occupied, although some of our wards collapsed when the monsoon came in July.

Other problems were mosquitoes, scorpions, kitehawks, vultures and howling jackals and it was always necessary to take precautions to avoid malaria, bacillary and amoebic dysentery, cholera, hepatitis, typhoid and poliomyelitis. Snakes could be a danger and monkeys sometimes entered railway carriages or huts and might snatch belongings. A group of men who had arrived from Bombay by train were marched a few miles to the camp and more than twenty had to be admitted with heat stroke or heat exhaustion. Two died despite all efforts to save them.

But now my activities unexpectedly became much altered as a result of the interest in haematology which I developed as a house physician in Stanley Davidson's ward. Although sternal puncture had been attempted in 1927, it was not until about the time of my graduation that this simple method of investigation of possible blood disorders came into common use, and while in Stanley Davidson's wards I performed many sternal punctures and reported on the marrows. A medical brigadier visiting Deolali learned that I had had this experience and had brought the needle with me, so immediately arranged for me to be attached to GHQ (Delhi), because there was considerable concern about an unknown disease which produced severe anaemia, diarrhoea, glossitis and wasting in Indian troops, apparently mainly in East Bengal and Burma. Sometimes the condition was fatal. In 1943, one hundred and twenty men had been evacuated with medical or psychiatric conditions for every one suffering from bullet wounds or other surgical conditions. Mepacrine had helped to control malaria, but the form of anaemia which I was to investigate was not like that encountered in malaria and was not understood.

In Delhi I studied the records for a short while in a temporary headquarters building within sight of the Viceroy's House, now the President's, which had been completed by Lutyens in 1930 on a site which he had chosen in 1912. I slept in a tent by the roadside where at times I could see the Viceroy, Lord Wavell, riding by. It was decided that I would be called a Nutrition Research Officer and I was given freedom to travel to any part of India where the disease was prevalent, arranging my own postings, always of course notifying GHQ. My equipment was modest, consisting of the sternal puncture needle, red and white cell pipettes and a Sahli haemoglobinometer. In this last a pipette is used to add a measured amount of blood to decinormal hydrochloric acid, which is then diluted to match a standard.

After I left Delhi, I checked the apparatus by doing blood counts on the medical staff (mainly European) of the first of several hospitals to which I had chosen to move. This was Sirajgunj, on the west bank of the Brahmaputra, on the direct line of evacuation from the eastern front to central India, and I was surprised to discover that all the results were abnormally low. This I discovered was because the large bottle labelled hydrochloric acid with which I had been supplied actually contained nitric acid and this gave completely invalid results. I disposed of it and, on checking

found that some other hospitals in India had also been issued with the wrong acid. In the early stages I found some evidence of the disease which I had come to investigate amongst the troops being evacuated to central India but it was no longer of epidemic proportions. However some such patients arrived at Sirajgunj and the marrows confirmed the suspicion that they had megaloblastic anaemia so I arranged through Delhi for crates of what was then called "crude" liver extract for injection to be sent out to me from the U.K.

There were several such preparations then available, but I did not use *Anahaemin* which was said to be a purer extract. I avoided using it since it was not then known what substance in liver was active in the treatment of pernicious anaemia or other forms of megaloblastic anaemia, such as the so called pernicious anaemia of pregnancy. Vitamin B12 and folic acid were not yet known and the substance required to treat the disorder which I was investigating might be something that could have been removed in part or entirely during the preparation of *Anahaemin*. The ampoules supplied to me were of *Campolon*. As controls, I carried out haemoglobin estimations in a month on capillary blood from 500 Indian patients in hospital who were being evacuated from the fighting front and did not have features to suggest that they were suffering from the disease which I was seeking. In them severe anaemia was not a problem other than that caused by hookworm infestation or malaria. Mepacrine was now supposed to be taken by all troops before and during the time they were in the fighting areas to prevent malaria, but many had been infected before they enlisted. I soon realised that I was not going to make much progress with my investigations in Sirajgunj because of lack of suitable patients and I travelled widely in my quest, making 29 moves by train or river steamer in ten months, including reporting at intervals back to Delhi.

In June 1945, however, the disease appeared again at Dhaka, in those days a town in India but now the chief city in Bangladesh, a country with so many rivers that there is frequently severe flooding. I was given a special ward to enable me to collect together during a period of six weeks a group of patients whom I thought should be investigated because of their clinical features. Marrow puncture was carried out in 96 of them and megaloblastic anaemia was found in 62. This usually responded to crude liver injections, but such was not always the case. Certainly undiagnosed malaria was a complication in some instances, with the parasites frequently appearing in the peripheral blood when the liver injections were given, perhaps a form of "shock therapy". It has to be realised too, that some of the men may have been suffering from a degree of malnutrition, although this was not an evident feature. In some instances the anaemia was very severe, necessitating blood transfusion to be carried out at once. The lowest haemoglobin level was 2.8 g/dl and red cell count 395,000 per cu mm while 19.75% of the cells in the marrow of this patient were megaloblasts. Blood transfusion was essential.

Although it had always been claimed that sprue did not occur in Indians, my conclusion was that this was the disease affecting most of those men and I reported this to G.H.Q. The active substance in the liver injections was not known and hence the amount present could not be assessed. No British or West African troops in this area were found to be affected with the clinical features of sprue. However it was reported in 1945 that in an R.A.F. unit in Chittagong, which is down the coast nearer to the Burmese border than the area where the troops whom I was seeing had been operating, 10% of British men developed sprue within three weeks of arrival and, of four Italian prisoner of war camps in northern India, sprue occurred in one and was quite a problem. Most of the 1073 British troops sent home because of sprue between 1943 and 1946 had a remission when they reached the U.K. or even during the voyage home, but this was not always maintained.

The conclusion therefore was that tropical sprue was a disease of unknown aetiology, which occurred only in certain areas of India and at this time affected Indian troops, sometimes severely. It had some of the characteristics of an infection, but the cause was and still is unknown. I thought it was possibly caused by a virus. In Sirajgunj I had earlier been asked to investigate 71 Japanese prisoners of war in hospital, a task that was made easier because one of them was a medical officer and he was keen to help and to translate. Where there was anaemia in the Japanese POWs it was due to malaria or other causes and there was nothing in their history or clinical findings to suggest that any of them had tropical sprue, a conclusion with which the Japanese medical officer agreed. I felt that to do marrow punctures on P.O.W.s would be unethical if it was not to alter treatment. There were also 223 members of the Indian National Army (followers of Subhas Chandra Bose) who had changed sides, possibly to avoid being killed by the Japanese, and were being treated in

the hospital at Sirajgunj. They were known to us as Jifs. (Japanese Indian Forces.) None of them had suffered from a sprue-like illness. The mean haemoglobin level of the Japanese patients was 11.8 g/dl, while that of the members of the I.N.A. was 12.6 g/dl compared with 500 of our Indian troops with nothing to suggest sprue, whose mean haemoglobin level was 14.18 g/dl. The difference between the figures in the prisoners and our troops reached a statistical level of significance. (It should be added that most of the Jifs were treated leniently when they reached central India, some even returning to their former Units.)

After Dhaka I was told by GHQ that I was to go to Burma to carry out further investigations along the lines that I had been doing, still seeking the condition which I believed to be sprue. I had become friendly with a nursing sister from Cornwall in the course of my visits to Sirajgunj and she was now working in an Army hospital in Calcutta so, as I was passing through I sought her out and suggested that we might become engaged; I was delighted when she agreed and, having purchased a ring, I had to speed off, but less than two weeks later I was passing through Calcutta again, fortunately arriving on a Sunday morning, so when I suggested that we should get married we dashed to the church and had the banns called. A dress was made the following day and on the Tuesday we were married, our guests being members of the hospital staff plus a few officers whom I knew and found in the dining room of the Grand Hotel where our reception was held. At that time because of instances of bigamy it was necessary to obtain a certificate from one's C.O. confirming that one was not married already, but as I did not have a C.O. I signed it myself. After a night in the Grand Hotel (during which somebody was murdered in the room next door) we had difficulty in obtaining accommodation, but became the first occupants of new Y.W.C.A. married premises.

On 6th August, 1945 we were still there as I awaited transport to Burma and learned that an atomic bomb, something of which we had not heard, had been dropped on Hiroshima. To us the importance of this was not appreciated, and I continued, successfully as it turned out, in my efforts to sail to Rangoon. I was probably the only officer to successfully claim hardship allowance for delay in transit when in fact a wedding was included in the delay. I sailed on 11th August, taking my stocks of liver extract with me, this being the day after the second atom bomb was dropped, on Nagasaki, and three days later we heard that Japan had surrendered unconditionally.

In Rangoon I was attached to the one hospital which admitted British patients and, later, Australians and Dutch but not Indian servicemen. In any case it soon became clear that I would have to cease my quest for patients with tropical sprue. Many Japanese troops were not aware that the war was over and in some areas fighting was still going on. Indeed this continued in some instances even after Admiral Mountbatten had accepted the formal unconditional surrender In Singapore on 12th September, 1945.

However men were now being released from Japanese POW camps and I dropped everything else to assist in the medical care of those who had survived the camps but had to be admitted to our hospital, the most seriously ill being retained in hospital in Thailand. Most were suffering from severe malnutrition, and only investigations which might convey benefit could be contemplated, but I had charge of wards through which there passed 180 men, 77 of whom were selected because they had amblyopia due to malnutrition. There was no tropical sprue but of course most had had frequent attacks of malaria. We had an expert ophthalmologist in the hospital and he assessed the visual acuity of those men with visual difficulties. I then gave them injections of crude liver extract together with mepacrine, multivitamin tablets and thiamine hydrochloride. Re-examination of their vision showed that 47% showed slight improvement of visual acuity after six days of treatment and a further 20% showed a more substantial improvement. Once again it was thought that some factor in liver contributed to this, but what it might be was unknown.

The stories told by these men were harrowing. Even some nurses had been bayoneted. One man showed me a radio which he had built into a water bottle; had this been discovered he might have been executed. Complete records could not be kept, but it is believed that between prisoners-of-war and impressed Burmese and Malay labourers, some 80,000 to 100,000 perished in the forced building of the Burma-Thailand railway. One patient told me that they hollowed out the supports for the bridges and introduced ants to ensure that they collapsed. By 20th September 30,877 Allied ex POWs were reported to have sailed from Rangoon and Singapore in more than 60 ships. We treated men with illnesses that now included tick typhus and smallpox.

Unexpectedly I was made entertainments officer in addition to my official duties and I remembered that in one previous attachment there had been a dance which included a competition in which the nurses were asked to leave the floor progressively according to the number of garments they were wearing, apart from shoes. I had not been surprised when those wearing more than three garments left the floor, but when I announced a similar entertainment in Rangoon I was amazed by the results, admittedly it was a very hot climate!

Moreover in addition to my clinical duties I was detailed to write and then deliver, at 21.15 hours from Radio Rangoon each Wednesday, a broadcast which dealt with medical matters but was of a propaganda nature. I had some contact with the clandestine Force 136, but they did not monitor what I wrote. Instead the text was sent to Admiral Mountbatten's headquarters in Ceylon for authorisation, but no changes were made. As the time passed the numbers of patients were still considerable, but the war was of course over and there was a general desire amongst the staff to return home; I was asked by 12th Army Headquarters to write a morale report and it made gloomy reading. However Christmas was coming and I was asked to arrange an appropriate entertainment. This I did in conjunction with Lt Col John Stokes, a physician from University College Hospital, in that I contributed most of the words and he wrote the music for a show which took place on 27th December.

The next day I had a phone call from Headquarters to inform me that all leave was to be stopped for the time being and if I wanted to see my wife soon I should leave for India. On that afternoon I managed to obtain a seat on the floor of a Dakota, which was just about to leave Rangoon for Calcutta, and we had a short holiday at the seaside resort of Puri in Orissa. In that same week some British troops who had gone to a cinema in Calcutta to see the film *Objective Burma*, with Errol Flynn as the star, began to smash up the seats, because the film gave the impression that the relief of Burma had been a purely American success. In fact most of our men had never seen American troops, who had mainly been with the Chinese under the American General Stilwell, (who had no love of the British), in the far-north of the country. Nevertheless there were men of the American Field Service fighting in various areas of Central Burma such as Meiktila, which is just to the south-west of Mandalay, itself 350 miles north of Rangoon. In addition there had been much cooperation between the RAF and the USAF.

After a few days I had to fly back to Rangoon, but there was a potential problem which was not widely known until, on 21st March 1998, a television programme entitled "Mutiny in the RAF." was shown in Britain. This revealed the dissatisfaction which came to a head at the end of 1945. There were 60 RAF airbases in the Far East in which were stationed many men who naturally wanted to go home now the war was over, but John Strachey, Parliamentary Undersecretary of State at the Air Ministry, had argued in October 1945 that for our foreign policy we required a small Army and a relatively large Air Force. This was no doubt a factor that led to an Air Force strike of some 2000 men on 2nd November 1945, this lasting only 4 days. I was, however, lucky in returning to Burma when I did, because the strike (which the Service chiefs sensibly preferred to call it) soon became a very large one, affecting many men below the rank of sergeant. Only four airmen were arrested, one receiving a long sentence which later was cut from 10 years to 22 months.

I was now officer in charge of a medical division in Rangoon, but it was past the authorised time for my release. A new problem had arisen in that, although an officer was available to take over from me, the regulations said that an officer in South-East Asia Command (which I now was) could not go home through India Command, which is where my wife was an Army nurse. I could not find anyone to sign an authority for me to go via India, so signed it myself and on 8th February sailed for Calcutta. (I could not easily obtain a flight because of the RAF 'strike') When I arrived in Calcutta I found that it was out of bounds to British troops in uniform because an anti-British 'Quit India' agitation had started and in addition there had developed strikes in the Indian Air Force and Indian Navy. However a truckload of Gurkhas came past and cheerfully took me to my wife's hospital at Alipore. Here we had no difficulty in negotiating her release and set off on the three day train journey westwards to Deolali. I was put in charge of a shuttered train which was going to Bombay, the fear being that it might be attacked, so apart from my wife and me all those on board were armed. We sailed to Greenock where we were not allowed to disembark because it was thought that there was a case of smallpox on board. As I had advised, it was chickenpox!

In 1963, Edinburgh University arranged with the medical college of Baroda in the state of Gujerat on the west side of India, north of Bombay, to give assistance under an agreement funded by the WHO and the Government of India. By this time folic acid was freely available and I had developed a differential folic acid test to help in the diagnosis of sprue, the urinary outputs of folic acid after 5mg by mouth being compared with that after 5mg by injection. In the normal person the outputs were about the same, but not so in sprue. In addition jejunal biopsy was now a simple procedure. During two periods each of a year, members of my Departmental staff went to Baroda with their families to learn about the diseases of India, but equipped to test for sprue; however no patients were found with evidence of the disease.

I went there in 1965, then went down to Vellore in south India, 90 miles to the west of Madras. A team there headed by Prof. Selwyn Baker was also looking for patients with sprue, but without success. It so happened that at this time there were language riots because Delhi was attempting to make Hindi the only official language in India. It is not understood in the south where Tamil and Telegu are amongst the main languages and there was much anger. I managed to board a train which was going to Bangalore which has an airport. An attempt by rioters to derail the train was unsuccessful and soon I was home again.

In the Services we had no problems about the different cultures of Hindus, Moslems and Sikhs provided we respected their dietetic traditions, but serious trouble was to follow soon after most of us had left India. When Partition came on 15th August 1947, there was a very major shift of Moslems to the states which had separated from India (then divided into West and East Pakistan) and of Hindus from the Pakistans to India. It was reported that there were some 12 million refugees, and trains loaded with the corpses of those who had been murdered arrived in India and Pakistan. It was said that the dead were lying like a carpet at New Delhi railway station. It occurred to me that many of those with whom we had dealings as sepoys or as patients may have been involved in the dreadful outrages which now developed. Then on 31 October 1984, Indira Gandhi was murdered by her Sikh security guard, and a fresh series of riots and killings occurred. I had treated many Sikhs and again wondered whether any of those whom I had treated lost their lives at this time.

STRENGTHS OF TROOPS IN THE FIELD IN BURMA AT THE TIME IT WAS BEING RECAPTURED BY THE ALLIES

Indian 340,000
British 100,000
West & East African 90,000
Chinese 65,000
American 10,000
47 US Air Squadrons
51 British Air Squadrons
(This included RAF, IAF, RCAF and RAAF)

Source of this information is *Campaign in Burma* HMSO 1946

THE EIGHTH HALDANE TAIT LECTURE

The Eighth Haldane Tait Lecture was held on Wednesday 5th May 1999 at the Pollock Halls, University of Edinburgh. Professor Robrecht van Hee of the University of Antwerp gave an address entitled "From Billroth's gastrectomy to laparoscopic gastric stapling: a hundred years of peptic ulcer surgery".

FROM BILLROTH'S GASTRECTOMY TO LAPAROSCOPIC GASTRIC STAPLING: A HUNDRED YEARS OF PEPTIC ULCER SURGERY

When on the 29th of January 1881, Theodor Billroth (1829-1894) performed the first successful partial gastrectomy, the bearded surgical professor was already 52 years old (1). Born in Greifswald in Germany in 1829, Billroth started his medical studies in Gottingen, together with his friend

Georg Meissner (1829-1905), the discoverer of the famous nervous plexus. In 1851 he moved to Berlin, where his clinical professors were the well-known Bernhard von Langenbeck (1810-1887) for surgery and Moritz Heinrich Romberg (1795-1873) for internal medicine.

Billroth's whole career had been devoted to fundamental research and clinical investigation, starting with his Ph.D. thesis on the pulmonary effects of cervical vagotomy in animals, and continuing during his surgical education at the Charite, with microscopical investigations at the time that Rudolf Virchow in 1855 published his "Cellular pathologie". Billroth was convinced that microscopic studies were essential to explain surgical pathologies and he was particularly interested in pyogenic wound disturbances.

He later described specific organisms, the cocco-bacteria, as being responsible for postoperative wound infections, but remained reluctant in these cases to implement antiseptic treatment with carbolic acid as proposed by Joseph Lister (1827-1912).

In 1859 Billroth was asked to become professor of surgery in Zurich and soon became one of the leading surgical authorities in German speaking Europe. He published his Opus Magnum, the "Allgemeine Chirurgische Pathologie und Therapie", which after its first edition in 1863, was re-edited several times and translated in various languages. In addition to his prolific scientific and surgical career, he found time for musical activities, which he cherished from his youth; he was an excellent piano player, studied the alt viola and became an intimate friend of Johannes Brahms, (1833-1897) who followed him to Vienna some years later (2).

In 1867 Billroth became professor in Vienna , and there began a highly productive surgical period that lifted the Viennese surgical school to one of the most important scientific centres in the world. It was not surprising therefore that in such surroundings, the first successful gastric resection took place.

We have a detailed account of this operation from the hand of one of his most talented pupils, Anton Wolfler (1850-1917) (3). After a historical introduction, relating two earlier gastrectomy attempts of Jules Pean (1830-98) (4) and Ludwik Rydygier (1850-1920) (5), Wolfler gave an anatomical description of the stomach and its blood supply. There followed an accurate account of the operative technique, starting with chloroform anaesthesia, discussing the type of incision and covering all details of resection. Illustrated by multiple drawings, Wolfler described several types of gastro-duodenal anastomosis, all developed in the laboratory by two of Billroth's assistants, Vincenz Czemy (1842-1916) and Karl Gussenbauer (1842-1903), both subsequently professors of surgery.

After discussing the pathology of the resected tissue, Wolfler's publication ends with the follow-up of the 43 year old patient Therese Heller, and of two other patients, subsequently treated the same way. Despite Mrs Heller's death 4 months later from extensive metastatic disease, the success of Billroth's operation was discussed worldwide, not least in the Deutsche Gesellschaft fur Chirurgie, one of the oldest surgical associations on the continent established in 1872, and also in the United Kingdom and the United States.

In the U.S. Billroth's techniques were popularised by William Steward Halsted (1852-1922), who spent part of his two European study years in Vienna, in Billroth's surgical laboratory and became a good friend of Wolfler (6). Meanwhile most European surgical schools had already adopted Lister's antiseptic technique with carbolic spray. The Belgian surgeon Charles Delstanche (1840-1900) published a French version of Lister's publication as early as 1871 (7). His colleague Louis Deroubaix (1837-1897) visited Lister in Edinburgh in 1879 and subsequently introduced Lister's technique in the Brussels' St. John's Hospital (7).

The introduction of asepsis and antisepsis was a great stimulus for gastric as well as other types of surgery.

Later in 1881, in Culm, Ludwik Rydygier performed another gastrectomy, but in contrast to Therese Heller, his female patient suffered a gastric ulcer, that bled recurrently for three years (8). It became the first successful operation of this kind for a benign peptic ulcer. "And hopefully the last" was the comment of a critical article in a leading 1882 journal (9). Luckily Rydygier's patient was cured and lived for several decades after bearing five children (9).

Acute ulcer perforation posed a quite different problem. By 1884 Billroth's assistant Johann von Mickulicz-Radecki (1850-1905) had recognized that sewing up the hole was necessary to avoid fatal peritonitis (9). It was not until 1892, however that a German surgeon from Wuppertal, Ludwig

Heusner (1846-1916) (10) and independently, an English surgeon from Reading, Hastings Gilford (1861-1941), did close acute perforations of the stomach (10). Some two years later, another British surgeon Percy Dean, operated on a patient in an equally emergency situation. This time however the ulcer perforation was located in the first part of the duodenum (9). At that time, the nature of these ulcers was not really understood, nor was the clinical diagnosis easy to make.

William Brinton (1823-1867) in his epochal work of 1857, "On the Pathology, symptoms and treatment of the Ulcer of the Stomach" claimed that a minimum of clinical symptoms be present to propose the diagnosis of ulcer(11). He noted "unless the pain possess the characters attributed to it, unless this pain be accompanied by vomiting, and unless there be evidence of haemorrhage having occurred in the course of the malady, there is no sufficient basis for a definite diagnosis of the existence of a gastric ulcer" (11). He added humbly "But I have not the slightest doubt that absolute enforcement of this rule would lead us to overlook a vast number of cases". (11).

It was almost forty years before Rontgen's (1845-1923) discovery of X-rays in 1895 led to a real break through. Two years later, Ismar Boas (1858-1938), gastro-enterologist in the Berliner Charite Hospital, use coated bismuth tablets as darker shadows to investigate contractions and peristalsis of the stomach behind a fluoroscopic screen. The Viennese radiologist Martin Haudek (1880-1931), using a watery bismuth solution, in 1910, detected the ulcer "niche", since then called Haudek-niche as a pathognomonic sign of gastric ulcer. Soon afterwards, investigators started to develop contrast media that could not only identify benign ulcers but could differentiate them from typically malignant tumours. A first step in a really conclusive diagnosis was taken.

Von Mickulicz adapted the mid 19th century concept of the cystoscope to construct a scope for oesophageal and gastric use. The 65 cm long scope consisted of a tube equipped with mirrors. A contemporary surgeon remarked that it "requires for its successful use a surgeon with the instincts of a sword swallower and the eye of a hawk" (12). Later construction, by the firm Wolf, of convex lenses along a curved tube, induced the German clinician Rudolf Schindler (1888-1968) to make a very useful gastroscope (13). In 1956 the use of very small glass fibres, assembled in strands, allowed Curtiss, Hirschowitz & Peters to construct the first flexible fibrescope (14). The famous gastro-enterologist Bockus immediately realized its value, when in 1963 he wrote "This is a pioneer instrument and holds tremendous promise for the future" (15). These were foreseeing words!

As for ulcer pathogenesis, a decisive landmark was posed by the Russian investigator Iwan Pavlov (1849-1936) who could demonstrate that seeing or smelling food induced a secretory gastric acid response, that could be inhibited by cutting the vagus nerves. These findings, leading to Pavlov's Nobel prize in 1904, were confirmed after the first World War by the Frenchman Andre Latarjet, who was able to selectively denervate the acid producing portion of the stomach (16). Both investigators had to rely on careful anatomical studies done following the epochal work of Andreas van Wesel, or Vesalius. The rotation of both vagus nerves around the oesophagus was however, wrongly described by the Flemish anatomist, with the right vagal nerve proceeding anteriorly and the left posteriorly. This description had for several centuries been accepted by all researchers including the English physician Thomas Willis (1622-1675). In his "Anatome cerebri" Willis gave a detailed Vesalian account of the vagal branches, together with the first description of the sympathetic gastric innervation (17).

The Frenchman Jacques Benigne Winslow (1669-1760) ultimately corrected the anatomical error in 1732 (17). From then on, investigators could rely on exact descriptions of the nervous branches when performing the first vagotomies in animals. These early vagotomy experiments led to a 19th century discussion on whether vagal trans-section diminished gastric secretion, as was assumed for instance by Benjamin Collins Brodie (1783-1862), or on the contrary, induced more gastric juice, as was proposed for instance by Leuret & Lassaigne or later by Rudolf Heidenhain (1834-1897). The discussion was definitively settled by the experiments of Claude Bernard (1813-1878) and Pavlov, and these results led to a worldwide interest in peptic ulcer research.

The English surgeon Berkeley Moynihan (1865-1936) carried out an extensive clinical study in patients with gastric complaints, resulting in a detailed description of duodenal ulcer, published in 1910 (18). This led to thoughts on the type of diet to be adopted, and considerable discussion on the choice of operation, whether gastro-enterostomy or gastric resection and, if the latter, which type of anastomosis should be performed. The surgical congresses of the early twentieth century

devoted entire sessions to this subject, with distinguished surgeons quarrelling about the optimal way to anastomose the gastric stump and small bowel. This discussion lasted for several decades, practically up to the Second World War.

Meanwhile attention was drawn to other gastric disturbances, particularly in infants. As the first successful corrections of oesophageal and duodenal atresia were being documented, Wilhelm Rammstedt (1867-1963) published his results on muscular cleavage of pyloric stenosis (19). His technique of 1912 remains the treatment of choice.

A totally different and curious surgical treatment, that remained popular for more than seventy years, was introduced by the Frenchman, Francois Glenard (1848-1920). He attributed different kinds of organic and psychological complaints to the ptosis of abdominal organs such as kidney, stomach or large bowel, a condition called splanchnoptosis and later coined Glenard's disease (20). As a result, repositioning and fixation of the stomach became a frequent operation, especially in the hands of otherwise less successful surgeons.

Surgery for gastric cancer was of a completely different order. The work of Rudolf Virchow (1821-1902) in the mid 19th century irrefutably undermined the Galenic concept of cancer as an excess of black bile, and tumours were shown to grow by cellular division. Cure therefore was thought to consist of as radical excision as possible. For gastric cancer this eventually meant total gastrectomy. The Swiss surgeon Carl Schlatter (1864-1934) is credited with performing this for the first time in 1897 (21). Such an extensive operation needed particular skills when surgeons had only chloroform and ether to anaesthetise their patients, without any form of curarisation to relax the abdominal muscles (22).

A surgeon with the necessary skills was Albin Lambotte (1866-1955), born in Brussels, who worked in the Stuivenberg Hospital in Antwerp from 1890 to 1910 (23). Known worldwide from his "Chirurgie operatoire des fractures" (1913) as the founding father of osteosynthesis, Lambotte was also an excellent abdominal surgeon, performing a near total gastrectomy as early as 1894 (24).Not only did he become well known for it, but his patient, known as Mary-without-stomach, became one of the best known Antwerp citizens, living for some years after the operation (25).

Lambotte performed his interventions in the operation theatre where I worked for a couple of months, before moving to a totally new operative complex, built in the early 70's. He was a very talented surgeon, spending many hours in his workroom, specially fitted out to make his instruments and equipped with two large wheels, two big workbenches and hundreds of tools to create not only surgical instruments, but also violins, fishing reels and various other items. He made 182 violins and there are many who are proud of possessing a Lambotte in their collection (26). At the 1935 jubilee of Lambotte in Antwerp, most of his pupils and admirers came to celebrate him, including such famous surgeons as Rene Leriche (1879-1955), Ferdinand Sauerbruch (1875-1951) and Hey-Groves.

The way was now open for gastric surgeons to treat the most common disorders of the stomach. In peptic ulcer research, investigators tried to understand gastric secretion mechanisms and their effects on the causation of gastro-duodenal ulcer. By 1859 gastric hydrochloric acid was presumed by Claude Bernard to be produced in the parietal cells of the gastric glands, cells previously described histologically in 1836 by Sproth Boyd. The irrefutable demonstration of the parietal cell source of hydrochloric acid was in 1950, by Bradford and Davies. Bannister in 1966 and Alonso in 1967 developed the theory of an energy dependent, secretory pump mechanism, necessary for the active secretion of hydrogen ions or so called protons. However, as Hollander asked 1954: "Why does the stomach not digest itself?" The answer, though suspected by Glover two hundred years previously, was first given by Wolf and Wolff in 1948, when they attributed this gastric wall resistance to a viscous mucus layer, working as a barrier. In 1970 Robert and Jacobson expanded this concept of cytoprotection by proving the role of prostaglandins in the defensive mucoid barrier.

For surgeons interested in peptic ulcer however, mechanisms of interfering with the physiologic drive of this secretion were more important. Uvnas, in 1942, showed that secretion stimulating mechanisms were induced not only by the vagus, but also by the hormone gastrin. This was identified by Gregory & Tracy in 1959, and synthesised by them in 1964. Three years later, Gobbel and Benton Adkins produced experimental duodenal ulcers by exogenous administration of gastrin.

Now surgeons were able to understand why distal gastrectomy, as performed by Theodor Billroth in 1881, was really able to work in patients with peptic ulcer, namely by excision of the gastrin producing antrum of the stomach.

There was also a major interest in eliminating the vagal drive of gastric secretion. During the Second World War, the American surgeon Lester Dragstedt (1893-1975) brought the experimental technique of truncal vagotomy into clinical practice as an effective treatment for duodenal ulcer disease (27). The frequency of peptic ulcers had risen sharply in those years, mostly affecting young men and Dragstedt & Owens wanted to avoid resective procedures. However, they soon understood the need to add a gastro-enterostomy or a pyloroplasty to the procedure to allow adequate drainage of the denervated, lazy stomach (28).

The technique of pyloroplasty had been known since 1888, when von Mickulicz-Radecki (1850-1905), one of Billroth's assistants, proposed this technique to treat patients with stenosing pyloric ulcer. In looking for more selective ways of denervating only the acid producing portion of the stomach, Johnston & Wilkinson in Leeds (29), and Amdrup and Jensen in Copenhagen (30) came up in 1970 with a so called highly selective or proximal gastric vagotomy. Not only did this offer the advantage of avoiding the drainage procedure, thus keeping the gastrointestinal tract closed, but moreover, in 1976 our team was able to prove that by inhibiting cholinergic neurotransmission in canine gastric arteries, this technique of highly selective vagotomy was also very effective in diminishing blood loss from bleeding gastro-duodenal ulcer (31).

An Italian surgeon, Grassi, stressed the importance of cutting an oesophagus-overrunning branch of the vagal nerve, innervating the fundal part of the stomach. Leaving this branch in place could compromise postoperative results, and the branch was known thereafter as the ramus criminalis or criminal branch. Highly selective vagotomy thus became in the 1980's the treatment of choice for drug resistant chronic peptic ulcer.

A clinical grading system introduced in 1940 by Visick to look at the outcome of gastric resections, also allowed highly selective vagotomy operations to be evaluated. As in other international series, our results reached approximately 90% 5 year cure rate for duodenal ulcers after such highly selective vagotomy (32). This changed the pattern of surgical treatment of peptic ulcer completely and by the mid 1980's, virtually all surgeons had exchanged their gastric resective procedures for a non resective, vagotomy type of operation.

New drugs however obviated the need for this new type of surgery. In 1967 Emas and Grossmann found histamine to be a pathway for hydrochloric acid secretion. Following the production of histamine receptor antagonists, well known as Tagamet and Zantac, inhibitors of the already mentioned proton pump were produced and proved to be extremely active in healing ulcers and eliminating their symptoms. The results were so impressive that peptic ulcer surgeons soon became unemployed. This was still more the case after the detection of the role of an infectious agent, Helicobacter pylori, in the pathogenesis of ulcer disease. This causative micro-organism, which is well demonstrated by the Whartin-Starry silver stain, can be totally eradicated by adequate antibiotic treatment, combined with proton pump inhibitors.

However some new difficulties arose with these miraculous drugs. In the first place, not all patients responded to the treatment, especially those who continued to smoke. Secondly, antibiotic treatment could induce resistance of the micro-organisms, which might break out again and so induce ulcer recurrence. Still more worrying was the fact that statistics worldwide indicated no decline at all in the frequency of perforated or bleeding ulcer. The reasons for this steady remaining incidence of ulcer complications is not known, but may be related to dietary habits, smoking or drug abuse.

Perhaps the most important drawback of these "miraculous" drugs is the need for life-long treatment in patients with chronic peptic ulcer disease. Some important side effects such as gastritis and metaplasia have already been described but the long-term morbidity associated with these drugs is not known. Surgeons therefore may continue to be confronted by a smaller number of patients needing definitive ulcer cure or emergency treatment of a peptic ulcer complication.

Happily, a real revolution in surgery has brought a new solution for these patients. Indeed, when L.T. Woodward pointed out that "the many achievements of 20th-century surgery are in reality only technical advancements, improvements in the state of the art rather than giant new leaps forward" (12), he did not take account of the introduction of minimal invasive surgery, used clinically in 1985 by the German Muhe of Boblingen for gall bladder excision (33) Laparoscopic surgery of the

stomach offered patients the same advantages of mini-invasive techniques, namely smaller wounds, less pain, easier postoperative recovery, and earlier return to work or family duties. The technique of highly selective vagotomy, was soon carried out laparoscopically, and proved, also in our hands, to be successful in a great majority of cases (34).

However, laparoscopic highly selective vagotomy proved to be a tedious and time-consuming operation. It made surgeons take up a variant of the vagotomy technique, introduced by Hill and Baker in 1978, a combination of truncal trans-section of the posterior vagal nerve, together with a highly selective vagotomy on the more accessible anterior side of the stomach (35).Laparoscopically this technique was easier to perform, as demonstrated by Kum and Goh from Singapore in 1992 (36).

Another technique, namely seromyotomy, also became feasible by the laparoscopic approach. This technique, described in open surgery by Vincent Taylor in 1979 for duodenal ulcer patients, consisted of an incision in the sero-muscular layer of the stomach, alongside the lesser curve (37). The operation aims at cutting intra-murally the various small vagal twigs, branching off the main Latarjet trunk to the fundus and body of the stomach. Such intervention on the anterior, combined with a posterior truncal vagotomy, was perfectly feasible with laparoscopic instruments.

My Belgian colleague, Azagra from Charleroi, performed 34 such operations and proved that this technique was very efficient in decreasing gastric acid secretion. However, it did result in a higher morbidity, consisting of iatrogenic perforation of the stomach in 5.8% of the patients (38). It made us use a safer technique of intramural trans-section of the gastric wall, namely gastric stapling.

Stapled anastomotic closure was specifically devised for the stomach by the Hungarian surgeon Humer Hulti in 1908. At that time, it was a rather heavy apparatus, designed to close the stomach wall during a partial gastrectomy. Refined in 1921 by Aladar von Petz, a lighter stapling apparatus was used for almost half a century, before it became replaced first by Russian, then American instruments. It was this type of longitudinal stapling machine that we used for the anterior intramural vagotomy we wanted to carry out .

After an extensive series of experiments in dogs, we were able to prove, in 1984, that anterior gastric stapling, combined with posterior truncal vagotomy, was perfectly capable of reducing gastric secretion adequately (39).

This type of operation not only diminished acid levels for longer periods (40), but also had no effect on gastric emptying of liquids or solids. (41,42). Subsequently, our group confirmed good long term clinical results in a large series of patients (43).

In 1992, the Spaniard, Fernando Gomez-Ferrer showed that our stapling method was perfectly feasible laparoscopically and was considerably easier and faster than laparoscopic highly selective vagotomy or seromyotomy(44). In 1993 the Americans Bailey and Zucker advocated our gastric stapling technique as one of the methods of choice of laparoscopic peptic ulcer management (45).

From what I have described, it can be seen that virtually no gastrointestinal disorder has been so dependent on changing physiological concepts and therapeutic regimens as peptic ulcer disease. Directly related to one of the most worrying concerns of our time, namely stress, gastro-duodenal ulcers have been treated surgically by at least ten different forms of operation.

From sodium bicarbonate to proton pump inhibitors, no drug has yet been able to eliminate completely the need for surgical correction of the disease or its complications. The recent introduction of a mini-invasive surgical method to treat this disease may obviate life-long treatment with drugs and may eventually offer a definitive chance of cure in patients not able to take long lasting medical treatment. It thus may offer a more economical solution for this disease in both western and third world countries.

Ideally of course, we should aim to prevent the disease, but this still seems unrealistic, and I don't have sufficient evidence to hope that peptic ulcer disease will vanish like diseases such as sweating sickness. Meanwhile we will probably see still more active and safer drugs developed, vaccination against Helicobacter pylori become a reality, or genetic manipulation change the predisposition of raised acid output or diminished mucous protection.

Will these treatments remain affordable in the future? I don't know. I certainly am more optimistic than many medical historians of the present time, who foresee great difficulties in the western world's ability to cover the huge expenses related to these life long treatments. As I have explained,

surgeons will always be ready to find new solutions and cope with problems that present when drugs or other treatments fail. Particularly in the field of gastric patho-physiology and surgery, we may conclude that there is a perfect link between medical history and actual medical concepts.

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THE ONE HUNDRED AND FIFTY FOURTH ORDINARY MEETING

The One Hundred and Fifty Fourth Ordinary Meeting was held at the Sunnyside Royal Hospital, Montrose on Saturday 19th June 1999. Three papers were read. Dr Helen Dingwall gave a paper on the history of the Royal College of Surgeons of Edinburgh and two students from St Andrews University gave papers based on medical history projects that they had undertaken during the year. Rebecca Corrigan talked on the history of General Anaesthesia and Hans Sharma gave a talk on the history of Tuberculosis.

THE HISTORY OF GENERAL ANAESTHESIA

Anaesthesia, "loss of sensation", was a goal towards which physicians the world over strove for centuries. Before the discovery of anaesthetics the development of surgery had been limited by the inexorable barrier of pain. Not only were patients loathe to come under the knife, students were often unwilling to pursue a career because of their experiences. Even J Y Simpson considered a change in this direction,

"After seeing the terrible agony of a poor Highland woman under amputation of the breast, he left the classroom and went to the Parliament House to seek work as a writer's clerk."

Despite advances in anatomical knowledge and understanding of disease, operations had to be quick and were usually confined to the surface of the body, minimising the time that a patient was bound to the operating table. Control of pain was vital, as was prevention of haemorrhage and infection, before surgery could develop to the complex and intricate operations possible today.

Early attempts at alleviation of pain for operations were made by the ancient Chinese, Egyptians, Assyrians and Scythians. Mandragora, cannabis, alcohol, soporific sponges and opium were all used to dull surgical pain. Mechanical techniques included compression of blood vessels to the affected area and producing unconsciousness by carotid artery pressure or excessive bleeding.

The major breakthrough came with the discovery of gases and increased understanding of the physiology of respiration and circulation. Between 1766 and 1772 hydrogen, nitrogen, oxygen and nitrous oxide were discovered. With these discoveries came the idea of treating illness with vapours and gases and this principle led Dr Thomas Beddoes, in 1798, to establish the Medical Pneumatic Institute in Bristol.

Humphry Davy, a young man from Penzance, who had been apprenticed to a surgeon apothecary, carried out detailed work at the Institute on the properties of nitrous oxide. Often putting his own life at risk Davy discovered the analgesic properties of nitrous oxide, having numbed the pain of an erupting wisdom tooth by inhaling the gas. In 1800 he published his findings in a paper "Researches, Chemical and Philosophical, chiefly concerning Nitrous Oxide" commenting,

"As nitrous oxide in its extensive operation appears capable of destroying physical pain, it may be used with advantage during surgical operations in which no great effusion of blood takes place."

As nitrous oxide became better known, not for its anaesthetic potential, but for its pleasant intoxicating effects, comparisons began to be drawn between it and ether. Ether was one of the earliest known synthetic organic compounds, having been described in 1540 by Valerius Cordus. In 1818 Michael Faraday, a student of Davy's, was studying gases and noticed the anaesthetic property of ether and its common properties with nitrous oxide. Writing in the English Journal of Science and Arts he stated,

"When the vapour of ether mixed with common air is inhaled, it produces effects very similar to those occasioned by nitrous oxide... It is necessary to use caution in making experiments of this kind. By the imprudent inspiration of ether a gentleman was thrown into a very lethargic state, which continued with occasional periods of intermission for more than thirty hours... "

Despite the writings of Davy and Faraday, the medical profession was very slow in using nitrous oxide or ether as anaesthetic agents. It was not until 1824 that the concept of surgical anaesthesia was born, when Henry Hill Hickman carried out painless operations on animals using carbon dioxide, naming the effect "suspended animation". This was the right idea but the wrong gas and Hickman failed to arouse sustained interest. If the physicians of the day neglected the discoveries of some of history's greatest chemists, the public did not. Ether and nitrous oxide became fashionable recreational drugs, used especially by medical students.

In 1842 ether anaesthesia was finally put into practice when William E Clarke gave ether to a Miss Hobbie, for removal of a tooth by Dr Elijah Pope. Clarke's work was not published, apparently because of persuasion from the University of Georgia, where experiments on hypnosis were being carried out. In the same year Crawford W Long, unaware of Clarke's work, also used ether as a surgical anaesthetic. Long had reputedly dabbled in nitrous oxide and ether while a medical student in Pennsylvania. On 30th March, he used ether to excise a tumour from the neck of one of his patients, yet he too failed to publish his work. He had been taught by his tutors not to publish incompletely verified work and it was not until 1853, satisfied with his fully investigated results, that he made known his remarkable discovery.

By 1844, Davy's research remained unheeded, although the intoxicating effects of nitrous oxide were widely known and it was used at public demonstrations. At one such display, a member of the audience, Horace Wells, a dentist, noticed that a volunteer from the audience had sustained injuries of which he was unaware. Wells realised the significance of this and on December 11 1844, having inhaled nitrous oxide, he had a tooth painlessly removed by a dental colleague. Excited by his findings, Wells organised a public demonstration at Massachusetts General Hospital, in January 1845. This was however a failure as the patient cried out as if in pain, although later claimed to have felt nothing. Wells was booed out with the word "Humbug". Wells, disappointed, eventually committed suicide by cutting his wrists while inhaling ether, ironically the anaesthetic which was to supersede his own discovery.

Although Crawford Long and William Clarke had recognised the anaesthetic properties of ether, it was Williarn T G Morton, unaware of their work, who was to make public the discovery that ether was a general anaesthetic. Morton, a dentist, had studied under Wells and seen his fiasco. On Wells' recommendation, Morton consulted Dr Charles Jackson, who suggested ether .On September 30 1846 Morton painlessly extracted a tooth from Eben Frost having used ether and he reported this in the Boston Medical and Surgical Journal. On October 16 that year Morton gave ether at a public demonstration, again at the Massachusetts General Hospital, and John C Warren removed a tumour from the mandible of Gilbert Abbott. On completion of the operation, Jackson is alleged to have turned to the audience and said "Gentlemen, this is no humbug". The news of the discovery was sent to London by Dr Bigelow, a surgeon who had seen the demonstration and shortly the news spread throughout the civilised world.

Change is often received with caution and even mistrust, but in the case of ether the concept was rapidly accepted. By the end of 1846, ether had been used in Britain and France, Robert Liston, in London, for example, amputating a thigh. The discovery was embraced by a change in attitudes in doctors and patients alike, not only in surgery, but also in midwifery. Ether was feasible in childbirth and in January 1847, James Y Simpson, Professor of Midwifery at Edinburgh University, used ether in labour without affecting uterine contractions. Although Simpson was pleased with the results, ether was inflammable, irritant to breathe and caused nausea and vomiting and he searched for an alternative.

In 1847, Simpson consulted Mr David Waldie, a chemist from Liverpool, for an alternative to ether. Waldie suggested chloroform, a compound known since 1831 and Simpson obtained a sample. Simpson experimented with the drug at a dinner party with friends and relations and found that it was remarkably effective, producing unconsciousness around the table. He wasted little time implementing and making known his discovery. By 8 November 1847 he had used chloroforrn in obstetrics and on 10 November he published "Notice of a New Anaesthetic Agent as a Substitute for Ether in Surgery and Midwifery". He stated:

"*As an inhaled anaesthetic agent, it possesses, I believe, all the advantages of sulphuric ether, without the principal disadvantages*"

Simpson faced religious objections, with some feeling that women should not be unconscious during labour, but Simpson, relishing an argument, was able to overcome this opposition and chloroform began to replace ether in surgery, midwifery and dentistry. However by January 1848, it had claimed its first victim, Hannah Greener, a young woman having an operation on her toe, and as time went on, more unexplained chloroform deaths occurred.

Many theories were put forward, but few were based on science. John Snow was the first physician to examine the anaesthetic effects of chloroform and ether on a sound scientifilc basis. In 1848 his book "On Chloroform and other Anaesthetics" was published and in this he described chloroform exerting its lethal effects by causing primary cardiac paralysis. Snow became the first true anaesthetist and, by giving chloroform to Queen Victoria on two occasions, helped to allay public concern about anaesthesia.

After Snow's death in 1858, his work on chloroform was continued by Joseph Clover. Clover improved equipment for both chloroform and ether administration, enabling the percentage of the vapour given to be regulated. The ongoing argument about the relative value of ether or chloroform continued for a number of years but eventually the issue became less prominent and the emphasis turned from the anaesthetic to the technique of application.

With the invention of the hypodermic syringe in 1853, drugs could be injected directly into the body. The development of drugs producing local anaesthesia, such as cocaine, allowed spinal anaesthesia to be introduced. The properties of curare, a poison used to tip the arrows of Indians from the Amazon led to the idea of muscle relaxation. This could be used to advantage when performing oro-tracheal intubation, a technique introduced by William Macewen in 1880, to overcome airway obstruction. With this new knowledge came the need for regulation and registration. The London Society of Anaesthetics was founded in 1893 and in 1894 anaesthetic record keeping was introduced by Harvey Cushing and Ernest Codman.

The fates of the three major anaesthetics of the nineteenth century were varied. Chloroform fell into disuse. Despite its many assets, it was discovered to sensitise the heart to adrenaline leading to ventricular fibrillation, particularly during the lighter phases of anaesthesia and on surgical stimulation. It was this and the resulting circulatory arrest that caused so many of the deaths related to chloroform anaesthesia. Ether still has a use in the third world where its ease of application and cheapness overcomes its disadvantages. Nitrous Oxide is still used extensively today, in conjunction with other anaesthetics and mixed with oxygen it is still used to dull labour pains.

Organic volatile agents have prevailed this century. Halothane was a popular anaesthetic used in the 1960s and 1970s. However it was discovered to be associated with liver problems and was superseded by desflurane and sevoflurane. These quick acting agents are favoured today because of the advantages of a rapid recovery when day case surgery is becoming increasingly common. Anaesthesia is often produced by a combination of volatile agent, nitrous oxide and oxygen and relaxants, which have developed from curare.

In contrast with the early part of the twentieth century, mortality as a direct result of anaesthesia is falling, with the anaesthetic very rarely being the primary cause of death. The quest for the perfect anaesthetic continues and it is difficult to predict what the future holds for the ever-developing domain of anaesthesia. Both hypnotism and acupuncture have been cited as possible aids to anaesthesia and both are areas for further study.

It is evident that since the ill-fated public demonstration of nitrous oxide by Wells in 1844, anaesthesia has become an integral and fundamental part of medicine. We have moved a long way in scientific and technological terms, yet the medical profession still strives to maintain Simpson's legacy,

"*And I most conscientiously believe, that the proud mission of the physician is distinctly twofold - namely to alleviate human suffering, as well as preserve human life*"

THE HISTORY OF TUBERCULOSIS

Tuberculosis is an ancient infectious disease, which has plagued humans throughout recorded and archaeological history. It is thought to be the oldest of human diseases and it has taken more lives than any other over the millennia. I decided to study tuberculosis not only because of its impact on the health of mankind, but because even people of my age who have not known someone with tuberculosis, can still appreciate the loss to society caused by the premature deaths of those such as the entire Bronte family, John Keats, Frederic Chopin and Edvard Grieg.

Tuberculosis was once considered a romantic disease associated with creative genius. Alexander Dumas wrote (sarcastically):

"It was the fashion to suffer from the lungs; everybody was consumptive, poets especially; it was good form to spit blood after each emotion that was at all sensational, and to die before reaching the age of thirty."

In the 18th century, John Bunyan referred to tuberculosis in Europe as "Captain of all these Men of Death" and a century later, Oliver Wendell Holmes described tuberculosis as the "white plague". Tuberculosis, also known as phthisis and consumption (both meaning wasting), is still one of the most prevalent infections of human beings. It is responsible for 25% of adult deaths in the developing world – more than diarrhoea, malaria and AIDS combined. About one third of the world's population is infected with Mycobacterium tuberculosis and the World Health Organisation has estimated that in the 1990s there will be approximately 90 million new cases, with approximately 30 million deaths.

To understand the history of man's struggle with tuberculosis it is helpful to know something about the disease. It is caused by Mycobacteria, organisms found in soil, animal dung and water

and potentially pathogenic to many animals including fish, reptiles and mammals. Mycobacteria are aerobic, (growing most successfully in tissues with a high partial pressure of oxygen, such as the apex of lung). They are slow growing intracellular pathogens, (with a generation time of 12 to 24 hours). They are extremely resistant to physical stress and, retaining the carbol-fuchsin stain after washing with acid and alcohol, they are classified as acid-fast bacilli.

Man is affected by both M tuberculosis and M bovis, which is usually pathogenic for animals. It has been suggested that cattle were the source of human tubercular infection, and that M tuberculosis was a mutant of M bovis, which has a wide host range including man. Skeletons, from the 6-7th century BC, when cattle were first domesticated, show evidence of Pott's Disease, a skeletal manifestation of tuberculosis.

Tuberculosis is a necrotising bacterial infection most often affecting the lungs. Lesions may also occur in the kidneys, bones, lymph nodes or meninges, or be disseminated throughout the body. Tuberculosis was named because of the firm nodules or tubercles found in the disease; these are granulomas. There are 2 stages in which the infection may cause clinical disease.

1. Primary tuberculosis, in which the bacilli invade a host having no specific immunity; at this stage the disease most commonly heals spontaneously, but may progress to clinical disease if immune mechanisms fail; this occurs mostly in childhood.
2. Post primary tuberculosis, which occurs mainly in young adults; this is as a result of progression of infection years later in spite of specific immunity.

Transmission occurs either by the airborne or gastro-intestinal route. When someone with active pulmonary tuberculosis coughs or sneezes, droplets containing 1-3 organisms are propelled into the air where they remain suspended. They may be inhaled and then taken up by alveolar macrophages. One organism may be enough to establish an infection, although individual susceptibility varies. If infection occurs, the bacilli multiply in cells and may disseminate through lymph and blood. Usually, however, the initial infection is clinically mild and there are no symptoms. In a minority of infected people this initial infection may progress to active clinical disease within two years, "progressive primary tuberculosis". Risk factors include a large inoculation, extremes of age and immuno-suppression, especially HIV infection. For the majority however, tuberculosis remains latent for years and only 10% develop clinical disease. Progressive disease may develop after 60 years of clinical dormancy. When nodular lesions of tuberculosis become active and coalesce, liquefaction and necrosis (caseation) develops from which liquid escapes, leaving a cavity within the lung. The sputum is then often highly infectious. As areas of destruction expand, blood vessels are often eroded. Organisms may reach other sites in the body via lymphatics or the blood stream. Reactivation is more likely in patients with a weakened immune status, for example the extremes of age, HIV infection, diabetes mellitus, malignancies, steroid dependent disease such as asthma and poor nutritional status particularly related to alcohol and drug abuse.

Bovine tuberculosis infects cattle and, in contrast to humans, nearly all cattle develop active disease and can transmit the organism to other animals including humans. Bovine infection is acquired by drinking milk or rarely eating beef and is related to non-pulmonary forms such as cervical lymphadenopathy, intestinal and abdominal tuberculosis, bone and joint disease, skin infections and tuberculous meningitis, especially seen in children.

Symptoms of tuberculosis include physical wasting, fevers, particularly at night, and fatigue. Pulmonary tuberculosis is associated with coughing, production of sputum, which may be bloody, shortness of breath, wheezing and chest pains.

Our knowledge of the disease has been built up over centuries and its history is linked to the history of civilisation. Information from the past has come from studies of skeletons by palaeo-pathologists, from ancient art depicting characteristic deformities, and from early medical texts.

Tuberculosis of the spine, Pott's disease, usually affects the thoracic vertebra, with collapse and anterior fusion of the mid-thoracic vertebrae, giving a knuckle-like projection of the spine and deformity of the chest. The oldest skeletal remains showing evidence of tuberculosis were found in Heidelberg and are thought to date from 8000 BC. Egyptian mummies, from around 3700 BC, have been found with tuberculosis and a concentration of skeletons found near Thebes has given rise to speculation that there may have been a hospital there.

That tuberculosis was widespread is demonstrated by writings from China and Babylon, India and Greece. There is dispute as to whether tuberculosis was present in the New World before European colonisation, but proof of infection has been found in Southern Peru in 700 BC.

At first, despite sometimes detailed descriptions of disease, there was little attempt to identify the cause of this or any other illness. The supernatural was usually felt to be responsible, perhaps as punishment from the gods for sin. All disease was likely to be treated similarly using rituals of religion, exorcisms, invocations, charms and amulets.

From the time of Hippocrates (460-377 BC), medicine began to be separated from religion. The Hippocratic writings contain an excellent description of the disease, describing phymata (tubercles) in the tissues of animals and using the term phthisis to refer to consumption with pulmonary symptoms. The early stages are documented as dry cough, yellow sputum, chest pains, malaise and wasting. Other non-pulmonary manifestations of the disease were, however, diagnosed as separate diseases and the contagious nature of tuberculosis was not recognised. Treatment was supportive rather than curative, although drainage of lung abscess was recognised " so as to give vent to a small portion of the fluid".

However treatment was generally ineffective and Plato (430-347 BC) recommended no treatment, because caring for chronic patients was no advantage to the patient or the state.

The next advance in the study of the disease came from Aretaeus of Cappadocia, in the second century AD, who noted additional details.

"of the bones alone, the figure remains, for the fleshy parts are wasted... Nose sharp, cheeks prominent and red, eyes hollow, brilliant red and glittering, swollen, pale, or lined is the countenance; the slender parts of the jaw rest on the teeth, as if smiling."

He also noted evening fever sweats. His contemporary, Galen made the important suggestion that the disease was contagious and that it was dangerous to live with a sufferer.

For hundreds of years there was no real progress in understanding the cause or in treating the disease. Scrofula, a tubercular infection of the lymph glands in the neck was apparently common in the Middle Ages and a popular ritual remedy in England and France was the "King's touch". Records show that Edward I touched 533 victims in one month and that Charles II bestowed the royal touch on 92,102 scrofulous patients. This secular approach to healing was similar to the religious rituals of the ancient world and was as ineffective. Arnold of Villanova (1235-1312 AD) noted that scrofulous patients always had a deeper source of infection and that there was no purpose in operating externally.

During the 16th century scientific study gathered pace. Jean Fernel performed post-mortem studies, identifying consumptive lesions in the chest and suggesting that the disease was common. Paracelsus (1490-1541), published a study of miners' susceptibility to tuberculosis and Girolamo Fracastoro (1483-1553), developed a germ theory of epidemic disease when he postulated that disease was caused by "imperceptible particles", some 300 years before their microbiological discovery.

Franciscus Sylvius, from autopsies, deduced that the disease was characterised by nodules, which he named "tubercles". Desault (1675-1737) and Stark (1741-1770), both believed that the disease was spread by infected sputum.

Pulmonary tuberculosis became endemic in Europe in the 17th century, with the first peak occurring between 1650 and 1675. In 1699, the Italian republic of Lucca introduced regulations for prophylaxis of consumption. An edict stated that

"Henceforth, human health should no longer be endangered by objects remaining after the death of a consumptive. The names of the deceased should be reported to the authorities, and measures undertaken for disinfection"

During the 18th Century, such regulations became common in Southern Europe; for example in 1783, Naples made it mandatory to report consumptive patients and failure to do so incurred a fine of 300 ducats, with 10 years imprisonment for subsequent offenders. Those personal belongings which could not be cleaned were destroyed. A consumptive's house had to be re-plastered from "cellar to garret" at the public expense; wooden door and window frames were removed and burned and new ones installed. New houses were to stand empty for a year or for six months after re-plastering. Any interference with the administration of these rules or non-compliance was punished by heavy fines. Unfortunately these measures were not sustained.

In 1720, Benjamin Marten, an English physician, published "A New Theory of Consumption", in which he suggested that the disease could be caused by "wonderfully minute living creatures" which, once they had entered the body, could generate the lesions and symptoms of the disease.

"It may be therefore very likely that habitual lying in the same bed with a consumptive patient, constantly eating and drinking with him, or very frequently conversing so nearly as to draw in part of the breath he emits, a consumption may be caught by a sound person. I imagine that slightly conversing with consumptive patients is seldom or never sufficient to catch the disease"

Unfortunately public health measures based on his theories were not instituted in Britain.

During the early 19th Century, two French physicians contributed greatly to knowledge of the diagnosis and nature of the disease.

Bayle (1774-1816), did research on the tubercles found in miliary disease. He realized that extra-pulmonary tubercles were identical with pulmonary tubercles and that tubercles were a specific entity of the disease.

Laennec (1781-1826), in 1816 introduced the stethoscope, first as a roll of paper and then a wooden cylinder. Using this he documented the abnormal sounds that he heard using terms such as rale, rhonchus and pectoriloquy. Importantly, he performed autopsies if patients died and thus was able to relate clinical signs to pathology. He described the development of tubercles to caseation and realized that healing by fibrosis could occur after caseous material had been removed. Before his book "Traite de L'Auscultation Mediate" was published in 1819 he wrote

"I risked my life, but the book I am going to publish will be, I hope, useful enough sooner or later to be of more value than the life of a man."

Ironically. Laennec, like many other doctors involved with patients with tuberculosis, died from the disease in 1826.

Despite the possibility of earlier diagnosis, improvements in medical treatment were slow in coming. Bleeding, emetics and purges were used and drugs included laudanum digitalis and antimony. Laennec even surrounded his patients with seaweed from Brittany. James Carson (1772-1843) an Edinburgh graduate, after experimenting on rabbits, suggested that producing an artificial pneumothorax might allow rest and healing of the lung.

"In these cases in which the disease is placed in one of the lungs only, the remedy would appear to be simple safe and complete"

He attempted to induce an artificial pneumothorax in 1822, but pleural adhesions and loculated empyema prevented collapse. Without antisepsis or anaesthesia the idea was not pursued.

Despite the lack of definitive treatment, mortality began to decline, because of economic reforms and improved living conditions. The dangers of certain occupations such as stonecutters or miners was recognised, and variations in racial susceptibility were noted. Attempts to treat the whole patient led to the development of sanatoria. In 1840, George Bodington of Sutton Coldfield, recommended that

"The tuberculous patient should be in an airy house in the country, which if on an eminence, so much the better. The neighbourhood should be dry and high, the soil a light loam, the atmosphere free of damp and fogs, and the cold never too severe to breathe in the open air."

He also encouraged exercise, a good diet and "generous wine". These principles were not applied initially in Britain, however but were first used on the continent.

Herman Brehmer (1826-1889) developed tuberculosis and was advised by his doctor to seek a healthier climate. He studied botany while he lived in the mountains. He was cured and began to study medicine. His doctoral dissertation was "Tuberculosis is a curable disease". In 1859 he opened a small sanatorium in Gobersdorf and patients were given not only a good diet and regular exercise, but were exposed on balconies to continuous fresh air. A patient of Brehmer's, Peter Dettweiler, opened a sanatorium in the Taunus mountains in 1876. In the USA another doctor, Edward Trudeau, developed tuberculosis and was expected to die within a year. Spending that year in the Adirondack Mountains, he survived and established a famous sanatorium at Lake Saranac. Another internationally known sanatorium was the Nordrach Kolonie in the Black Forest established by Dr Walther. Many British physicians visited this and set up similar sanatoria in England Wales and Scotland including Nordrach on Dee at Banchory.

The establishment of sanatoria was a very important development in that it not only gave hope of a prolonged life and possible cure, but it also isolated patients, reducing the risk of transmission to others. Sanatoria were used well into the 20th Century.

A breakthrough in showing the cause of tuberculosis came with the demonstration by Jean-Antoine Villemin (1827-1892) in 1865 that the disease could be passed from man to rabbit and cow to rabbit by inoculation of tuberculous material. The significance of this work was still not appreciated, however and it was the work of Robert Koch that cast the greatest light on the mystery. Koch, working on anthrax, was able to grow the organism responsible in pure culture and show that it could still cause the disease. He also showed that dormant spores could be formed by the organism. He confirmed the definite causal relation of a particular organism to a particular disease.

Koch's postulates concerning infectious disease were that a specific organism should be found in all cases of the disease in question, that it should not be present in the tissues of healthy individuals, that the organism could be grown outside the body of the host for several generations and could then reproduce the disease in another susceptible animal.

After anthrax, Koch turned his attention to tuberculosis. Using aniline dies and an oil immersion microscope, he identified an organism which he called the tubercle bacillus. This could be cultured outside the body and then cause tuberculous lesions when a healthy animal was inoculated. These findings were presented on March 24th 1882 and revolutionised the understanding of tuberculosis. Koch believed at first that the human and bovine tubercle bacilli were identical, but in 1898, Theobald Smith in Harvard, showed there were a number of differences between them. It is now thought that the human bacillus, spread by coughing and sneezing via droplets, accounts for 98% of pulmonary tuberculosis and 70% of non-pulmonary disease, whereas bovine tuberculosis is acquired by drinking infected milk or rarely eating infected meat and is related to non-pulmonary forms. In 1885 Ehrlich (1854-1915) developed an acid-fast stain which enabled early diagnosis of bacilli in sputum.

Koch's work inspired a young Scottish physician, Robert Philip, who graduated in Edinburgh in 1882 and decided to study tuberculosis. By studying the location of each case of tuberculosis in various parts of Edinburgh, he was able to show the presence of small epidemics. He was able to look at family members and close contacts of the original patient. This was the start of epidemiology, how disease spreads in a family or community. In addition to the dispensary that he had started, he opened a sanatorium outside the city to isolate patients and with the help of public subscription, he opened the Royal Victoria Hospital, which became a model world wide for coordination between community dispensaries, hospitals, sanatoria and medical officers of health, allowing contact tracing and subsequent home visiting.

In 1896 Conrad von Roentgen (1845-1823) discovered X Rays, and by the 1920s these were being used in the diagnosis of lung disease. Increasingly this was to play a useful role in diagnosis of tuberculosis and assessment of the progress of treatment.

One approach to treatment, taken by Koch himself, was to try to prevent the disease rather than cure it. This led to the production of a vaccine, tuberculin. This found its most useful role in diagnosis, as it causes an allergic reaction in patients who have already had tuberculous infection, even if all other signs of the disease have gone or if the original disease was so mild as to cause no symptoms at all. In 1908, Mantoux pioneered an intra-dermal method of giving the tuberculin, which became the standard technique although the Heaf method is now most commonly used.

Towards the end of the 19th century, a number of National Associations were inaugurated, starting in 1889 in the USA. In 1898 in Britain the NAPT was formed (National Association for the Prevention of consumption and other forms of Tuberculosis) and concentrated on three areas, education of the public, provision of institutional treatment and the elimination of tuberculosis from cattle.

In France Albert Calmette (1863-1933) and Camille Guerin, working over many years gradually developed a strain of the tuberculosis bacillus that could produce a non-fatal type of disease in guinea pigs, which caused no disturbance but still provoked an immune response. This became known as the bacillus-Calmette-Guerin (BCG). After initial concerns about its safety it was eventually widely adopted and from 1954, most Health Authorities in the United Kingdom began voluntary vaccination of 13 year olds.

The great breakthrough in treatment came in 1944 with the discovery of streptomycin through the work of Selman Abraham Waksman. Initial use of this showed its ability to kill the tubercle bacillus but problems with toxicity and the development of resistance. Lahman in Sweden discovered para-amino salicylic acid (PAS), which had bacterio-static activity. An MRC trial showed that a

combination of streptomycin and PAS allowed treatment for prolonged periods without the development of resistance. Prolonged treatment over two years was developed in Edinburgh by Crofton. In 1952, a third drug, isoniazid, was discovered by Robitzek and Selikoff in New York.

This proved very effective in combination with streptomycin. In comparing these drugs alone or in different combinations, the principles of the modern clinical trial were laid down. Further work led to the development of pyrazinamide (1954), ethambutol (1962) and rifampicin (1969) and to the introduction of different regimes including intermittent and short course therapy.

In industrialised countries, the incidence of tuberculosis had declined significantly before 1940 because of improvements in nutrition, housing, ventilation of buildings, pasteurisation of milk and isolation of infected patients in sanatoria. Mortality and morbidity were further reduced by the addition of chemotherapy to these measures, but unfortunately there has been a resurgence of the disease since 1985. In underdeveloped countries, increasing populations with overcrowding, poverty and malnutrition has led to rapid transmission. Having started treatment, patients often stop because they feel better, but before the full course has been completed. This has led to the rapid emergence of drug resistant bacteria. The spread of HIV infection in sub-Saharan Africa and some parts of Asia has allowed tuberculosis to spread in these immuno-compromised patients, to the extent that it is the leading cause of death in such patients.

In developed countries too, cases are increasing. Those at particular risk include the immuno-compromised, the poor with poor nourishment, poor living conditions, without homes, alcoholics, immigrants and prisoners. A serious problem is that of multi drug resistant tuberculosis, (MDR TB). In the 1960s 1-2% of isolates were resistant to 2 or more drugs, while by 1991 33% of isolates were resistant to 1 or more drugs and 13% were resistant to the front line 4 drugs.

The WHO is sponsoring the DOT system (Directly Observed Treatment). This ensures that health workers watch patients swallow their medicines throughout their 6 months of treatment. This results in a decrease in the number of patients with MDR TB. Nevertheless in 1993 the WHO declared tuberculosis a "Global Emergency", the first such declaration in WHO history.

Present research involves several disciplines. The development of new drugs includes rifampetene a drug taken weekly rather than daily. The genome of M. tuberculosis is being studied and a host gene (NRAMP) that determines susceptibility and resistance has been found in humans. Research continues into vaccines.

The history of tuberculosis covers several thousand years. Over the centuries man has discovered the cause of the disease, the methods of transmission and ways of reducing mortality and morbidity. Unfortunately, unlike the scourge of smallpox, the history is not yet complete.

As Dubos and Dubos wrote in 1987 in the White Plague; Tuberculosis, Man and Society:

"Elucidation of the mechanisms of tuberculosis disease will long continue to require analysis by the methods of medical sciences. And the case of the stricken tuberculosis patient calls upon all the resources of medical practice. But the complete control of tuberculosis in society goes beyond medicine in its limited sense. It is a problem in social technology."

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The Scottish Society of the History of Medicine

REPORT OF PROCEEDINGS
SESSION 1999-2000

THE FIFTY FIRST ANNUAL GENERAL MEETING

The Fifty First Annual General Meeting of the Society was held at the Scottish Health Service Centre at the Western General Hospital in Edinburgh on the 31 st October 1998.The President, Dr John Forrester was in the chair. The minutes of the Fiftieth Meeting were approved and the Secretary's and Treasurer's reports were accepted. Dr James Gray was elected as a new Council member in place of Professor Ian McCallum who was thanked for his contributions. A new Constitution, which owed much to the work of the President and had been worked on at several council meetings, was approved.

THE ONE HUNDRED AND FIFTY FIFTH ORDINARY MEETING

This meeting directly followed the Fifty First Annual General Meeting, at the Scottish Health Service Centre in Edinburgh. The President, Dr Forrester introduced two speakers. The first, Dr James Gray talked on the History of the City Hospital, Edinburgh. The second paper was by Dr Derek Dow, one time Secretary of the Society and Archivist to the Greater Glasgow Health Board and now living in New Zealand, on Glasgow's Contribution to New Zealand Medicine.

A HISTORY OF THE EDINBURGH CITY HOSPITAL

Until the mid-1880s, hospital accommodation for Edinburgh's fever patients was haphazardly provided. In medieval times when smallpox, syphilis, leprosy and plague were prevalent and feared by laymen and doctors alike, leper houses were established and fever patients were cared for in the religious houses of charity. Thus the Chapels of St Roque and of St Katherine of Sienna at either end of the Burgh Muir took in plague victims and their close contacts who had been ousted from the city under a series of draconian laws designed to limit the spread of contagion. The fear of infection was such that ships arriving from foreign ports known to be plague infected were ordered to be disinfested and quarantined at Inchcolm Island in the Forth before being allowed to unload their cargoes at Leith or Newhaven.

Much of the morbidity and mortality from infectious diseases in bygone Edinburgh can be attributed to atrocious living conditions. The inhabitants of the Scottish capital were reluctant to live outside the city walls which they hoped would protect them from English invaders. Consequently, as the population grew, overcrowding became a health hazard. Tenements or 'lands' soared upwards cutting out light and fresh air from the wynds and closes below. Drainage was primitive and the disgusting practice of tipping refuse and human excrement from tenement windows with the warning cry of 'gardy-loo' or 'gardez-l'eau' earned Edinburgh the unenviable title of the filthiest city in Europe. Pig-sties and byres occupied many of the basements. The unofficial slaughtering of animals in unpaved backcourts produced piles of decomposing offal, which encouraged vermin and added to the stench from other refuse.

'Epidemic fever' of the 18th and 19th centuries was probably louse-borne typhus which flourished among the overcrowded tenants whose personal hygiene remained negligible even after the city's water supplies were improved and some citizens were slowly and reluctantly moving to James Craig's spacious New Town. In 1818 and 1832, coinciding with an influx of Irish and Highlanders into the old town flats as they sought employment in the capital, Edinburgh suffered the first of several disastrous epidemics of asiatic cholera.

Queensberry House, in the Canongate, became a hospital for typhus and cholera patients from 1818 to 1835. The city's charity workhouses were commandeered during epidemics to house fever victims and the 'Little House', forerunner of the Infirmary, and, from the 1740s onwards, the new 200 bed Royal Infirmary itself set aside some beds for fever patients.

In 1862 Edinburgh wisely chose as its first Medical Officer of Health the illustrious Sir Henry Littlejohn. Among his many public health achievements were slum clearance, the introduction of compulsory smallpox vaccination, the notification of infectious diseases by medical practitioners and the rapid transfer of fever patients into hospital. All these measures helped to control infection in the still crowded, insanitary common stairs of the old town.

During the later 19th century controversy surrounded the question of who was to look after fever patients. Following the Public Health (Scotland) Act of 1867 the Town Council as the local authority was the body responsible. The Managers of the Royal Infirmary, however, wanted to keep a token number of infectious patients for clinical teaching but firmly declared the financial burden of epidemics must be borne by the Town Council. After years of bickering, the Managers agreed to sell off very cheaply the old Infirmary site to the local authority, providing the Council thereafter accepted total responsibility for managing infectious diseases either on that site or at the Canongate Charity Workhouse which the Town Council had bought in 1870.

From 1884, then, Edinburgh's fever hospital occupied the old Infirmary site (the original William Adam's building having been demolished). Included in the complex of buildings on the site were the old Royal High School, the New Surgical Hospital fronting Drummond Street and the original buildings of the Royal College of Surgeons of Edinburgh and the Royal Medical Society. Plans were well advanced for developing the whole site from Infirmary Street, High School Wynd and Drummond Street down to the Pleasance. A suggestion was even made to construct a bridge over the bottom of the Pleasance to connect the main hospital with new wards to be built on St John's Hill. Everyone, including the Medical Officer of Health himself, agreed that this was the ideal site on which to develop the modern fever hospital.

These plans, however, were overtaken by events. In 1894-5 Edinburgh was at the height of a severe smallpox outbreak, and it became obvious that the old Infirmary site was inadequate. The citizens complained to the Town Council about the wooden buildings hastily erected in the Queen's (Holyrood) Park to harbour smallpox victims. The Fever Hospital Sub-Committee asked advice from the Royal College of Physicians of Edinburgh as to whether they should continue to develop the old Infirmary site or move, as the Royal Infirmary itself had done in 1879, to a green field site. Only one Fellow voted against moving to a new venue. Then the Lord Provost's Committee, which had taken over the task of finding the new site, revisited the city's poor houses to see if one them could provide the nucleus of a new fever hospital. The most modern of these was Craiglockhart Poorhouse erected in 1870. What attracted the Committee members most on their visit was not the poorhouse, however, but the land of Colinton Mains Farm lying immediately to the south.

After taking advice from the city's Architect, Engineer and the Medical Officer of Health, the Town Council purchased 131.5 acres of the farm and a small adjacent feu belonging to the Water Board for £20,500. With its elevated position on the city's southern boundary, its excellent drainage and the gently south sloping ground, Colinton Mains Farm was an ideal site.

Robert Morham, City Architect and Superintendent of Works, designed the new hospital with meticulous care and incorporated features gleaned from a visit he had made previously with Bailie James Pollard to inspect continental fever hospitals. Patients with different diseases would be segregated in separate, mainly two-storeyed, pavilions oriented on a north-south axis so as to obtain maximum sunshine and placed sufficiently far apart that they never overshadowed each other. The wards were built with cavity walls, brick and plaster within tied to pink Dumfriesshire sandstone outside. Underground corridors connected the buildings and acted as conduits for gas and water pipes, electric cables and for the coal carts to supply the solid fuel ward fires.

At a grand ceremony in 1897 Lady McDonald, wife of Lord Provost Sir Andrew McDonald, cut the first sod. Shortly thereafter, however, began an acrimonious legal dispute between Robert Morham and the builder, John Lownie, over alleged construction delays, the quality of workmanship and alterations in the building specifications. After a series of court proceedings, involving no less than four arbiters, Lord Kincairney found for the builders against the Town Council in 1904.

Despite this set back and the costs, which had escalated from £240,000 to nearly £340,000, the Town Council seemed pleased with their new hospital. It was opened with regal splendour on 13th May 1903, by King Edward VII and Queen Alexandra, to the cheers of thousands of spectators who crowded into the hospital grounds. The decision to build a 600 bed hospital rather than a cheaper 400 or 500 bed hospital had not been easy for the Town Council and had been vigorously opposed by the city's Treasurer. All the eastern pavilions, accounting for 329 of the 600 beds, were dedicated for scarlet fever. To the west side there were separate pavilions for diphtheria, typhoid, erysipelas, whooping cough, measles and chickenpox. The most isolated ward near the hospital's west gate was for typhus patients.

Smallpox patients were accommodated in the wooden hospital to the north-west of the main hospital on ground which later became Wester Hill estate . Some of the wooden buildings from the Queen's Park that had not been used in the 1894-5 smallpox outbreak had been transported to form the nucleus of this 'temporary' hospital at Colinton Mains. Smallpox occurred intermittently in Edinburgh from 1903 to 1942 and was always a cause of anxiety for the medical and nursing staff. Specially chosen revaccinated nurses were often incarcerated in the smallpox hospital for six or more weeks until the outbreaks subsided; there was never a shortage of volunteers.

Dr. Claude B. Ker, the first Medical Superintendent at Colinton Mains, had been appointed in 1897 at the old Infirmary site. He laid down strict rules for patients and staff, not only in the smallpox hospital but also in the infectious diseases pavilions of the main hospital. Nurses disregarding regulations designed to limit cross infection could be instantly dismissed 'with loss of certificate'. Despite being a martinet, Dr. Ker was a respected and much loved physician whose premature death from pneumonia in 1925 dealt the hospital a severe blow. Dr. Ker's successor was the talented, academic doctor, Walter T. Benson, who unfortunately retired in 1936 on the grounds of ill health.

The third Medical Superintendent, Dr. Alec Joe, saw the hospital through the shortages of staff and equipment of the Second World War and, in 1948, the introduction of the NHS which transferred the management of infectious disease in-patients from the local authority to the newly formed South-Eastern Regional Hospital Board, Scotland. In 1942 Dr. Joe and his staff coped with 36 patients with smallpox in Edinburgh's final outbreak and in 1947, when poliomyelitis was rife, six or seven iron lung respirators were in use at any one time in the hospital. Perhaps the most exciting events of Dr. Joe's career were the advent first of sulphonamides and then of penicillin. He marvelled at the beneficial effect these drugs had on his patients with erysipelas and meningococcal infection.

The City Hospital had a significant role to play from the start of the 20th century in Sir Robert Philip's Edinburgh Tuberculosis Scheme. Patients with moderately severe tuberculosis went to the (Royal) Victoria Hospital and some of the convalescents to the farm colony at Polton. Those with advanced, cavitating, open tuberculosis were isolated at the City Hospital where it was expected they would soon die. With the efficient, kindly nursing and the fresh air from the Pentland Hills, however, more of these patients survived than was ever intended and a few recovered sufficiently to be discharged home. This had certainly not been part of Sir Robert's original scheme! Several pavilions were devoted to tuberculous patients and the two-man revolving shelters within the grounds proved particularly popular. Later on Professor Sir John Fraser looked after children with bone and joint tuberculosis at the City Hospital.

In the 1950s Professor (later Sir) John Crofton, Edinburgh's third Professor of Tuberculosis, moved his team to the City Hospital where he pioneered triple drug therapy. He showed that, with meticulous care and follow-up, patients not only got better on this treatment but their tubercle bacilli did not become resistant which had almost invariably happened previously using streptomycin injections alone. In 1958 Dr. (later Professor) James Williamson and Mr. Alec Welstead, the Group Secretary and Treasurer, led the successful mass chest X-ray campaign which resulted in almost all Edinburgh's adults being screened. By better detection of the disease and the application of the usually curative triple therapy, the incidence of and mortality from tuberculosis fell sharply and some of the stigma of the illness was lifted.

Another important member of Sir John Crofton's team was the dedicated bacteriologist, Dr. Archie Wallace, whose painstaking diagnostic and sensitivity testing skills were essential to the campaign against tuberculosis. Since 1903 the hospital had emphasised the teaching of nurses and medical students and the importance of laboratory back up. From the early days of simply helping clinicians

to differentiate between streptococcal and diphtheritic illness to the later sophisticated techniques covering all the bacterial pathogens, the laboratories have proved their worth. In the 1960s interest in non-bacterial respiratory tract organisms led to the formation of the Wellcome Trust Virus Laboratory which took over the upper part of Pavilion 4. This is now the prestigious Regional Clinical Virus Laboratory, which deals with viruses such as hepatitis and HIV and some non-virological organisms. It now serves all the Edinburgh hospitals and general practices. Later the Scottish Mycobacterial Reference Laboratory came to the City Hospital adding lustre to the already excellent microbiological services provided.

Much needed blood gas analysis, respiratory function testing and ECG services were provided in Dr. G. J. R. McHardy's laboratory. Later Professor David Flenley, who succeeded Sir John Crofton, obtained funding for the Rayne Laboratory which was built at the City Hospital for his research in emphysema.

Among the leading physicians forming Sir John Crofton's team against tuberculosis were Drs A. C. Douglas, I. W. B. Grant, James Williamson and N. W. Horne. In succession to Dr. Joe, Dr. Horne was Physician Superintendent from 1960-74. A much respected chest physician, he soon showed himself also to be an able administrator. Together with Mr. Alec Welstead, Dr. Horne spearheaded an ambitious programme of new building and the refurbishing of the old pavilions at the City Hospital. Among their many other achievements were the new medical residency, non-resident staff changing rooms, pharmacy and stores and lastly a brand new kitchen and dining room whose excellent cuisine raised the morale of staff and patients. The former servants' home was redeveloped to provide a new X-ray department and out-patient clinic on the ground floor and, upstairs, offices and a medical reading room.

A strong thoracic surgical team under Mr. Andrew Logan and his successors initially carried out lobectomy and pneumonectomy operations for tuberculosis and subsequently cardiac surgery starting with mitral valvotomies for rheumatic heart disease. Later on surgery for carcinoma of the bronchus and oesophagus was performed and more sophisticated heart operations. Before the thoracic surgery unit moved to the Royal Infirmary in 1997, Mr. W. Walker, the cardio-thoracic surgeon who pioneered minimal access thoracic techniques, had successfully removed a lung from a 12 year old boy using 'key hole' surgery.

As fewer infectious diseases beds were needed in the wake of immunisation and antibiotics, ENT surgeons took over the refurbished scarlet fever pavilions in 1965. They were also at the cutting edge of new developments with stapedectomies, cochlear implants and, under Professor Arnold Maran, extensive head and neck surgery often concentrating on cancer of the larynx. Times had certainly changed since the hey-day of almost indiscriminate tonsillectomies and adenoidectomies of earlier years. In 1990 the maxillofacial surgeons under Mr. G. Lello and Mr. R. Mitchell shared some of the ENT surgical pavilions and introduced new techniques for facial reconstruction following trauma and malignant disease.

In 1978 Dr. David Flenley succeeded Sir John Crofton. In his all too short time in the chair, Professor Flenley concentrated his internationally renowned research on obstructive airways disease. Through the generosity of the Rayne Foundation and the Salvesen Emphysema Research Trust he built up a team of experts in respiratory medicine some of whom worked in the newly constructed Rayne Laboratory at the City Hospital. Professor Flenley's sudden and premature death in 1989 robbed the hospital and respiratory medicine of one of its leading lights. Under Professor Flenley's successor, Professor Chris Haslett, whose interests are in white blood cell migration and the adult respiratory distress syndrome, the Respiratory Medicine Unit left the City Hospital for the Royal Infirmary in 1993.

In the City Hospital's Regional Infectious Diseases Unit (RIDU), Dr. J. McC. Murdoch was appointed in 1960 to succeed Dr. Alec Joe. Dr. Murdoch had clinical responsibility for the infectious diseases wards but was not in administrative charge of the whole hospital, which was under the care of Dr. N. W. Horne. Dr. Murdoch became recognised internationally in antibiotic medicine and in the management of urinary tract infection. He was ably assisted by Dr. George Sangster who possessed legendary skills in diagnosing obscure infections. In 1969-70 the RIDU coped with a virulent outbreak of hepatitis type B, which claimed the lives of several members of staff from other hospitals.

In the early 1980s, just as the RIDU was contracting further, particularly with its paediatric component, Edinburgh was hit by a wave of intravenous heroin misuse which coincided with the introduction of HIV infection into the city. Intense needle sharing in some deprived areas of Edinburgh quickly infected drug misusers with HIV. Dr. Ray Brettle, who had been appointed to the RIDU in 1983 after Dr. Murdoch retired, grasped the nettle of managing these difficult patients. In 1985 he set up at the City Hospital the UK's first confidential, self-referral, HIV screening clinic for people who felt they might be at risk of infection.

A new out-patient clinic was formed at one end of the infectious diseases corridor and a HIV-dedicated in-patient ward at the other end. The services which Dr. Brettle set up in the hospital, prisons and out-reach clinics became a model for workers managing HIV-infected drug misusers elsewhere. Milestone House, the first purpose-built AIDS hospice in the UK, was erected near the hospital's west gate and was initially funded jointly by the Lothian Health Board and the Waverley Care Trust.

As the measles, chickenpox and whooping cough admissions diminished, pavilions 18-21 fell vacant. First to move in was the Tropical Diseases Unit from the Eastern General Hospital under the care of Dr. F. J. Wright. Tropical disease screening of colonial service employees and VSO workers formed most of the work and there was a small in-patient commitment. When Dr. Wright retired in 1973 the work of the tropical diseases department was taken over by the RIDU which also screened and assessed ex-Far East Prisoners of War for their war pensions.

In the meantime, some of the wards had been occupied by long-term elderly patients. Dr. James Williamson, formerly a tuberculosis chest physician with Sir John Crofton, assumed responsibility for these patients until 1972 when he was appointed to the first chair of geriatric medicine in the University of Liverpool. Professor Williamson returned in 1976 as Edinburgh's first Professor of Geriatric Medicine and set up the Care of the Elderly Unit at the City Hospital. Much refurbishment of Pavilions 18-21 ensued and a day care assessment centre was established in the former typhus Ward 22. Professor Williamson concentrated all his medical, nursing and paramedical team on the same site and he laid particular emphasis on teaching and the rapid assessment of elderly patients referred from general practice. The service he provided in the emerging discipline of geriatric medicine became the gold standard for the UK.

In 1948 Dr. Joe had expressed concern over the transfer of the City Hospital from the local authority to the South-Eastern Regional Hospital Board, Scotland, of the new NHS. Fortunately the change over was smooth and the hospital's links with the city's Public Health Department remained strong. Since then and particularly after 1974 when the hospital came under the aegis of the newly formed Lothian Health Board, there was a bewildering number of changes in management and in the grouping together of different hospitals, clinics and districts. By 1993-4 the City Hospital was incorporated into the Edinburgh Royal Infirmary and Associated Hospitals NHS Trust and in 1999 what remained of the City Hospital came under the newly named Lothian University Hospitals NHS Trust.

During much of this time consultations were taking place over the planning of a new Royal Infirmary and the decision was at last made in 1994 to build it in conjunction with a relocated Medical School at Little France to the south east of Edinburgh. This had echoes of the City Hospital's last move from Infirmary Street out to Colinton Mains Farm nearly 100 years before. Among the sacrifices to be made for the new Royal Infirmary were the closure of Princess Margaret Rose Orthopaedic Hospital and the City Hospital. Lacking an intensive care unit, the City Hospital was already at a serious disadvantage as a viable hospital caring for acutely ill patients. In addition, the nurses' home, badly damaged by fire in 1988, had subsequently been demolished.

The Care of the Elderly Unit, now under the charge of Professor W. J. MacLennan, was gradually closed down, and Respiratory Medicine under Professor Haslett moved to the Royal Infirmary in 1993 followed by Thoracic Surgery in 1997. In the summer of 1998 the Maxillofacial Surgery Unit was transferred to St John's Hospital at Howden and the RIDU moved to a prefabricated building at the Western General Hospital. The remaining paediatric beds and cots of the RIDU were transferred to the Royal Hospital for Sick Children.

After the hospital's bacteriology laboratory moved to the Medical School, only the Scottish Mycobacterial Reference Laboratory, the Regional Clinical Virus Laboratory and the ENT Surgery

Unit remained at the City Hospital whilst awaiting for accommodation to become available either at Little France or at the Western General Hospital.

Redevelopment of old sites and buildings around the City Hospital was continuously taking place. The Craiglockhart Poorhouse, later Greenlea Old Peoples' Home, became The Steils, a prestigious new housing complex. Wester Hill estate has grown up where the temporary smallpox hospital used to be. The City Hospital itself is being developed for private houses by Morrison Homes and Cala (Scotland) Homes.

This brief account of the Edinburgh City Hospital should also acknowledge the contribution of the nurses who risked their lives, some indeed succumbing, by looking after patients with then untreatable illnesses and, more recently, those who cope bravely with sometimes abusive HIV-infected drug misusers. The social workers, dieticians, and all those in the professions allied to medicine also deserve recognition. The laboratories, X-ray department, pharmacy and stores, records department and secretarial staff, the caterers, estate managers, domestics and porters have all made valuable contributions.

For a number of years the upgraded City Hospital laundry catered for the needs of almost all the hospitals in the area. From 1985 the hospital became home to the Area Sterilising Service, which cleaned, sterilised and packed instruments and theatre linen for many other hospitals. The library service for patients and the medical reading room for staff were both much appreciated. The City Hospital chaplains have unstintingly given spiritual comfort to patients, relatives and staff at difficult times and in changing circumstances. The service provided by the City Hospital tea room volunteers since 1965 and the fund-raising by the hospital's League of Friends also deserve much praise.

It is for the contributions of people such as these to the citizens of Edinburgh for nearly a century that the City Hospital will be remembered best.

Footnote: For fuller details, sources and references please consult: Gray J.A. *'The Edinburgh City Hospital'* East Linton, Scotland: Tuckwell Press, 1999.

GLASGOW'S CONTRIBUTION TO NEW ZEALAND MEDICINE

Some of you will remember my initial venture into Antipodean medical history, a 1986 paper to the British Society for the History of Medicine conference on 'Medicine Furth of Scotland'.(1) The opening paragraph referred to the lack of research into the influence of Scottish medicine on Australasia and suggested a number of relevant lines of inquiry. The emphasis of the paper was quantitative, with some qualitative colouring and a few tentative conclusions. Today's offering is a reversal of this profile. It will stress the qualitative rather than quantitative, it will concentrate on New Zealand rather than Australia and, thirteen years on, it will be less tentative.

I have now spent almost a decade in New Zealand, working as a contract historian, with a strong bias towards medical history. For the past four years I have contributed a monthly medical history column to *New Zealand Doctor*. Not surprisingly, many of the contributions have a distinctly Scottish flavour.

There are several reasons for this. First and foremost, I feel a natural affinity with the Scottish doctors who emigrated to New Zealand. When I first went to Dunedin as a visiting fellow in 1989 I assumed that no one would notice my nationality since, as an educated Scot, I was without accent. That misapprehension was bolstered by the discovery soon after my arrival of Dr Fulton's *Medical Practice in Otago and Southland in the Early Days*, a pot-pourri of biographical sketches published in the 1920s. Of Dr John Gibson Smith, the son of a Lanarkshire farmer, who practised in Balclutha in the second half of last century, Fulton wrote: 'He wore a neatly trimmed grey beard, and had a pleasant musical voice, quite free from the accent one would have expected from a person trained and brought up in Scotland.'(2) When I first quoted this - while stroking my neatly trimmed grey(ish) beard - I could not understand why the audience laughed so heartily, but it was quickly made clear to me that I could not emulate Smith's neutral tones. Soon afterwards I was cheered by the discovery of a second observation about the impact of a Scottish accent in New Zealand. Dr James Malcolm Mason emigrated to the colony in 1895, served as the country's first Chief Health Officer from 1900 to 1909, and remained until his death some forty years later. As one obituarist commented: 'Dr Mason was a man of Arbroath, and his Scots tongue agreeably accented the many stories of a doctor's life with which he lightened the afflictions of his patients.'(3)

There is, however, a second and more compelling reason to focus upon the Scottish medical diaspora, for Scots, or at least doctors trained in Scotland, have wielded a disproportionate influence in New Zealand medical circles. I have to confess there is also an evangelical aspect to my interest, for I continue to become irate when English and Scots doctors are conflated by historians and others as 'British'.

The assumption, which I shared at the time of my 1986 paper, has always been that it was almost exclusively Edinburgh medicine which exercised an influence over events in New Zealand, most noticeably through links with the Otago Medical School in Dunedin and the subsequent flood of New Zealand applicants seeking higher qualifications through the two Edinburgh colleges. This orthodoxy was based in large measure on a paper by Rex Wright-St Clair to the 23rd International Congress on the History of Medicine in 1974.(4) As some of you know, Rex penned the definitive biography of Sir David Monro, son of Monro Tertius, who fled to New Zealand rather than face the prospect of becoming 'Monro Quartus'. There he became a wealthy sheep farmer and politician.(5)

Monro was only one of a number of late 1830s Edinburgh graduates who migrated to the infant colony.(6) One of the most prominent was John Logan Campbell, son of a well-known Edinburgh doctor. Campbell was so eager to shake the dust of Edinburgh from his feet that in 1839 he became the first MD candidate to graduate in absentia.(7) On arrival in Auckland he wasted no time in establishing various business enterprises, an altogether more profitable calling than medicine. As he wrote to his father in 1846 after one particularly adroit deal: "What a botheration lot of pulses one would require to feel, tongues to look at & prescriptions to write before the fees would come up to the above sum and as for a poor devil of a Dentist he would require ... to slay at least 310 teeth before he made £105."(8)

A third member of this club was Arthur Saunders Thomson. Stationed in Auckland as an army medical officer from 1847-58, Thomson wrote the first general history of New Zealand.(9) A fourth early medical arrival in New Zealand, though with an LRCSEd rather than an MD, was the younger brother of Edinburgh's notorious anatomist, Robert Knox. Frederick, the former conservator of Robert's anatomical museum, was appointed Wellington's first librarian in 1840 at a salary of £75 p.a., more than he could have earned through medicine at that time. He had a particular interest in the anatomy of the whale, having dissected a large specimen cast ashore near North Berwick in 1831. During his last decade - he died in 1873 - Frederick revived this interest with a series of talks to the fledgling New Zealand Institute (now the Royal Society of New Zealand) on whales.(10)

As someone who graduated from Edinburgh but worked for a decade in Glasgow University Archives, I was more keenly aware than a New Zealand historian would have been about the respective impact of the two centres. Despite the high profile Edinburgh men listed above, it seems to me that the Glasgow connection has been almost as important in shaping New Zealand medicine. My interest was first aroused by the visits to New Zealand of two Glasgow medical professors, Joseph Coats and William Macewen, who came to the country in 1897 and 1923 respectively. The remainder of this paper will consider the wider implications of the Glasgow influence.

Joseph Coats taught pathology in Glasgow during the last three decades of the nineteenth century and in 1894 was appointed inaugural professor of pathology in the University of Glasgow. Although regarded by some colleagues as a rather distant figure - photographs reveal a dignified but apparently aloof individual - Coats was quite the opposite in practice. In 1876 he joined the Western Medical Club, an elite dining club, and presided over the January 1891 dinner in Glasgow's Grand Hotel.(11) That same year he was a prime mover in the formation of the Glasgow University Club and chaired its meetings until his untimely death in 1899. He was also one of the first of the university teachers to host student parties in his home - brave man!(12)

Coats had a major input to the professionalisation of Glasgow medicine, co-founding the Glasgow Pathological and Clinical Society in 1873 and later filling the presidential chair (1876-8). This was only one of three over-lapping roles which he assumed in the mid 1870s. He was also secretary (1875-82) and president (1891-3) of the Glasgow Medico-Chirurgical Society and editor of the *Glasgow Medical Journal (GMJ)* (1877-1899).(13)

In the second half of the nineteenth century the long sea voyage to New Zealand was often recommended as a last resort for the seriously ill. Doctors and medical students were among the most assiduous followers of this advice. One of the earliest casualties of such a venture was Andrew

Stevenson, a Glasgow medical student who died of tuberculosis in Auckland in March 1861 aged just 21.(14) Joseph Coats, who became a medical student just two years after Stevenson's death, followed in his footsteps in 1897, after a six month stay in the Channel Islands failed to cure the mysterious symptoms which had baffled his medical advisers. His diary of the trip, published upon his return to Glasgow in April 1898, sheds some light on the expatriate medical community, and the continuing influence of 'the Chief' as he was known to generations of students.(15)

During a short stay in Christchurch Coats met up with several of his former students, of whom more anon. From there he travelled south, reaching Dunedin in mid December. There he lunched with Mr Gairdner, manager of the New Zealand Bible Depot and a cousin of his old teacher and valued friend, William Tennant Gairdner, professor of medicine at Glasgow (1862-1900).(16)

None of the Dunedin contacts named by Coats were Glasgow University men, which partly reinforces Wright-St Clair's 1974 comments. The medical school and hospital staff with whom he dined and conversed were almost all trained in Edinburgh or England, though Coats rather plaintively claimed Daniel Colquhoun, a London graduate who was lecturer in medicine and a former editor of the *New Zealand Medical Journal (NZMJ)*, as a native of Glasgow.(17)

Coats fared better in the North Island. At New Plymouth he met up with William Christie, the son of a Glasgow ironfounder and one of his old students, who had graduated in 1885 and settled in New Zealand in 1890. Christie's assistant, Ernest Walker, also came from Glasgow industrial community, as the son of a chemical merchant. He arrived from Scotland in 1898, having graduated from Glasgow with first class honours in 1895. There is no obvious connection between the two men, given the age gap, and one wonders whether Coats played any part in bringing them together.(18)

Sadly, there is no happy ending to Coats's trip. He arrived home in July 1898, became seriously ill in November of that year, and died post-operatively on 24 January 1899, some twelve months after his departure from New Zealand.(19)

Our second Glasgow visitor, Sir William Macewen, was professor of surgery at Glasgow from 1892-1924. Another imposing but austere figure he, like Coats, inspired loyalty among his former students. Macewen visited New Zealand in 1923 en route for Melbourne and the 1923 Australasian Medical Congress in his role as president of the British Medical Association. His stay, restricted to a two-day stopover in Wellington, was further curtailed when immediately upon arrival he was whisked off to Government House to meet Governor General Lord Jellicoe; the two men had become friends during World War One when Jellicoe was First Sea Lord and Macewen acted as the Royal Navy's surgeon-general in Scotland.(20)

Despite the brevity of the visit, the impact on the local medical community was considerable. Following his private reunion with Jellicoe, the doctors claimed Macewen as their own. That evening Dr William Herbert, a New Zealander who had been educated at Edinburgh University and was now president of the New Zealand branch of the BMA, hosted a dinner for their distinguished guest. Writing to his wife the next day, Macewen enthused about this 'great banquet', the attendance at which exceeded any previous gathering of New Zealand medical men. 'Saw many old Glasgow students', he wrote, 'and many more also who were from London & other parts of England.' He did not mention Edinburgh.(21)

In a second letter to his daughter Daisy, Macewen supplied additional information. Some of the forty or so doctors in attendance had travelled almost 400 miles to be present. Others would have attended, he stated, had his ship not docked forty-eight hours ahead of schedule. As a concession to the difficulties of getting to Wellington, Dr Herbert had decreed that 'In order to meet the conveniences of many of your old students who are travelling by motor a great distance to be present, I have made the dress a dinner jacket.' One wonders what he would have specified for a formal occasion!(22)

Less than twenty-four hours after reaching New Zealand, the BMA president set sail for Sydney. In the previously quoted letter to his wife, Macewen noted that a hearty invitation had been extended to return to New Zealand for a lecture tour. 'They must think me very young', he concluded. He was then seventy-five years old.

As with Joseph Coats 24 years earlier, the rigours of travel proved too much for Macewen. On 13 February 1924, shortly after his return to Britain he reported on his trip to the BMA Council in London. Less than six weeks later he was dead, following an attack of influenza.(23)

When placed in context, the accounts of these journeys are of considerable historical value, for Coats and Macewen shared more than an unfortunate reaction to Antipodean travel. The remainder of this paper will explore some of these parallels and the implications for the history of New Zealand medicine.

Although their visits were separated by almost exactly a quarter of a century, the two men were near contemporaries. Coats graduated from Glasgow University in May 1867; Macewen followed suit in 1869. Both had been participants in Joseph Lister's pioneering work on antisepsis, carried out at the Glasgow Royal Infirmary (GRI) in the mid 1860s; Coats was actually one of the first to publish an account of antiseptic treatment, in a letter to *The Lancet* of 2 May 1868.(24) Six months later, he was appointed one of Lister's assistants at the Infirmary. When the latter was elevated to the peerage in 1897 it was Coats who took the chair at the celebratory banquet, just weeks before his departure for New Zealand.

Macewen was another member of Lister's inner circle. Arguably the most talented of Lister's protegés, he was also one of the most precocious. During his visit to Wellington he captivated his audience with personal memories of pre-Listerian surgery. As befitted a man who was notoriously prone to use the 'Royal we' in conversation, Macewen was not averse to claiming some of the credit for Lister's success, having boosted the master's flagging spirits at a time when Lister was discouraged by criticism: 'Never heed them, sir', he reported himself as saying, 'They are only midges and will disappear with the light.' "You think so," replied Lister, "and you are the youngest student here. Very will, I will go straight on."'(25)

Lister's impact, directly and indirectly, on New Zealand medicine was considerable. At the end of his first session as professor of surgery in Glasgow, he was presented with a testimonial in support of his application for clinical beds at the GRI, signed by 161 students.(26) The signatories were analysed in a 1965 *Glasgow Herald* article marking the centenary of antiseptic surgery. According to this, six of the 161 had settled in New Zealand under the government emigration scheme — a reference to Prime Minister Julius Vogel's 1870s policy to boost white settlement. In fact, eight, not six, came to New Zealand.(27)

The Glasgow doctors who settled in New Zealand gained a certain kudos from the Lister connection. This applied even to those who had not actually been taught by him. Dr John Guthrie, who graduated in 1874, practised in New Zealand from 1875 to 1896. A good example of this is the profile of Guthrie which appeared in McMillan's history of medicine in the New Zealand province of Canterbury: 'After qualifying at Glasgow University he held the posts of Resident House Surgeon and House Physician at the Glasgow Royal Infirmary, famous as the birthplace of Listerism.'(28)

Dr Edward Mackellar was another whose name was linked with Lister. His death in 1923, just three months after the *NZMJ*'s report of William Macewen's visit to Wellington, prompted the following appreciation by his *NZMJ* obituarist: '... educated at Edinburgh and at Glasgow, where he was associated with Lord Lister in some of the latter's discoveries'.(29) There are difficulties with the chronology of this claim. Mackellar first matriculated at the University of Glasgow in the autumn of 1869 and did not graduate until 1877. His arrival coincided with Lister's departure for Edinburgh, where he had been appointed to the chair of surgery. The 'association' must therefore have been short-lived.

A more credible scenario for Mackellar's Listerian connection is that the link came at second hand, through Joseph Coats and his pathology classes. In 1884, when Mackellar was appointed as Otago University's first lecturer in pathology, Coats's *GMJ* described him as one of Glasgow's 'most distinguished graduates'.(30) A decade later he was the only Auckland-based doctor to be mentioned in Coats's diary of his New Zealand trip.

Other alleged New Zealand links with Lister are equally tenuous. In 1922 R.V. Fulton claimed that the pioneer of antiseptic surgery in New Zealand had been Duncan Macgregor, Professor of Mental and Moral Philosophy at Otago University from 1870 to 1886. The claim, though subsequently shown to be erroneous,(31) was not as far-fetched as might at first appear. Macgregor had qualified in medicine shortly before accepting the Otago chair and later returned to the medical sphere as the colony's Inspector-General of Hospitals (1886-1906). Fulton's summary of his medical training, however, is suspect: 'In 1867 there entered the Edinburgh School of Medicine Duncan Macgregor, MA, of Aberdeen, and he quickly absorbed the best of Lister's teaching,

graduating MB CM in 1870.' Since Lister did not arrive in Edinburgh until late 1869, Macgregor must have had limited contact, though he claimed before operating on Fred Fulton (R.V.'s cousin) that he had walked the Edinburgh Infirmary under Lister's 'personal tuition'.(32)

In the light of these examples, it can be argued that the Lister-Coats-Macewen nexus created a special kind of camaraderie among the Glasgow graduates in New Zealand, based in part on the GRI. A good example of this fraternity can be found in the rather improbable setting of Christchurch, one of the four main population centres and quintessentially English in character. The previously mentioned John Guthrie was medical superintendent of the Christchurch Hospital from 1875 until 1877. His younger brother Thomas, who graduated from Glasgow in 1876, began practice at nearby Lincoln in 1878, before moving soon afterwards to Lyttelton (Christchurch's port).

In 1896 John Guthrie returned to Scotland where he reputedly lectured in surgery at the GRI, though his name does not appear in the staff lists in Comrie's *History of Scottish Medicine* (1932).(33) His departure coincided with the arrival in Christchurch of Dr Walter Fox, Macewen's house surgeon and assistant during many of his pioneering operations on the brain. Fox was earmarked to accompany Macewen when the latter was offered the Johns Hopkins chair of surgery in 1889. When Macewen rejected this opportunity, to remain in Glasgow, where he was rewarded with the University chair of surgery in 1892, Fox emigrated to Australia. He then joined Christchurch's Glasgow coterie, which included Dr Walter Thomas, the nephew of Dr Moses Thomas who was medical superintendent of the GRI from 1867 to 1902.(34) After graduating from Glasgow in 1873 Walter Thomas too had spent some time in Australia before entering into partnership with John Guthrie in 1882. Fox later recalled that Thomas's brother, a ship's surgeon on the Australasian run, had acted as his locum while he was still in Glasgow. It seems likely that this networking influenced Fox's decision to move to Christchurch, where he was almost immediately appointed medical superintendent to the local hospital.(35)

Such networks could embrace the second, and sometimes the third, generation, raising questions about the extent to which Glasgow teachers continued to influence former students who had travelled to the furthest corners of the globe. It was not uncommon for the children of doctors to be sent to their father's alma mater. When John Guthrie returned to Scotland, two of his sons accompanied him. They qualified in medicine at Glasgow University in 1902 and 1906 respectively. Both held residencies at the GRI before returning to Christchurch; their father, his paternal chaperoning completed, retired to New Zealand in 1908.(36)

Some non-medical families with Glasgow roots also sent their children home for this purpose. William (1895), Helen (1903), Annie (1905), James (1905) and John Baird (1910), the children of a Scots Presbyterian minister in Otago and Southland, all completed their medical degrees at Glasgow, where their maternal grandfather had practised as an architect. Just recently I came across another and more poignant example. Robert Hamilton emigrated from Kilmarnock in 1866 to join his brother in a farming venture in New Zealand. Seven years later he entered Glasgow as a medical student to meet the wishes of his mother who had remained in Scotland. He graduated in 1879 but did not return to New Zealand to practise until 1905.(37)

One element which I have not yet been able to factor in, is the extent to which the examples cited above fairly reflect the wider New Zealand pattern. What did influence local students in their choice of a Scottish, or British, medical school? It may be reasonable to suggest that local medical practitioners, if asked, would propose their own alma maters as a first option but I currently have insufficient data to verify or refute this conjecture. For those individuals whose circumstances can be verified, the results are sometimes ambiguous. For example, when William Macewen visited Sydney in 1923 he called on the aged widow of Dr George Pringle, one of Joseph Lister's Edinburgh contemporaries. James Hogarth Pringle (their son) had followed in his father's footsteps, graduating MB CM Edinburgh 1885; he subsequently became Macewen's surgical assistant at the GRI, presumably as a result of the common links with Lister.(38) This raises the question of the degree to which apostolic succession played a role in the shaping of Australian - and New Zealand - medicine.

There were, of course, other means of influence than direct association. At a time when there were no telephone links between Scotland and New Zealand, the written word was a crucial means of maintaining contact. The two principal avenues for this were medical journals and personal letters. The following paragraphs offer some insights into how this operated.

For a short time in 1884 and 1885, the *GMJ*'s 'Current Topics' included 'New Zealand notes', penned by an anonymous correspondent. The first of these columns noted the appointment of Edward Mackellar to the Otago Medical School. The second, and last, solemnly reported an attempt by a New Zealand parliamentarian to introduce legislation 'that all doctors once found drunk be struck off the New Zealand medical register'.(39) The reporter refrained from comment on the justification or otherwise for such a proposal.

At this remove it is impossible to identify the unnamed New Zealand correspondent, although there are several plausible candidates. These include the Guthrie brothers and Walter Thomas, all of whom maintained links with 'Home'. My own preferred candidate is a Dunedin doctor, Alexander Tinling Thomson, who practised in central Otago from 1869 until 1876, when he moved to Dunedin. Ten years later he returned to Scotland. His departure coincided with the demise of the *GMJ*'s short-lived New Zealand column, which tended to focus on events in Dunedin.

Thomson had been closely associated with both Lister and Coats. As a medical student in Glasgow from 1863 until 1867 he was an exact contemporary of the latter. Like Coats, Thomson claimed to have been a dresser at the time of the first historic use of antiseptic surgery in ward twenty-four of the GRI in 1865.(40) More than three decades after leaving Dunedin, Thomson supplied the biographer/chronicler R.V. Fulton with reminiscences of his career. He recalled that during his student days 'Typhus fever was not only very prevalent but very fatal, assistants dying one after another while attending the wards'.(41) One of those who nearly succumbed was Joseph Coats. He was hospitalised in one of the GRI's private rooms for about four weeks in 1865. The shared hazards of those days may have forged a continuing link between Thomson and Coats over the years.

Whatever its authorship, the *GMJ*'s 'New Zealand Notes' provided a conduit for the transfer of both personal and clinical information between Scotland and New Zealand. The first such column reported that 'The friends of Dr Wm. Stenhouse, a well known Glasgow graduate, will be sorry to learn that he had to submit to an amputation of the right foot lately, the result of an injury in boyhood.' The additional comment that Stenhouse had recently performed the first successful case of double ovariotomy in Dunedin strengthens the case for Tinling Thomson as the anonymous author. A former house physician and assistant to William Leishman, professor of midwifery at Glasgow from 1868 until 1894, Thomson had himself twice performed a successful ovariotomy in 1879, with one case reported in *The Lancet*.(42)

More detailed evaluation of the medical press reveals some very interesting parallels. In July 1890 Stenhouse published a report on the first successful Caesarean birth in New Zealand, conducted by himself in Dunedin. He explained that his growing opposition to craniotomy, after performing nineteen destructive operations since graduating from Glasgow in 1875, had made him determined to perform a Caesarean as soon as a favourable case presented itself. This transition, he asserted, was as much an ethical as a clinical consideration.(43)

Stenhouse's account struck a chord. His arguments paralleled those of John Stuart Nairne, a Glasgow doctor who had qualified in 1872, three years before Stenhouse. Nairne, too, had rebelled against performing craniotomies, and had voiced his revulsion at a meeting of the Glasgow Obstetrical and Gynaecological Society in February 1887. The paper, published later that year in the *Edinburgh Medical Journal*,(44) encouraged a Glasgow obstetrician, Dr Murdoch Cameron (MB CM 1870) to perform his first Caesarean in April 1888. This was reported in the *BMJ* on 26 January 1889; the account of the second in what was a series of successful operations appeared on 15 March 1890.

There are additional parallels between the strategies of Stenhouse and Cameron. The former attributed his success to a number of factors; these included the good health of the mother and the fact that 'no time was lost by futile attempts at delivery by other means'. Glasgow obstetric tradition has it that Cameron was in the habit of accosting rachitic pregnant women in the streets, handing them his card and urging them to come directly to the maternity hospital at the onset of labour in order to undergo a Caesarean. Had the two men arrived independently at this view, or did it owe something to their obstetric teacher, William Leishman?

Given the time lag in papers reaching New Zealand, it seems improbable that Stenhouse had read Cameron's *BMJ* article before publishing the account of his own approach. But had he been informed by letter of Cameron's earlier successes? We may never know, although there are grounds for believing that Stenhouse had maintained contact with his alma mater, and those of his

contemporaries who were involved with obstetrics and gynaecology colleagues. When W.I. Addison's *Roll of the Graduates* was published in 1897 Stenhouse was the only New Zealand subscriber to be listed in the appendix. His own entry, describing him as '(honorary Physician, Dunedin Hospital, etc., etc., etc.'(45) was also unusually effusive compared with the norm for the volume.

Finding concrete evidence of contacts on a personal level is not easy; few collections of letters are known to survive. One exception is the surviving correspondence of John Malcolm, the first Professor of Pathology in the Otago Medical School (1905-43). The vast majority of Malcolm's letters were to family members left behind in Scotland but the collection does include a dozen communications to his old Edinburgh professor, Edward Sharpey Schafer, covering the first eight years of Malcolm's tenure. These have been commented upon in a short article by Douglas Taylor, emeritus professor of physiology at the Otago Medical School and the Monro family's bibliographer.(46) The article reveals how, isolated from the wider academic world, Malcolm used Schafer as a confidant and sounding board.(47)

The most voluminous example unearthed to date of a New Zealand doctor's correspondence with 'Home' are the papers of Dr Mason, the colony's first Chief Health Officer. These languish, unlisted, in Wellington's Alexander Turnbull Library. A preliminary evaluation while writing the history of New Zealand's Health Department demonstrated the problems of using such raw data. It is difficult, perhaps impossible, to interpret Mason's correspondence without a detailed knowledge of both the Scottish and New Zealand settings.

Mason's mentor for much of his career was Dr Alexander Robertson, lecturer in medicine at the GRI Medical School (later St Mungo's College) from 1889 until 1901. In 1887, when Mason was awarded the Scottish Triple Qualification, his certificate was signed by Robertson as FPSG Inspector. Mason emigrated to New Zealand in 1895 and corresponded regularly with Robertson until at least 1906. The letters were a conduit for clinical information and medical gossip. Robertson, for example, aided Mason in drawing up the Health Department's agenda, keeping his former student up to date with current advances in treatment for tuberculosis and venereal disease.(48) In return, Mason praised his old teacher in the *NZMJ*, which he edited from 1900 to 1905. At times Mason seems to have forgotten he was in New Zealand rather than Scotland. In 1902, for instance, his editorial lauding Robertson's 'thoughtful and thought-inducing' essay on the hallucinatory effects of alcohol identified neither the writer (referred to simply as 'Dr Robertson') nor the title of his publication. It is unlikely that the wider community of New Zealand doctors could have recognised either from Mason's synopsis.(49)

Mason also corresponded with William Macewen, who had lectured at the GRI before his 1892 university appointment, and supplied his old teacher with a number of Moriori skulls (from the Chatham Islands) in 1906. Macewen's tardiness in acknowledging this gift brought a stern rebuke from Robertson, who had obviously been kept aware of developments.(50)

Macewen's correspondence reveals other ties between the three men and New Zealand. During his stay in Wellington, Macewen had little time for meeting friends or acquaintances, although he did manage to rendezvous with Robertson's sons, Alexander Jnr and Peter. Alexander, who had settled in Wellington in 1913, some seventeen years after graduating MB CM Glasgow in 1896, was obviously satisfied with the move for according to Macewen he 'is now very stout and is married'. Peter, who had spent five years as Macewen's assistant after qualifying in 1903, had joined his brother in 1921, not long before Macewen's visit, and was still homesick for Glasgow. It is tempting to detect Mason's influence in the brothers' decisions to move to Wellington in mid-career.(51)

Sadly, the Macewen-Mason-Robertson story had no happy ending. Macewen died in March 1924, to be followed less than two months later by James Mason, while Peter Robertson died of meningitis and septicaemia in July 1925, aged 49.(52) The passing of Macewen severed the last direct link back to Joseph Lister and almost certainly lessened the appeal of Glasgow as a fount of medical knowledge, a trend reinforced by the development of the Otago Medical School from the 1890s. For half a century, however, the Glasgow connection had played an important part in the evolution of New Zealand medicine.

CONCLUSION

Taken in their entirety, the findings described in this paper suggest more extensive and meaningful contacts between the Glasgow Medical School and its New Zealand-based graduates than previously acknowledged. Coats and Macewen, as two of Lister's associates at the moment he cemented his place in history, occupied a special place in the minds of many of their students. This was reflected, and reinforced, during their visits to New Zealand, visits which were of themselves unique enough amongst academic doctors of that era to be of interest. The Mason-Robertson and Malcolm-Shafer letters suggest that other teacher-pupil relationships were equally important in succouring doctors abroad. Indeed, it may have been the distances between them which encouraged a franker exchange of views than would have been possible in Glasgow or Edinburgh.

I promised at the start that this paper would be less tentative than the one I delivered in 1986. It is, but it perhaps raises almost as many questions as answers. How typical were the links described here? Were the medical teachers of Edinburgh and Glasgow on equally relaxed terms with their former students? Were Coats and Macewen the exceptions rather than the norm? The challenge is there for historians to re-assess the Scottish influence on New Zealand medicine and, by extension, on practice in the other white dominions of Canada, South Africa and Australia, which swallowed up a goodly proportion of the over-supply of Scottish doctors in the nineteenth and early twentieth centuries.

1. D.A. Dow, 'Scotland and Australasia', in D.A. Dow (ed), *The Influence of Scottish Medicine*, Carnforth, 1988, pp.129-41.
2. R.V. Fulton, *Medical Practice in Otago and Southland in the Early Days*, Dunedin, 1922, p.104.
3. Unidentified obituary notice, Mason Papers, Alexander Turnbull Library, Wellington, New Zealand. For a summary of Mason's career see D.A. Dow, 'James Malcolm Mason', in *The Dictionary of New Zealand Biography: Volume 3. 1900-1920*, Wellington, 1996, pp.335-6 and D.A. Dow, '"Here is my habitation": Dr James Malcolm Mason and Otaki', *Historical Journal/Otaki Historical Society*, 1997, vol.20, pp.54-7.
4. R.E. Wright-St Clair, 'The Edinburgh influence on New Zealand medicine', *Proceedings of the XXIII International Congress of the History of Medicine*, London, 1974, vol.1, pp.748-53.
5. R.E. Wright-St Clair, *Thoroughly a Man of the World: A Biography of Sir David Monro MD*, Christchurch, 1971. See also R.E. Wright-St Clair, *Doctors Monro: A Medical Saga*, London, 1964.
6. After giving this lecture I had an opportunity to check on the student records of the 19 individuals who graduated MD Edinburgh from 1834 to 1839 and subsequently ended up in New Zealand. The distribution of place of birth/residence as recorded in the matriculation albums was Edinburgh (3), elsewhere in Scotland (5), England (4), Ireland (3), India (2), Barbados (1), not given (1).
7. R.E. Wright-St Clair, 'The medical qualifications of Sir John Logan Campbell', *New Zealand Medical Journal (NZMJ)*, 1983, vol.96, pp.212-13.
8. R.C.J. Stone, *Young Logan Campbell*, Auckland, 1982, p.128. Stone speculates that Campbell may have intended to write 210, not 310 - i.e. 10 shillings per extraction.
9. A.S. Thomson, *The Story of New Zealand*, London, 1859. See also R.E. Wright-St Clair, 'Surgeon Thomson of the 58th Foot', *NZMJ*, 1976, vol.84, pp.153-6 and M. Belgrave, 'Thomson, Arthur Saunders 1817?-1860', *The Dictionary of New Zealand Biography: Volume 1: 1769-1869*, Wellington, 1990, pp.534-6.
10. D.A. Dow, 'Anatomy preoccupies the mistaken Dr Knox', *New Zealand Doctor*, 17 April 1996, p.48.
11. A.A. Clark, *Just a Minute or Two: The History of the Western Medical Club 1845-1902*, Glasgow, 1994, pp.225, 239.
12. *Doctor & Mrs Joseph Coats: A Book of Remembrance*, compiled by their daughters, Jackson, Wylie & Co, Glasgow, 1929, pp.49ff, 69.
13. ibid, pp.49-57.
14. See R.E. Wright-St Clair, 'Causes of death in colonial doctors', *NZMJ*, 1978, vol.88, pp.49-51. The author's survey of 910 New Zealand doctors who practised from 1840 to c.1910, and who died prior to 1951, found that at least 47 (almost 5%) died of pulmonary tuberculosis.

15. ibid, pp.36-7; J. Coats, 1898, *Notes on Land and Sea: A Diary*, Glasgow 1898. Coats described this as the diary of a Journey to New Zealand, Australia, Ceylon and Egypt (October 1897, till April 1898).
16. ibid, p 103.
17. ibid, p 107. Colquhoun ultimately suffered a fate akin to that of Coats. Having retired to London in 1918 he decided to revisit New Zealand in 1933, became ill on the voyage out, and died in Dunedin in February 1935 aged 85.
18. ibid, p 132-3. Christie, who had recently imported the first x-ray machine to the Taranaki district, graduated 3 years after John Macintyre who established one of the first clincial X-ray hospital departments at the GRI. There is no way of knowing whether this helped foster Christie's interest in the new field.
19. For obituaries of Joseph Coats see *GMJ*, 1899, vol.51, pp.108-18, *Edinburgh Medical Journal (EMJ)*, 1899, vol.5, p.321, *British Medical Journal (BMJ)*, 1899, vol.1, p.317 & *Lancet*, 1899, vol.1, p.271.
20. William Macewen to Daisy Macewen, 10 October 1923, Glasgow University Archives (GUA), DC79/25; A.K. Bowman, *The Life and Teaching of Sir William Macewen: A Chapter in the History of Surgery*, Glasgow, 1942, p.389; Jellicoe was one of 10 patrons, mainly state governors, of the 1923 Australian Medical Congress
21. William Macewen to Mary Macewen, 11 October 1923, GUA, DC79/16
22. William Macewen to Daisy Macewen, 11 October 1923, GUA, DC79/16; Herbert to Macewen, 9 October 1923, Macewen Correspondence, Royal College of Physicians of Surgeons of Glasgow, RCPSG10, box 18, file 11.
23. Bowman, p.404.
24. *Doctor & Mrs Joseph Coats*, 1929, pp.12, 114; R.B. Fisher, *Joseph Lister 1827-1912*, London, 1977, p.162 wrongly identified the author of this article as James Coats.
25. *NZMJ* editorial, 'A retrospect', 1923, vol.22, pp.347-9.
26. The Infirmary managers had rejected his initial application in order to make the point that university professors had no right of appointment to their wards. See R.B. Fisher, *Joseph Lister 1872-1912*, London, 1977, pp.102-3.
27. E. Munro, 'Tracing Lister's students', *Glasgow Herald*, 23 January 1965, & E. Munro, 'Personal memories of Lister and his students', *Glasgow Herald*, 3 July 1965.
28. D. McMillan, *Byways of History and Medicine, With Special Reference to Canterbury, New Zealand*, Christchurch, 1946, pp.368-9.
29. *NZMJ*, 1924, vol.23, p.85.
30. *GMJ*, March 1884, pp.220-1. In the event Mackellar settled in the rapidly expanding city of Auckland in preference to accepting the lectureship in Dunedin.
31. The first New Zealand surgeon to adopt Lister's technique was Ryley of Hokitika, in 1868. See R.E. Wright-St Clair, 'J. Rutherford Ryley: pioneer of antisepsis in New Zealand', *Journal of Medical Biography* (1999), vol.7.1, pp.32-34. Although Ryley, who qualified LRCSEd in 1862, claimed to have been one of Lister's students his name does not appear in any Glasgow matriculation records or surviving class lists.
32. Fulton, pp.292-3.
33. For Guthrie's obituary see *NZMJ*, 1922, vol.21, p.301. His brother, Sir James, was President of the Royal Scottish Academy (1902-19).
34. McMillan, 1946, *Byways*, pp 368-9. Obituaries of Thomas & John Guthrie appear in *NZMJ*, 1919, vol 18, p 67 & *NZMJ*, 1922, vol 21, p 301; for Walter Thomas, see McMillan, 1946, *Byways*, p 370
35. For Fox's career see McMillan, pp.371-3, Bowman, p.310, and *NZMJ* obituary, 1945, vol.44, pp.151-2.
36. For obituaries of John Jnr & Robert N. Guthrie see *NZMJ*, 1942, vol.41, pp.184-5 & *NZMJ*, 1953, vol.52, p.501; for T.E. Guthrie see obituary, *BMJ*, 1916, vol.2, p.123. The only member of the family to break with tradition was Thomas Guthrie's son, Thomas Errol Guthrie, who graduated MB ChB Edinburgh in 1909.
37. For a partial account of his career see L. Walker, 'The Hamiltons of Awhitu', *Journal of the Auckland-Waikato Historical Societies*, 1979, vol.34, pp.1-7.

38. For Pringle's career see obituaries in *GMJ*, 1941, vol.17, pp.153-7, *BMJ*, 1941, vol.1, p.734, & *Lancet*, 1941. vol.1, p.651.
39. *GMJ*, March 1884, pp.220-1, and February 1885, pp.95-7.
40. Interestingly, Coats's name does not appear in the list of associates produced for the Lister Centenary Exhibition of 1927, although Thomson's is there. See Anon, 1927, *Lister Centenary Exhibition at the Wellcome Historical Medical Museum*, London, p.65.
41. Fulton, pp.250-2.
42. *GMJ*, March 1884, p 221; Fulton, 1922, *Medical Practice*, p 252; *Lancet*, 1879, vol.1, p 895. Thomson later recalled using a clamp for the pedicle which he had constructed in a 'blacksmith's shop somewhere near the Octagon' [then, as now, Dunedin's civic centre]. See D. Dow, 'Blacksmiths made tools for early ovary surgery', *New Zealand Doctor*, 13 May 1998, p.41.
43. W.Stenhouse, 'Successful Case of Caesarean Section', *NZMJ*, 1890, vol.3, pp.225-30.
44. For Nairne's influence on Glasgow medicine see D.A. Dow, *The Rottenrow: The History of the Glasgow Royal Maternity Hospital 1834-1984*, Carnforth, 1984, p.67 and D.A. Dow *The Royal Samaritan Hospital for Women, Glasgow 1886-1986*, Glasgow, 1986, p.13.
    For an extended analysis of Stenhouse's activites see D. Dow, 'Barbaric birth practice replaced by Caesarean', *New Zealand Doctor*, 15 April 1998, p.41.
45. Addison, 1897, pp. 695, 576.
46. D.W. Taylor, *The Monro Collection in the Medical School Library of the University of Otago*, Dunedin, 1979. The volume is sub-titled 'A descriptive catalogue with annotations and introduction'. Like Taylor, Malcolm hailed from the north of Scotland.
47. I. Carr & D.W. Taylor, 'Physiology in the Otago Medical School. The John Malcolm letters', in L. Bryder & D.A. Dow (eds.), *New Countries and Old Medicine: Proceedings of an International Conference on the History of Medicine and Health*, Auckland, 1995, pp.229-35.
48. See Roberson to Mason, 14 July & 23 October 1902, Letters & Invoices (L&I) M-Z 1901-4, & 25 August 1903, Letters A-W 1903-4, Mason Papers (MP).
49. *NZMJ* editorial, 1901, vol.2, p.107 and Robertson to Mason, 18 February 1902, L&I A-W 1901-2, MP.
50. Macewen to Mason 19 December 1906, Letters G-O 1905-7, MP.
51. Macewen to Daisy Macewen, 11 October 1923, Glasgow University Archives, DC79/16; William Macewen jnr to Mary Macewen, 16 October 1923, DC79/25.
52. See obituary notices in *GMJ*, 1924, vol.101, p.217 & *BMJ*, 1924, vol.1, pp.603, 644 (Macewen) *NZMJ*, 1924, vol.23 p.365 (Mason), *NZMJ*, 1925, vol.24, p.231.

THE ONE HUNDRED AND FIFTY SIXTH ORDINARY MEETING

The One Hundred and Fifty Sixth Ordinary Meeting of the Society was held at the Royal College of Physicians and Surgeons in Glasgow on 256 March 2000. Dr David Wright, Vice President, took the chair, as the President, Dr John Forrester was presenting a paper. Three papers were read, the first by Mr James Beaton on William Mackenzie 1791-1868: A Glasgow Ophthalmologist and his Library. This paper was followed by two papers on Thermometry, one by Dr Forrester on the development of Clinical thermometry and one by Dr John Burnett on the Scottish Contribution to Medical Thermometry.

THE DEVELOPMENT OF CLINICAL THERMOMETRY

Sanctorius (1561-1636) was inventor or co-inventor of the first thermometers of any kind. He was born near Trieste, was for a time physician to the King of Poland, and during his most active period a professor at Padua. He was notable for studies on his own body weight and the reasons for its short-term changes. His thermometers were incapable of calibration and susceptible to interference by barometric changes, but nevertheless he attempted an assessment of fever by measuring the *rate of change* of the fluid level when it began to move on insertion of the instrument into the patient's mouth, and concluded that in fever the rate was higher than in normal health, which might certainly be so in some cases.

George Martine (1702-43) lived all too short a life. He was a notable scientist at St Andrews, and composed thorough and well-referenced writings. He had access to accurate thermometers by this time, reading on the Fahrenheit scale, and thus was able to take body temperatures on himself, obtaining readings of 97–98°F on skin. He found substantially the same result between the thighs, in the mouth, and in urine as voided. He investigated some animals, and noted that in birds the reading was higher than in man. He also held that men were hotter than women, but presented no figures to support this opinion. He documented a fever in himself, when he attained a temperature of 106° in an ague. Early in this ague, he felt very cold even when his temperature was raised by 2-3°, and this was at the time impossible to explain.

Anton de Haen (1704-76), a Viennese physician, carried out much clinical thermometry in a hospital setting. The instruments at the time and until much later were very slow to reach equilibrium, and once they had been removed from the patient the column began to sink at once. In de Haen's practice it took a quarter of an hour to get a reading. He devised a practical method of using 7½ minutes and adding 2° to the reading, with results which could hardly be very reliable. He also showed that temperatures in the hand were usually low, and took sequences of readings from individual patients during the course of intermittent fevers.

Francis Home (1719-1813) became in time Professor of Materia Medica at Edinburgh. In his youth he had served as a physician in the army in the Low Countries, and in 1748 was measuring body temperatures in the soldiers, even though they took 15 – 20 minutes each to do. He found that temperatures in remittent fevers (probably malaria) could reach 104°F. He was very clear that the physician risked contagion through taking temperatures so near the patient's mouth. A remarkable achievement of his at the time was to find the spleen protruding after a gunshot wound, and believing it beyond saving, he did a splenectomy with recovery.

Joseph Black (1728-1799) was for the major part of his career Professor of Chemistry at Edinburgh, and a teacher so distinguished that it was written of him that: "Dr Black became a favourite lecturer; and many were induced, by the report of his students, to attend his courses, without having any particular relish for chemical knowledge, but merely in order to be pleased." He normally opened his course of chemistry with heat theory. He was the identifier and measurer of latent heat, a discovery which led in the hands of his friend James Watt to major advances in the application of steam power, and his must have been the mind that stimulated subsequent temperature-takers trained in Edinburgh.

James Currie (1756-1805) was born in SW Scotland, and sent to America at the age of 15 to make his fortune; in the event, since his parents died relatively young, he became the main support of 7 sisters, 4 of whom died of tuberculosis. He had to make his way back across the Atlantic as the War of Independence loomed, and took up medicine, training in Edinburgh. Heat was an interest even of his student days.

In time he became a physician at Liverpool. In 1790 a shipwreck in the Mersey led to curious survivals and casualties, those perched up out of the water perishing and those in it surviving. He then modelled the exposures with young men as subjects in a tank, using thermometry. He showed clearly that exposure wet in the air lowered the body temperature more rapidly than exposure in water at the same temperature, but did not reach a clear grasp of the explanation. An excellent practical thermometrist, he thought of using cold water treatment for fever because of his acquaintance with the experience of **William Wright** (1735-1819), who wrote of himself that: "September 9th 1777, having given the necessary directions, about three o'clock in the afternoon I stripped off all my cloaths, and threw a sea cloak loosely about me till I got upon deck, when the cloak also was laid aside: three buckets full of cold salt water were then thrown at once on me; the shock was great, but I felt immediate relief. The head-ach [sic] and other pains instantly abated, and a fine glow and diaphoresis succeeded. Towards evening, however, the febrile symptoms threatened a return, and I had recourse again to the same method, as before, with the same good effect. I now took food with an appetite, and, for the first time, had a sound night's rest."

Currie, in his advocacy of cold water for fever, used a sound mix of common sense and thermometry, certainly not seeking obsessively to push any raised temperature down to normal. The treatment achieved for a time a huge popularity. Children were perhaps especially enthusiastically treated, by their own fathers in various documented instances. Central Europe and America too got the message, but in the course of decades the vogue came to a natural conclusion.

Currie himself came to a lamentable end. He insisted on having himself frequently and copiously bled for fever in his latter years, being persuaded that he had proved it provided immediate benefit, by observing the drop in his own temperature *as recorded in his right hand* during blood-letting from the left hand. Although at autopsy the only findings recorded were "enlarged heart with incipient ossification of blood vessels, great wasting and adhesion of R lung.", important anaemia may be suspected by hindsight.

After Currie, a great wave of competent thermometry might have been predicted, sweeping over all clinical practice in relation especially to fever. This did not take place for many years. In the briefest terms: fever had always traditionally been assessed mostly by pulse rate and by feel of skin. Inevitably gross discrepancies between this evidence and thermometer reading were of everyday occurrence. In addition, the whole nature of heat and its origin remained for long obscure and controversial; for instance, there were those who thought of "boiling rage as the product of scorching heat in the brain". Thus in Currie's time it was commonly felt that "physiology, so far as it is known, is totally or nearly useless in explaining anything which happens in fever".

August Wunderlich (1815-77) was a professor at Leipzig for much of his career. He was a leader in a huge accumulation of clinical thermometry results, and had notable followers in Britain. He honoured Currie as a distinguished precursor, and he gave credit to **Sir William Aitken** (1825-92; MD Edinburgh 1848) for inventing the self-registering ("shake-down") thermometer, a notable practical advance. Aitken was Professor of Pathology at the Army medical school at Chatham, and author of a distinguished textbook, which reached numerous editions. In one of these there is no mention of the self-registering thermometer, and in the next he keenly and promptly endorsed it. But he did not actually invent it, and the process of its devising and introduction are described by Dr John Burnett in his paper.

[The main components of Dr John Forrester's address have been published (*The Origins and Fate of James Currie's Cold Water Treatment for Fever*, Medical History 2000, 44, 57-74.).]

THE SCOTS CONTRIBUTION TO MEDICAL THERMOMETRY

Daniel Gabriel Fahrenheit (1686-1736), born in Danzig, was the first to make thermometers that gave the same readings at the same temperatures. He did this by calibrating each instrument at two fixed points, the freezing point of water and his own blood temperature. By 1712 he had settled in Amsterdam and came into contact with Hermann Boerhaave who held various medical chairs at Leiden from 1719 to 1738.

Among the many foreign students whom Boerhaave attracted was George Martine (1702-41) of St Andrews. Martine wished to investigate variations in body temperature and so needed a supply of thermometers: he encouraged a young St Andrews man, Alexander Wilson (1714-84), to find out how they were made. Wilson was intelligent and dextrous, and soon succeeded in copying Fahrenheit's work: reliable thermometers were thus available in Scotland before they were made in London. Wilson moved to Glasgow where he became a typefounder, making the type that the famous Foulis Press used. He was a versatile man: in 1760 he was appointed Professor of Astronomy at the University of Glasgow. Joseph Black used Wilson's thermometers while he was evolving the concepts of specific and latent heat, and William Herschel was using a Wilson instrument when he accidentally discovered infra-red light.

Two of Wilson's thermometers survive, in the National Museums of Scotland and the Science Museum, London. They are small and very delicate, protected by sealed glass sheaths. Martine said they took 20 minutes to register a steady temperature. When in the 1760s John Hunter (1723-93) started his researches on physiology, he devised a thermometer with a thick, robust stem and a small bulb that contained far less mercury than Wilson's instruments. He had them made by the leader of the London instrument trade, Jesse Ramsden.

James Currie (1756-1805) carried out his researches on fever with a modified Hunter instrument. He had it made with a 60° bend in the stem, so that it could be placed in the patient's axilla and read by a physician standing behind the patient. Currie instruments were quite widely used, and although they rarely appear in the literature, they are well represented in museums. A thermometer, almost certainly of Currie's design, was used by the Scots surgeon Archibald Arnott on the dying Napoleon.

Systematic studies of the use of the thermometer to diagnose disease were made in France and Germany, most notably - and in awesome detail - by Karl August Wunderlich (1815-77) in Berlin in the late 1840s. His work impressed the Dundee-born and Edinburgh-trained William Aitken, physician at the Royal Naval Hospital at Netley, near Southampton. Aitken devised a pair of thermometers which were sold together by Luigi Pasquale Casella, a London glassblower and instrument maker of Venetian descent.

One was like a Currie instrument, except rather larger and so easier to read. The other was straight, but for the first time recorded a maximum reading. It did this by having the end of the mercury column detached from the rest of it, a design which had been produced some 20 years earlier by John Phillips of Oxford, but apparently never used. Aitken thermometers were first made in 1853 and found a limited use for some decades.

However, about 1867 Clifford Allbutt (1836-1925) devised the short clinical thermometer with a constriction above the bulb, the form which soon became standard.

THE NINTH HALDANE TAIT LECTURE

The Ninth Haldane Tait Lecture was held on Wednesday 24th May 2000 at the Pollock Halls, University of Edinburgh. Professor Ynez O'Neill of Los Angeles gave an address on The Death of King Henri II of France.

THE DEATH OF KING HENRI II OF FRANCE

France's most Christian King, Henri II was dead. The injury that killed him was sustained not on the field of battle, but in a display of mock warfare, a joust or tilt as it was sometimes called. Though warfare in the sixteenth century was fought with cannon and muskets, the ideal of the knight as a defender of honour, jousting before a gallery of ladies and his peers still held great appeal. Henri was proud of his prowess in tournaments, a skill that was to result in his death.

The English ambassador, Nicholas Throckmorton, who was in the gallery that day witnessed the accident and wrote about it initially in rather light terms. Reporting to his superior, Robert Cecil, he stated *"As far as I could discerne, the hurt seemed not too great: whereby I judge, he is but in little danger"*

Still despite all the efforts of the most prestigious physicians and surgeons in Europe the king died. His death changed the course of European history. France was plunged into crisis. A strong and competent king was suddenly removed from the scene. His Italian wife, Catherine de Medici was left as regent with clutch of sickly sons in their minority.

Rather than attempting to unite an increasingly restive and sectarianised state under a strong and permissive crown, Catherine backed the Catholic factions, thus directly alienating much of the court and a significant range and strata of French society, as well as destroying the crown's credibility as a mediating force.

The result was near anarchy. In the broader context, Philip II of Spain had little interest in seeing a resolution of the problems besetting the French monarchy, particularly while facing a revolt of his own in the Low Countries. Elizabeth of England by and large, took a similar stance.

Ill at ease with a monarchy that stood in alliance with Scotland and yet unwilling openly to support any party, she attempted mostly successfully to stay on the sidelines. The issue was not resolved until almost thirty-five years later with the accession of Henri of Navarre in 1593. Henri the Second's death therefore, held important consequences for the European realpolitik.

Why were the physical consequences of the king's injury universally misdiagnosed? Why did contemporary analysis of the fatal pathology focus so completely on apparent damage to the meninges at the back of the cerebrum and nearly ignore the danger from penetration by splinters?

Before concentrating on these topics, let me demonstrate the accident's impact on Scotland. The death of Henri II elevated Mary, Queen of Scots and Dauphine of France, to the French throne. Mary was born on the 7th or 8th of December 1542 at the Palace of Linlithgow, West Lothian. Her father, James V of Scotland died less than a week later and hence on the 9th of September 1543, at the age of nine months, Mary was crowned Queen of Scotland in the chapel at Stirling Castle. Mary of Guise, the child-queen's mother was left behind when in 1548, the less than six years old Mary, Queen of Scots was sent to France. There she was to remain for the next twelve years, a favourite of the French court and of the French monarch. As Henri II who was to become her father in law described her, "the most perfect child that I have ever seen"

On April 24th 1558, Mary married Francis, Henri's eldest son. This union, which though it carried sinister political overtones, seems to have been truly a love match. The young dauphin, Francis, loved Mary and she certainly seems to have believed that she loved him. In any event, Mary thoroughly enjoyed the elevated rank of queen-dauphiness and she fulfilled the role for which she had been trained since childhood to perfection.

A little more than a year later, she played a major role in the regal panoply that attended two other French royal weddings. To celebrate and seal the peace of Cateau Cambresis, the French king's sister Marguerite was to be wed to the Duke of Savoy, and his daughter Elizabeth was to marry Phillip II of Spain, whose English wife Mary Tudor had recently died.

Against a background of seemingly endless pageantry and festivities, Henri's love, if not virtual mania for, jousting showed forth. On the third day of tournaments, after having broken lances with various opponents, the king on a sudden whim challenged Gabriel de Lorge, Count of Montgomery, Colonel of his Scottish archers, to break a final lance with him. De Lorge refused at first, until Henri ordered him to obey.

According to Throckmorton's account of the events that followed,

"The King, after he had roone a good many courses very well and fair, meeting with young Monsieur de Lorge, capitaine of the Scottishe Garde, received at the said de Lorge his hands such a counterbuff, as the blow first lighting upon the King's head, and taking away the pannage which was fastened to his hedpece with yron, he dyd break his staff withal; and sowith the rest of his staff hitting the King upon the face gave him such a counterbuff, as he drove a splinte right over his eye on the right side"

The fatal injury was thus delivered by a Scot, in full view of his Queen, the young bride Mary. The history of the close relationship of the Scots archers with the French crown can be traced to the twelfth century, as is that of the Swiss Guards to the Vatican. Though pardoned by Henri of any wrong doing, de Lorge's life from that time forward was to be one of exile and tragedy, which has been chronicled elsewhere.

To deal with Henri's injury, Chapelain, the Protomedicus or official court physician, rapidly gathered his colleagues together, including Ambroise Pare, the distinguished military surgeon. Pare, who was very likely the most experienced of all in attendance at treating traumatic injuries, was later to write an account of the case.

Thus we learn that first aid, consisting of gross splinter removal and bandaging, was administered at the scene of the jousting. The king was then removed to the Palace where he was put to bed, fed and then vomited. He remained weak and lethargic, but didn't complain of any pain. On the second day, the heads of three executed criminals were dissected in an attempt to reproduce the wounds of the king for the surgeons to examine. Trepanation of the king's skull was considered but rejected.

When Philip of Spain learned of his future father-in-law's injury, he dispatched Andreas Vesalius, one of his court physicians, and the most famous medical researcher in Europe to assist at the king's bedside. Vesalius arrived the fourth day after the accident, and after examining the patient, pronounced that the king had suffered a *"Vulnus Chironicum"* which in Graeco-Roman terminology meant a wound that cannot be cured. This prognosis proved sadly accurate.

The fever that had begun shortly before Vesalius arrival signalled the beginning of a steady decline in the king's condition. The court ceased issuing royal pronouncements. The head of another executed criminal was procured for Vesalius to dissect, but as far as we know, no useful insights were obtained.

By the ninth day, the patient's condition had greatly deteriorated. In addition to the constant fever, the king's neck became rigid, severe pain recurred, seizures stiffened the right side of his body and his face suffered severe swelling and an exudance of pus. The king fluctuated in and out of consciousness. Trepanation was considered again but was again held to be contra-indicated.

According to Vesalius, a group of nobles insisted that the king be administered a potion of wine and sage. This measure, apparently a wound remedy, was opposed by Vesalius as he believed that it would heat the body, increase the rate of respiration and thus hasten death.

On the tenth of July 1559, eleven days after the accident, and at the age of 40 the king died. A brief and somewhat hurried post-mortem examination was conducted, of which we have two records, one purportedly written by Vesalius and the other by Pare. Vesalius tells us that no splinters were found in the brain, the lateral wall of the right eye socket was *"peppered with many splinters"* and an area of the dura and pia mater at the back of the skull was found to be purulent, surrounded by yellow and brownish tissue. Vesalius believed this finding confirmed his determination that the king had died of a contrecoup injury.

Although Pare reports the case in a rather different context, listing first a number of cases in which patients survived deep penetration of the brain by a variety of pointed objects, he contends that the splinters themselves had little effect. The king's death, he believed was caused by damage to the brain tissues opposite to the point of contact above the right eye. In short like Vesalius, Pare contended that the cause of the king's death was a contrecoup injury resulting in the putrefaction of the affected tissues at the back of the skull.

The concept of a contrecoup injury had been known since ancient times. It explained injuries resulting not from the impact of an external object on the head, but from the velocity of the blow causing the brain to collide with the opposite wall of the skull. The fact that on post mortem examination, membranes at the back of the skull were found to have sustained injury, led the skilled practitioners, who attended the king, to conclude that their patient died because the blow to the front of his head resulted in damage to the cerebral membranes on the opposite side of his skull.

With the acuity that hindsight and modern medical progress affords, we can emend their opinion. Our study of the case has led us to the opinion that Henri II died as the result of a virulent infection. The infective agents were the wooden splinters that penetrated the orbit of his eye. The question however is not why the king died, but rather why his injury was misdiagnosed by the most experienced medical scientists in Europe at that time.

The first answer to that question has already been suggested. In the minds of those men, the idea that death resulted from a contrecoup injury was not contradicted by the post-mortem findings. Even Vesalius who had published the second edition of his great De Humani Corporis Fabrica in 1555, only four years before the accident, had little detailed knowledge of the cranial orbital area. Moreover, in the sixteenth century, brain sectioning during an autopsy was unknown, and the underside of the brain was not examined by anatomists until Varolio in 1573. Most important of all, however, was the belief held by medical practitioners as late as the seventeenth century, that the cerebrum was of little value, and that ideation as well as all neurological function was controlled not by the brain itself, but by its ambient cerebral membranes.

This concept has a long history of which I shall trace a few highlights. About the middle of the twelfth century, a Norman scholar, William of Conches, asserted that all of the nerves in the human body derive from the meninges, and hence these membranes were called maters or mothers. Space does not permit us to explore why Constantine the African, who translated so many of the texts upon which William depended, termed the cerebral meninges maters, but suffice it to say that William believed that the sensory nerves originating in the pia mater stretched out in an anterior direction towards the windows of sense, while the nerves of voluntary motion, which he believed to derive from the dura mater, extended in a posterior direction. This concept was soon to be elaborated in the accounts of the brain found in certain Latin anatomical texts attributed to investigators in Salerno, a centre for medical training in the southern part of Italy.

Three versions of these texts survive, and they may comprise the lectures of one or several Salernian masters named Richard or Nicolaus. In the version attributed to master Nicolaus, the author, after discussing the brain's composition in classical terms, makes an extra-ordinary statement about the form and function of the meninges. Though modern anatomists count three of these

membranes, medieval thinkers such as William of Conches believed that there were only two, counting the pia mater and the arachnoid as one, and the dura mater as the other. To return to Nicolaus' statement:

"Three cells are set off. Indeed two membranes, that is to say the pia mater and the dura mater, are connected to each other in two places. In the front part of the head, they constitute the cell of fantasy. In the middle of the head, they constitute the cell of logic. In the posterior of the head, they constitute the cell of memory."

What makes this statement extraordinary is not Nicolaus' assignment of the mental faculties but his localisation of the three chambers. In his schema, the three cells which medieval anatomists and philosophers believed to be the site of fantasy, cognition and memory are found not beneath the cerebral cortex in what we know as the ventricular system, but above it, in the brain's ambient membranes. While discussing neural function, moreover, Nicolaus amplifies his theory of meningeal localisation by explaining that the sensory nerves arise from the cell of fantasy, and the motor nerves from the chamber of memory, which he describes as the thesaurus memoriae.

But Nicolaus was not alone in his advocacy of this strange doctrine. Another twelfth century anatomical text attributed to a Richard the Salernitan tells us

"the two meninges make three folds and within them there are a certain three cells phantasy, logistic and memory"

Still more ornate is the account also believed to have been written by a Richard, who may have been English:

"Therefore there are two mothers, which are called meninges or vulgarly webs, which knotted, make three foldings by which together, in a wonderful fashion of nature, cells are distinguished, namely the anterior, posterior and middle."

Richard assures us, moreover, that he is describing human not animal cerebral anatomy.

"And note that this is the anatomy of the human brain and of its cells, not of brutes, because they are not believed to possess all the cells, because they do not have the middle one..."

Unfortunately we do not have time to trace that development today, but what is important to emphasise is that as early as the twelfth century medieval thinkers believed mental activity to be localised in the ambient membranes of the human brain. Moreover this doctrine explains a puzzling medieval anatomical diagram of the brain and the eyes, which is found in the earliest illustrated anatomical manual known in the Latin West.

Recent discoveries concerning this diagram and its analogues, enable us to establish that the diagram depicts a sectioning of the brain with the membranes retracted. The cells of ideation having been bisected, the two connections between the meninges, which Nicolaus mentioned, appear as green bands.

It may be recalled that William of Conches tells us that the nerves of sensation originate in the pia mater. In the diagram the sensory nerves are depicted in red and we can conclude that the figure represents that membrane. A statement in the text accompanying another figure of the illustrated manual confirms that a network analogous to the meninges exercises sovereignty over the whole brain.

In the surgical writings of the thirteenth through the seventeenth century, we find the clinical implications of this doctrine. One group of thirteenth and fourteenth century surgeons, who advanced several innovative concepts, called themselves the Moderni. One traditional idea, to which they adhered firmly, however, was that meningeal injury resulted in great damage to the mental faculties.

Theodoric of Cervia, for example, warns against intervening in cases of meningeal lesion, as did a later disciple of the school, Lanfranc of Milan.

In his discussion of head wounds, Ambroise Pare tells us

"Within a short while after inflammation seizes upon the membranes and the brain itself which is caused by corrupt blood proceeding from the vessels broken by the violence of the blow and so spread over the substance of the brain. Such inflammation communicated to the heart and the whole body by the continuation of the parts causes a fever... A great part of these accidents appeared in King Henry of happy memory a little before he died"

Pare's later account of the case of Henri II is less clear on this point. Pare noted that the brain itself was injured as well as its principal ambient membranes. He does emphasize, however, that the injury to the eye was not a factor in the king's death.

"His skull being opened after his death, there was a great deal of blood found between the dura and pia mater, poured forth in the part opposite to the blow, at the middle of the suture of the hind part of the head; and there appeared signs that the native colour turned yellow, that the substance of the brain was corrupted, as much as one would cover with ones thumb. Which things caused the death of the most Christian king, and not only the wounding of the eye as many falsely thought. For we have seen many others who have not dyed of farre more grievous wounds in the eye."

Nevertheless the meningeal doctrine persisted well into the seventeenth century, as is illustrated in the writings of an eminent English surgeon, Richard Wiseman, who returned to England on the restoration of Charles II, He emphasizes that in treating head wounds, the surgeon's principal concern should be to heal the meninges. After reviewing several cases, Wiseman concludes

"I could tell you of many more wounded into the brain, but I think these may serve to prove what I would demonstrate viz that the Brain is of itself insensible; that those symptoms which accompany these wounds proceed from the pain which the Meninges, Dura and Pia Mater suffer, which if oppressed.... Do quickly suffer the greatest Symptomes as Vomiting, stupor, paralysis and much more if they be pricked by any speel of a Bone, or other extraneous sharp body."

Although by the middle of the sixteenth century, the idea that the brain was controlled by the meninges was beginning to change, the doctrine had enjoyed a long and venerable history. Like other medieval scientific theories, it "saved" or "preserved" the appearances. Medieval surgeons, faced on the battlefield or at tournaments with the difficulties of a variety of head injuries, believed that the meningeal vessels carried the vital spirit necessary for the conduction in the brain of the animal spirit, which was the agent of all nervous action. Interruption of these passageways led inevitably to the symptoms Wiseman enumerated. By focussing attention on the ambient cerebral membranes, rather than on the cortex, early investigators may have delayed research into the brain's form as well as its functions, but they were struggling with problems many of whose solutions continue to evade us even today.

THE ONE HUNDRED AND FIFTY SEVENTH ORDINARY MEETING

The One Hundred and Fifty Seventh Ordinary meeting of the Society was held at the Tor-na-Coille Hotel, Banchory, Aberdeenshire on Saturday 3 June 2000. After an excellent lunch, there were two papers. Mr Alexander Adam talked on Dr Francis Adams of Banchory and Dr John Simpson talked on the Plague of the Philistines.

DR FRANCIS ADAMS OF BANCHORY

It is appropriate that, here in Banchory, we should honour Dr Francis Adams, the first doctor in Banchory and classical scholar extraordinary.

A contemporary wrote of him "It is something to be proud and ashamed of that the most learned physician in Britain and probably in Europe is at this moment a country surgeon in a small village in Deeside." He was named "Doctissimus medicorum Britannorum, the most learned of British doctors", by his friend Dr John Brown, author of "Rab and his friends" and William Geddes, Professor of Greek at Aberdeen University noted "Of all the physicians Scotland has produced, the most familiar with the treasures of literature and in the riches of material science."

Since these eulogies the world has moved on and now Francis Adams seems largely forgotten. In an address to his Canadian students, Sir William Osler instanced Francis Adams as an example which they should follow, but today very few, if any medical students have heard of him. In two large works on medical history published in the last decade, in which it seems to me accuracy is sometimes sacrificed in the interests of productivity, Adams is not mentioned.

Francis Adams was born on 13 March 1796 and the family bible notes that he was baptised at Lumphanan a week later. He was the son of James Adams and Elspet Black and was the fourth child of the marriage. His father was variously described as a gardener, farm labourer and small-holder. In fact, at the time of Francis's birth, he was a tenant of the small farm of Easter Mains of Auchinhove, 11 miles north west of Banchory.

Francis was educated at the local school and with a bursary, entered King's College, one of Aberdeen's two Universities, graduating MA four years later. At that time the Arts course contained the whole corpus of knowledge and this included the theoretical medical teaching of the day. Evidently he had decided on a medical career, and for the next two years he pursued this aim in Edinburgh and London, graduating MRCS London in 1815.

His movements thereafter are unknown but in 1819 he put up his plate in the village of Banchory, the first doctor in the area. Soon after he married Elspeth Shaw, the daughter of a local land-owner, and lived in the old manse at Bellfield. Demolished and rebuilt by his son in law, Dr McHardy in 1893, it is now an old people's home. The manse had previously been the home of George Campbell, Professor of Divinity at Marischal, a classical scholar and luminary of the Scottish Enlightenment.

Banchory at that time, more than thirty years before the railway came, was a tiny village, but the surrounding countryside, now largely deserted, was densely populated, the inhabitants supplementing their basic existence by the illicit distilling of whisky. The birth rate was of course high. Adams' practice covered a large area extending, it is said, over the range of hills to the south into Angus. Travel must often have been very difficult as winters were severe. When in Adams' time, the first doctor in Cove, a coastal village near Aberdeen, was funded, he was to be supplied with a horse drawn sledge, the horse to be "sharp shod".

For the rest of his life, Adams served the area with diligence and skill, observing and recording his patients' afflictions. He wrote many papers for medical journals on such subjects as burns, adder bites, club foot and a fatal case of aortic aneurysm with autopsy - he had made the diagnosis in life - a remarkable achievement then. His account of a dislocation of the knee provides an example of his surgical skill. A 55 year old man trodden on by a bullock was found to have an anterior dislocation of the knee. This Adams reduced but the leg became gangrenous and nine days later he amputated through mid thigh. The patient survived and in three weeks the wound had healed (in hospital he would probably have died of hospital gangrene). Adams dissected the knee and gave a detailed account of the bones, ligaments and muscles around the knee.

His chief interest however was obstetrics in which, in those days of large families, he had extensive experience. When president of the Med Chi in 1844 he presented a paper on uterine haemorrhage, where his mistaken view that bleeding was due to laceration of the parts and not to uterine atony, was hotly disputed by Robert Dyce, later the first Professor of Obstetrics in Aberdeen. Adams continued to study the placenta and published a number of papers on the subject. He dissected specimens and went to Glasgow to study those in the Hunterian museum. His final conclusions were published in a booklet in 1858 "On the Construction of the Human Placenta".

As he travelled about the countryside, he observed and recorded the plants and animals of the area. In Dickie's "Botanist's Guide", Dr Adams' name appears frequently among the recorders of the species. In 1859, with his son Dr later Professor Leith Adams, he published a book on the birds of Deeside comparing them with Kashmir (Leith Adams spent 7 years in India and later was Professor of Natural History in Dublin and Cork).

Adams also wrote poetry, although it is said with no great success. His poetic translations from English to Greek were considered to better those from Greek to English. Among his publications is a book of poems entitled "Arundines Devae" literally "Reeds of the Dee."

So Francis Adams was a country doctor, surgeon, botanist, ornithologist and poet, A Victorian polymath not unusual in his day. His lasting fame was based on none of these but on his eminence as a classical scholar and his translation of the classical texts into English. Before his time the corpus of knowledge was available only in Greek, Latin, Arabic and, it may surprise some, Gaelic.

When a student in Aberdeen aged 15, he met an older student from Montrose, who had had an excellent classical education. Adams determined to emulate him. He later wrote "In early life I had been shamefully mistaught. I began by devoting seventeen hours a day to Virgil and Horace, and read them 6 or 7 times in succession. Having mastered the difficulties of Latin, I naturally turned my attention to Greek. It was Dr Kerr of Aberdeen who drew my attention to the Greek literature of Medicine and at his death (in 1826) I purchased a collection of the Greek Medical authors that he had made. However, I have read almost every Greek work that has come down to us from antiquity with the exception of the ecclesiastical writers, all the poets, historians, philosophers, orators, writers on science, novelists and so forth. My ambition was always to combine extensive knowledge of my profession with extensive erudition."

In 1825 Adams entered a competition at Marischal College on the subject of the "Differences between Latin and Greek Syntax". He did not win but he published his essay and this brought him into contact with Greek scholars at home and abroad. His first publication on medical history in 1829 was entitled "On the nervous system of Galen and other ancient authors" in which the teaching of Charles Bell was investigated with much learning.

He next turned his attention to his greatest achievement - the translation of the classical medical works into English. At that time, Adams wrote, there was no work in English, nor he believed in the continental languages, which gave an account of the medical knowledge of the Greeks, Romans and Arabs. To remedy this, he first intended to produce an original work, but found that this would be impossible. Instead he chose the "Seven Books of Paul of Aegina" (Paulus Aeginata) which Paul himself claimed without undue modesty, would contain the descriptions, causes and cures of all diseases.

Paul of Aegina (c625-690 AD) was one of the last of the Greek scholars. His seven books were based on a much larger collection, the 770 books of Oribasius (325-403 AD), some of which survive today, which Paul considered too unwieldy to carry about. To Oribasius' account, he wrote, "I have added little of my own"

"I began the translation of Aeginata" Adams later wrote "in the end of November 1827 and finished it in April 1829. I never in all my life underwent such literary drudgery. During these months, I sat up late and rose early and stole every minute that I could from the duties of my profession. My practice though not lucrative, was extensive especially in the obstetric line. I arranged however to work at my translation 10 hours a day."

The finished work was no simple translation. The task he set himself was to add to Paul's account, a commentary on all the authorities, Greek, Roman and Arabian, from Hippocrates onwards, after consultation with British and continental authorities, including Littre, whose 10 volume translation of the Hippocratic corpus is said to be still the only complete one. In addition, he gave the opinions of modern authorities on the diseases described.

Charles Singer, Professor of Medical History in London, wrote in 1942 "The work was worthy of the effort. The masterpiece "The Seven Books of Paulus Aeginata" maintains to this day, its position as far and away the finest venture in pure historical medical research in the English language. It is more than a translation, more than a commentary. The extensive and accurate notes that accompany it trace the practice backward into remote antiquity and forward into mediaeval times. This makes it a reference book both to Greek and to Arabian medicine. It is the best and most useful work of its kind in the English language and one of the best in any language."

The first volume of the book was published in 1834 to favourable notices, but owing to the failure of the publisher, the project came to a halt. Ten years later the recently formed Sydenham Society took up the matter, and the three volumes were published from 1844-1847. In this edition at the request of the society, reference to modern authors was omitted.

Adams' translation was not a simple academic exercise. When he undertook the project, Hippocratic medicine was still being taught in medical schools. In the second half of the nineteenth century, Sir Andrew Clark, son of an Aberdeenshire surgeon, applied the Hippocratic principles of detailed history-taking, frequent careful examinations, and a strictly controlled regimen of diet, fresh air and exercise and "Primum non nocere"- above all, do no harm. He established in London, a large practice and huge reputation. His death in 1893 was considered a national misfortune.

In recognition of his achievement, Glasgow University awarded Adams the degree of LLD. He next turned his attention to the work of Hippocrates. He was well aware that the Hippocratic collection contained material added later than Hippocrates' time and he chose the works he considered "genuine". Modern historians consider that the Hippocratic corpus, mostly written between 430 and 330 BC, was probably the library of a medical school or schools, collected later than Hippocrates' time and deposited in the famous library at Alexandria: and only a small part, if any, was written by Hippocrates himself.

Hippocrates -"the father of medicine" lived 460-375 BC. He was credited with having removed medicine from superstitious and religious beliefs into a science. Adams' translation of the "Genuine works of Hippocrates" though much less comprehensive than the 7 books of Paul of Aegina, is much better known. It contains the Aphorisms, of which number one must be the most quoted of

all medical sayings. "Ars longa-vita brevis" "Life is short and the art long, the occasion fleeting and judgement difficult. The physician must not only be prepared to do what is right, but also to make the patient, the attendants and externals co-operate."

It could be taken as an example of Adams' method. After giving the translation above, he added a whole page of alternative translations, quoting authorities from Galen onwards. The work also contains the Hippocratic oath, the foundation of medical ethics. A modified form was taken by medical graduates for many years. Aberdeen still asks its medical students to make a "declaration" on graduating.

"I finished the translation of Hippocrates" Adams wrote "in about four months. The certainty of obtaining a fair remuneration for the trouble it cost me made this by far the most delightful task I ever engaged in" The work appeared in 1849 in two volumes.

The genuine works of Hippocrates was followed in 1853 by "The works of Aretaeus the Cappadocian". Aretaeus lived in the second century AD. The book contains interesting descriptions of many diseases such as diabetes, tetanus and cholera, with the treatment in use at the time. It is the shortest of the translations and an easy read. After its publication, Aberdeen University belatedly awarded him an honorary MD. During the years that he was working on his translations, Adams made journeys to Oxford and London, where he numbered among his friends scholars of the day and such medical scientists as Quain and Sharpey, the founders, in Britain, of the modern teaching of Anatomy and Physiology. Also among his friends were two eminent physicians with local roots, Sir John Forbes, who introduced the stethoscope to Britain and Sir James Clark, Queen Victoria's physician. Aretaeus was the last of his classical translations, after its publication, he confined his activities to his practice, his family, his birds and plants and his poetry.

As a translator of the classical medical texts, Adams is unique – only he was able to combine extensive knowledge of classical literature with up-to-date experience, practical and theoretical, of medicine. Also his botanical knowledge was invaluable in interpreting the medicines in use by Hippocrates and his followers.

So what sort of a man was he? He was strongly built and hardy, necessary in one exposed to the hazards of travel in the hills and glens in all weathers. He was able to carry out his many activities only because he could do with little sleep. His practice was to return from visiting patients in the evening, have a meal and sleep for 2-3 hours. He then got up and worked through the night, returning to bed for another 2 hours or so. He was no retiring scholar. He loved social gatherings, discussion and argument, in which he occasionally came off second best. It was his competitive nature that drove him to emulate the student from Montrose.

He was dedicated to the care of his patients, which led him to refuse the Chair of Greek at Aberdeen. When his grateful patients made a collection on his behalf, he courteously refused it, saying that their gratitude was sufficient reward. A poor horseman, awkward in the saddle, he was so dependent on his horse that his friends said that his books should be inscribed "by Adams and Dobbin". Schoolboys would follow him chanting, "this is the way the doctor rides, this is the way the doctor rides". He is said to have been rather unworldly, easily taken in by practical jokes played on him by his sons and their friends.

He was a devoted family man. His wife died in 1845, leaving him with seven children. A daughter wrote of him "He was everything a loving parent could be. Although so busy in his profession and literary work, he always found time to superintend my brothers with their Latin and Greek, and encouraged all of us to have a taste for improving studies. He encouraged my brothers to have a taste for natural history and explained any wild plant he found on his rounds."

He died in 1861, of a chest infection, after visiting a patient on a wild night. After his death, his bust by Brodie was given to Aberdeen University, by his son Professor Andrew Leith Adams. After being rescued by Professor John Craig from a junk room at the old brewery of Kings College, it was shown at the first Aberdeen meeting of this Society in 1951. It was then restored to the University, but now sits in the Council room of the Aberdeen Medical Chirurgical Society of which he was President in 1844.

His diplomas were given to the University Library by his grand daughters in 1962. Also in the library is a small book of "Consolation of Philosophy" by Boethius, in Latin of course, which he always carried with him. In it he had written, in Latin, "This golden book I have read, re-read and read yet again for nearly 40 years and which I shall read often as long as I live" FA 1852.

Boethius (480-524) was the pre-eminent scholar and philosopher of his age. Theodoric, the Ostrogoth king of Rome appointed him his chief minister. He made enemies whom accused him of treason. "The Consolation of Philosophy" was written in prison awaiting execution. It had enormous popularity for more than 1000 years. Among its translators were Alfred the Great, Chaucer and Queen Elizabeth the first.

After Adams died, the people of Banchory erected a granite obelisk in his memory. It sits today near the site of his home in Bellfield. The inscription, written by his friend Sir William Geddes, Professor of Greek and later Principal of Aberdeen University is in Latin. In translation it reads "In memory of Francis Adams MD LLD of all physicians that Scotland has produced, most familiar with the treasures of literature and with the resources of science, long in this retired vale, far from court and academe, a true votary of Apollo, he devoted himself to medicine and the muses."

On the occasion of the bicentenary of his birth in 1966, a plaque with a translation of the inscription was unveiled by Charlie Adams, Francis's great grand-nephew; he is here with us today.

Many of Adams' publications are available in the University library, the City library and the Med-Chi library and I have consulted those. There are many obituaries of which that by his friend Dr John Brown of "Rab and his friends" is the most helpful. Professor Charles Singer visited Banchory and spoke with Dr McHardy, Adams' son in law and with surviving patients. His article "A Great Country Doctor" is most valuable. Finally I have to thank Dr Ian Porter who has given me many papers on Francis Adams and Charlie Adams and Mrs Phyllis Whitmarsh, great grand-nephew and niece of Francis for information about the family.

THE PLAGUE OF THE PHILISTINES

"Now Israel went out against the Philistines to battle, and pitched beside Eben-ezer; and the Philistines pitched in Aphek" (1 Samuel 4).

Aphek lay on the northern rim of the Philistine country. A mound of ruins, Tell el-Muchar, conceals all that is left of the place which lay on the upper reaches of the River Kenah, a river which flows into the sea north of Jaffa. (1) From a strategic point of view, Aphek was extremely favourably situated: eastward lay the road to the mountains of central Palestine where Israel had settled. On the edge of the mountain range lay Eben-ezer where the opposing forces met. At the first encounter, the Philistines gained the upper hand and the Israelites retired to regroup; they had also decided, in addition, to send to Shiloh for the sacred Ark of the Covenant with the Lord. The second encounter was a disaster for Israel, the army was completely routed and the Philistines carried off the Ark together with the other spoils of war (1 Samuel 4). The hill country was occupied, Israel disarmed, garrisons were located in tribal territories and the Ark was carried into Ashdod.

"Then the Lord laid a heavy hand upon the peoples of Ashdod; He threw them into distress and plagued them with tumours and their territory swarmed with rats. There was death and destruction all through the city" (1 Samuel 5)

Who were the people of Ashdod? The Philistines were the ultimate enemy on whom it was open season for smiting throughout the pages of the Old Testament: but who were they and where did they come from? Amos tells us:-

"Have I not brought Israel out of the Land of Egypt and the Philistines from Caphtor?" (Amos 9)

It is now thought that Caphtor refers to Crete, which suggests that the immigrant invaders were Mycenean in origin, a suggestion emphasized by the close resemblance to that of Homer's heroes. For example, Goliath's armour is the same as that worn by Achilles.

"From the Philistine's camp came a man named Goliath, from Gath. He had a bronze helmet on his head and he wore plate armour of bronze. On his legs were bronze greaves and one of his weapons was a dagger of bronze. The shaft of his spear was like a weaver's beam. His shield bearer marched ahead of him." (1 Samuel 17).

And from the Iliad, Book XIX (2) we read that Achilles, before his encounter with Hector:

"began by tying round his legs the splendid greaves with their silver ankle clips. Next he put the cuirass on his breast, and over his shoulder he hung the bronze sword with the silver studded hilt. Then he took up the great thick shield; the massive helmet he placed on his head. Finally, he took his father's spear from its case, the heavy long and formidable shaft that no Achaean could wield but he."

The Philistines entered history by way of carved reliefs and inscriptions on the walls of the Temple of Amun in Medinet Habu, west of Thebes and dating from the reign of Ramesses III (1195-1164 BC). The Sea Peoples, as the Egyptians called them, came from the coasts of Greece and advanced through Asia Minor, but in 1188 BC they suffered a severe defeat by both land and sea at the hands of the Egyptians. In spite of this, some thirteen years later, they were firmly settled on the coastal plain of southern Canaan, between the mountains and the sea (1). The Bible lists the five cities which they possessed; Askelon, Ashdod, Ekron, Gaza and Gath (1 Samuel 6). Each of the cities and the land adjoining was ruled by a lord who was independent, but for political and military purposes, the five rulers worked hand in hand. For all matters of importance the Philistines acted as a unit.

It was about 1050 BC that Israel was defeated by the Philistine army and the Ark captured and taken to Ashdod. There it was set up in the Temple of Dagon, the fish god, next to the statue of the god. The following day, the statue of Dagon was found face down before the Ark. This happened on the subsequent two days, but on the third morning, the head and arms of Dagon had been broken off. At this point, epidemic disease broke out; the people were affected by tumours and their fields swarmed with small rodents.

The rulers of Ashdod consulted with the Princes of all the Philistines as to what should be done and a decision was made to transfer the Ark to Gath. After its arrival, the plague broke.

"The Gethrites consulted together and made themselves seats of skins" (1 Samuel 6)

The Ark then went to Ekron where the inhabitants summoned the Princes and required them to find a way to return the Ark to Israel. The priests, when consulted, recommended a gift of gold to accompany the Ark and the gift was specified as five golden emerods and five golden mice; one of each to represent the five Philistine cities. The priests emphasised this need for reparations by reminding the Princes what had happened to Pharaoh and the Egyptians.

"Why should you be stubborn like Pharaoh and the Egyptians? Remember that this God made sport of them until they let Israel go" (1 Samuel 6)

And so after seven months in Philistine hands, the Ark returned to Israel. It was left on the farm of Joshua of Beth-shimesh where the men were at harvest. The epidemic ended: so, what was it? The continued use of the word "plague" makes it tempting to consider Yersinia pestis, especially when widespread death is associated with tumours. However, it is necessary to take account of the Jacobean translators of the Bible's preference for "emerod" as the translation of the Hebrew word "aphalim" which according to Lloyd Davies (3) means tumour or swelling in the groin. These authors consider bubonic and pneumonic plague to be the probable diagnosis since buboes are enlarged lymph glands, in the groin most frequently, followed by glands in the axilla and neck. Granuloma inguinale is a possible alternative but can be discounted in view of the apparently high death rate and the possible rodent vector.

Any wild rodent, mouse, rat, shrew or vole can form the reservoir of plague, but rats infected by wild rodents are the usual means of transferring the infection to man, since rats tend to live near man. The mouse is identified as the rodent in the Authorised Version of the Bible, but is changed to rat in the modern versions. Fleas, living on the rodent, become infected by biting animals already infected by Yersinia pestis. The bacillus multiplies in the proventriculus of the flea's stomach and blockage occurs. In 1914, Bacot and Martin (4) showed that the bite of a single flea could transmit plague when the proventricular valve was blocked and rendered incompetent, since blood, incompletely siphoned by the flea, is regurgitated into the wound made by the proboscis, together with fragments of bacillary growth.

Rattus rattus is the species of rat most likely to give rise to severe epidemics of human plague and was widely prevalent until early in the eighteenth century. It supports the flea most likely to transmit plague to man (Xenopsylla cheopsis). Rattus norvegicus is essentially an outdoor animal and its fleas, in temperate climates, belong to a species unlikely to carry plague to man. Mice carry few fleas of any kind.

Wood (5) examined early Hebrew texts and could not identify the species of rodent from the descriptive word used "akbar". He felt that the word derived from the Hebrew words used to signify the destruction of corn. He concluded that the animal in question was one which destroyed corn and was feared by agrarian communities since it attacks seeds, the developing crop and the stored

grain. He quotes Flavius Josephus, the Hebrew historian of the first century AD who tells of swarms of mice appearing at the time of sowing, remaining throughout the growing season and with the harvest disappearing.

Crawford (6) identified many ancient records of plagues of field mice; and refers to a report by Herodotus of a nocturnal invasion of mice in the camp of an Assyrian invasion force, during which the bow-strings of the archers were destroyed.

In II Kings 19 we read that Sennacherib with an Assyrian army entered Canaan and threatened Jerusalem. Isaiah prophesied for King Hesekiah saying:

"By the way that he came, by the same shall he return and shall not come into this city. And it came to pass that night the angel of the Lord went out and smote in the camp of the Assyrians an hundred four score and five thousand and when they arose early in the morning, behold they were all dead corpses.

So Sennacherib, King of Assyria, departed and went and returned to Nineveh"

Short (7), discussing this disaster, considers the cause to be either pneumonic plague or cholera. Hirst (8) believes the Philistine plague to be epidemic bubonic plague, and that the emerods are plague buboes. But cervical gland buboes from tonsillar septicaemic plague cannot be considered as arising in the "secret parts", for in 1 Samuel 5 we read that when the Ark was in Ekron,

"Even those who did not die were plagued with tumours in their secret parts"

Descriptions of the clinical symptoms of plague are many for the centuries that followed the great pandemic of 1348, but describing symptoms is one thing: diagnosing a pathological condition quite another.

Ingrassia (13/9) records that when he was a student in Padua in 1535, a pestilence broke out which the doctors were unable to identify. Benaglio (13/10) wrote that some signs of plague are said to be typical while others are common in other fevers as well as in plague. The signs typical of plague are buboes in the groin, under the arm and in the neck and true carbuncles. Signs in common with other fevers are high pyrexia, petechiae, parotitis, headache, delirium and vomiting. Ingrassia (13/9) and Parisi (13/11) claim that there is a differential morbidity and mortality by social class: with a higher morbidity but lower mortality in the poor "because of the misery and distress in which they always live", but that fatality was higher in the upper classes" because the nobility is more delicate and less strong." However such opinions were based on haphazard observation, guess and speculation and could only have been made as an epidemic ended, since Shrewsbury (12) shows that in European epidemics of bubonic plague from the fourteenth to the end of the seventeenth century the case fatality approached 90% in the initial weeks, dropping to 30% as the epidemic ended.

Plague epidemics could randomly start in Spring, Summer or Autumn and the curve of the epidemic conformed roughly to the normal or Gaussian curve, although it could be skewed to left or right. The advent of cold weather in winter caused a lowering of the rat flea density, because the fleas either went into hibernation or died, and with a low rat-flea density an epidemic dies out. In this way modern scientific knowledge confirms the fourteenth century view that warm weather favours the spread of plague and cold weather generally acts as a deterrent. (13)

The Plague of the Philistines lasted seven months and ended in the Autumn: the Ark was left at the farm of Joshua of Beth-shemesh when the men were at the harvest.

So was it Plague? The contagion certainly ran a rapid course and in fatal cases it seemed to be a fast killer. Tumours were a feature in the "secret parts" - presumably the groin, but what of the axilla and the neck, and what of the statement that "the Gethrites made themselves seats of skin." Could the contagion, therefore be something else, and if so, what?

Short (7) in the Bible and Modern Medicine, discusses the meaning of tumour or emerod and makes the point that "ophalim" (the Hebrew word) is allied to the name "Ophel" which is a mound in Jerusalem. The Authorised Version translators rendered ophalim as emerod; however in the New English Bible ophalim becomes tumour in 1 Samuel 5 but haemorrhoid in 1 Samuel 6. This latter is also the definition in Gould's Medical dictionary. Hirst (8) suggests that ophalim had an offensive sexual implication to the Hebrew mind and points out that later Old Testament scribes substituted t-chorim for ophalim and t-chorim may be translated as haemorrhoids. Driver (14) also in a discussion of the philology of ophalim claims that ancient tradition connects "ophel", the singular form, with

the buttocks and so the anal region becomes the "secret parts" The question of sodomy therefore arises. Sodomy was anathema to the Jewish faith, as we read in Leviticus 18: 2

"Thou shalt not lie with mankind, as with womankind: it is abomination."

It is interesting too to note that Harvey (15) - George Harvey, author of "Little Venus Unmasked", used the phrase secret parts when discussing venereal infection.

From a scientific point of view what we have to consider is a communicable disease striking a people, recently at war, and accompanied by "swellings" amongst the survivors. Driver (14) suggests that such a disease could only have been dysentery, which is frequently responsible (because of tenesmus) for the development of piles. A further persuasive argument in favour of piles is to be found in 1 Samuel 5 "the men who died not were smitten with emerods". As piles are a late sequel of dysentery, they will not occur in those who die of the active disease; whereas in bubonic plague only those who die have buboes.

The infectious diseases that have affected armies during historical times are typhus fever, typhoid fever and dysentery. The first two do not cause violent enteritis, and so piles, but severe dysentery does, Shrewsbury (16). The geographic distribution of bacillary dysentery is world-wide and, given two essential features for its occurrence and dissemination, the presence of carriers and primitive sanitary conditions, the appearance of the infection can be anticipated (17). Meteorological conditions play little part in its distribution except that they provide climatic conditions favourable to the breeding of flies, which are important agents in the transmission of dysentery. For this reason, dysentery is more common in tropical conditions than in temperate zones, but given inefficient sanitary conditions, the disease will appear. In war, the disease has always been an important menace to the health of troops: and indeed there has been no military operation of any magnitude in which dysentery has failed to make an appearance (17). In India and Pakistan and Egypt, the maximum incidence of the infection occurs at the beginning and end of the warm season. These are the two periods that provide the optimum climate for the breeding of flies (17). Mortality from dysentery depends on the type of infection, the resistance of the patient and the adequacy of treatment. In general, the more serious infections are caused by the Shigella bacillus and when this appears as an epidemic manifestation the mortality may be as high as 30% (17).

"Now Israel went out against the Philistines to battle and pitched beside Eben-ezer, and the Philistines pitched in Aphek" (1 Samuel 4)

This Biblical record does not tell us how long the combatants remained in camp before they joined in battle but we do know that in a subsequent encounter, the one in which David slew Goliath, Goliath, as the champion of the Philistines, made his challenge;

"I defy the armies of Israel this day: give me a man that we may fight together." (1 Samuel 17)

The two armies would take time to complete their initial concentration and disposition plans following the first clash of their probing patrols. The first major encounter left the Philistines as victors but the Israelites did not leave the field, they regrouped and sent to Shiloh for the Ark; and Shiloh was some considerable distance from Eben-ezer. This battle took place at the beginning of the hot season since we know that after a seven month epidemic, the Ark was returned to Israel at the farm of Joshua when the men were at the harvest and the epidemic began with the returning troops together with the Ark and the Israelite prisoners of war. It is tempting to think that a severe gastro-intestinal outbreak in the Israelite army was in part responsible for their complete defeat in the field. Clearly the Israelites began the campaign with confidence in their competence since they were the aggressors and equally clearly something went disastrously wrong: one difficulty in accepting Shigellosis as the cause of the Plague of the Philistines is that it would be normally endemic in Palestine and would not cause a great mortality in the 15-50 age group, since that age group would have a satisfactory degree of immunity, and yet this age group was equally affected with younger and older age groups.

Man is a poisonous animal and needs either science or elbow room to keep himself from poisoning himself or his fellows. In the earliest cities there was neither science or elbow room. Thick walls and gates kept out visible foes but bred invisible enemies. The first steps of the nomad in civilisation, the gathering of houses into towns, increased the intensity and variety of disease and cost innumerable lives. Widespread sanitation is of relatively recent origin . The marvels of a well-appointed bathroom are the peak of sixty centuries of science. New diseases brought by discoverers,

conquerors and colonists have again and again found virgin soil in primitive races and wiped out whole populations. The Plague of the Philistines has the appearance of such a new disease: so what could it be?

In the past year or so we have heard much about Escherichia coli 0157. In 1971 Sack (18) and his colleagues described a "new" dysenteric disease manifested by a severe cholera like illness and caused by an enterotoxigenic E coli.

During the 1968 cholera season in Calcutta, patients were admitted to the Infectious Diseases Hospital with severe diarrhoeal disease, but for whom no apparent enteric pathogens could be cultured and isolated. These patients had secretory abnormalities of the small bowel similar in kind to those seen in cholera, but the patients each had small bowel colonised by a single serotype of E coli. Enterotoxin from this coliform was shown to be similar to that produced by the Vibrio cholerae. Sack also suggested that toxin-producing strains of E coli may be causative agents in a significant number of acute cholera-like diarrhoeal syndromes which occur in adults in the tropics. Dupont (19) and his colleagues discussed the pathogenesis of this coliform in the production of diarrhoea and concluded that at least two mechanisms exist by which disease may be produced. In one the organism penetrates the intestinal epithelium and produces a syndrome similar to shigellosis; in the other the organism does not have invasive properties but produces an enterotoxin which can cause a diarrhoeal syndrome similar to clinical cholera.

The clinical picture produced by the invasive form of each coliform will vary with the host but in its more severe forms includes dysentery (blood and mucus), tenesmus, urgency, hyperpyrexia and hypotension with severe toxaemia.

Tulloch (20) and his colleagues described an outbreak involving 28 adults who developed a fulminant diarrhoea with severe constitutional upset and suggested that this condition is relatively common. They emphasize that the true incidence of such invasive E coli disease will never be known until both clinicians and laboratory personnel admit that the disease entity exists. In this context it is interesting to note that about two years ago there was a report in our papers of a severe cholera-like outbreak in the Calcutta area caused by an E coli. Gorbach (21), in 1974, writes that one of the animal carriers of this toxigenic strain of E coli is the mouse and this was the animal that the Jacobean translators of the Authorised Version of the Bible decided was one aspect of the Plague of the Philistines.

Since the 1970s, laboratories have identified, by serological techniques, many strains of E coli which are primary intestinal pathogens. These have now been classified in four groups:-

1. Enterotoxigenic E coli, which cause an acute watery diarrhoea, sometimes of cholera-like severity.

2. Entero-invasive E coli, which like Shigella can penetrate the epithelial cells of the large intestine and multiply intra-cellularly, giving rise to blood and mucus in the stool.

3. Vero-cytotoxin producing E coli (entero-haemorrhagic strains), which are associated with a mild to bloody diarrhoea which is clinically important because it may precede a severe haemorrhagic colitis and is also a cause of acute renal failure. These strains produce one or both toxins and one of the toxins is similar physically, biologically and antigenically to the shiga toxin of Shigella dysenteriae.

4. Enteropathic E coli strains, which are now of relatively minor importance in countries which have good hygiene, but can cause outbreaks in countries with poor hygiene.

"But the hand of the Lord was heavy upon these of Ashdod, and He destroyed them and smote them with emerods, even Ashdod and the coasts thereof. And in the villages and fields in the midst of that country there came forth a multitude of mice; and there was the confusion of a great mortality in the city."

This record in 1 Samuel is consistent with a toxigenic strain of E coli making a cross species transfer from mouse into the biological orbit of man, firstly into the Israelite army and subsequently to the Philistines. Once into the cities, with their insanitary conditions, the disease would spread widely and quickly.

"What shall be the trespass offering we shall return to Him? Five golden emerods and five golden mice...for the one plague was on you all"

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The Scottish Society of the History of Medicine

CONSTITUTION AS REVISED AT AGM OF 1999

1. The Society shall be called "THE SCOTTISH SOCETY OF THE HISTORY OF MEDICINE," and shall consist of those who desire to promote the study of the History of Medicine.

2. A General Meeting of Members shall be held once a year on the last day of October or within four weeks of that date, to receive reports and to elect Members of Council and (when required) Office Bearers. The quorum shall be 20 members and decisions shall be taken by a majority. The President shall have a casting vote, and there shall be no proxy voting.

3. The management of the affairs of the Society shall be vested in a Council, comprising a President, a Vice-President (serving as Deputy President and President-Designate), a Secretary, and a Treasurer (the four Office-Bearers), along with nine other members ("Ordinary Members Council"). The immediate Past President may also be included as a member of Council, as provided below. The quorum at Council meetings shall be six and there shall be no casting vote.

4. The President and Vice-President shall be elected at an Annual General Meeting, to serve normally for a tenure of three successive years, and shall not hold their post for more than three successive years, but shall be eligible to serve again after the lapse of one year if re-elected. In addition, the immediate Past President may remain a member of Council for two years after the end of his or her term of office as President.

The Secretary and Treasurer shall be elected at an Annual General Meeting, to serve normally for a tenure of three successive years, and shall be eligible to serve again if re-elected, but should not normally hold office for more than six consecutive years.

The names of all candidates for election as Office-Bearers and of their proposers shall be made known to the Secretary before the Meeting at which election is to take place.

5. Any Office-bearer may be required to retire from office by resolution at any AGM, but the proposer and seconder of the resolution shall give a month's notice in writing to the Secretary (or in the case of the Secretary to the President), and the resolution must be precirculated to Members in the papers for the AGM.

6. Three Ordinary Members of Council shall be elected at each Annual General Meeting, to serve normally for a tenure of three successive years, and shall not be eligible for re-election at the end of their tenure until a year has elapsed; each year, the three Ordinary Members most senior by date of election shall demit office. If an Ordinary Member is otherwise unable to complete his or her term of office, the Council shall co-opt a replacement to complete the term, and this replacement shall be eligible at the end of the term to be elected for a further full term, despite having already served part of a term.

7. The Council shall have power to co-opt at any time other members who in their opinion are fitted to render special service to the Society. Such co-opted members shall be in addition to those in clause 6 above, and the co-option shall require the approval of each subsequent Annual General Meeting if it is to continue further.

8. To recognise outstanding service to the Society or to Medical History in general, upon occasion an Honorary Member of the Society may be elected at any Annual General Meeting. Any name proposed (with the name of a proposer and seconder, and details of the case) must be intimated in writing at least three months before the meeting to the Secretary, so that they are included in the precirculated Agenda for the meeting. Honorary Members shall pay no subscription.

9. The Annual Subscription shall be reconsidered from time to time by Council and reported to the Society at the Annual General Meeting. The Subscription (or revised Subscription) will fall due immediately following the AGM. A Member whose subscription is outstanding for a full year shall cease to be a member of the Society.

10. The Council shall ensure that full and punctual Accounts are kept for the Society and shall cause to be prepared once a year a Statement of Accounts and a Balance Sheet for the previous year.

11. The Society's funds shall consist of funds in the hands of the Treasurer, together with other sums of money and securities. These funds shall be held by the Treasurer, acting with the President and the Secretary (the Trustees), in trust for the Society's aims and objects, and in furtherance of this purpose the three Trustees shall have the following powers:

(a) Payments shall be made out of income or capital of the Society as the Trustees shall determine; all cheques shall require the signatures of two of the three Trustees.

(b) The Trustees may purchase and sell stocks, bonds, securities and other investments.

(c) The Trustees may delegate the management and investment of the Society's funds to the Treasurer and will consult with him on a regular basis as to the performance of the investments and assets comprising the Society's funds.

12. The Secretary shall keep brief Minutes of the proceedings both of the AGM and of the Council, shall prepare Agenda, and shall conduct the correspondence of the Society.

13. Meetings shall be held at least twice yearly, and the place of meeting shall be in any of the University centres, or elsewhere, as the Council may decide.

14. This Constitution may be amended at any General Meeting of the Society on four weeks' notice of the proposed amendment being given by the Secretary, such amendment to be included in the Agenda circulated for the Meeting. No such alteration or amendment shall have the effect of prejudicing the Society's charitable status in law.

15. The Council may resolve that the purposes for which the Society's funds are held can no longer be carried out by them or could be carried out more efficiently by some other body, fund or institution, and shall so report to a General Meeting of the Society; and if the General Meeting agrees, require the Trustees to make over the Income and Capital of the Society's funds to that other body, fund or institution whose aims and objects most closely resemble those of the Society, and so bring the Society to an end.

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