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Development of Interest in Lepidoptera

My father had been an accountant, but natural history was his major hobby. He was a typical 'Victorian naturalist' and had amassed collections of butterflies, beetles, minerals, shells (land and fresh water mollusca and shell cameos) and had a room full of cabinets. His interest had been life-long and I have a reprint of a paper on beetles given by him to the Kings College School Natural History Society in the 1870s. In his late years he had concentrated particularly on mollusca and had been for many years secretary to the London branch of the Conchological Society of Great Britain and Ireland. They held their meetings in his office.

I mention my father's hobbies because they encouraged my nascent interest in natural history (and collecting). Genetic influences have played a part in this, too. My father's mother had been a Miss Elizabeth Farn and she was related to A. B. Farn, a very well known 19th century British entomologist. H. C. Huggins, too, who died only recently, was a cousin of my father on the Farn side of his family and he had been an eminent microlepidopterist – by profession he was, however, a bank manager. As far as I know, none of my forebears had been professional scientists or doctors. My paternal grandfather had, I believe, been a lawyer.

Perhaps I should mention here that as a boy I helped my father with his shell collecting until his death in April 1928 and I continued for some years actively collecting land and fresh water mollusca until this interest was overtaken by my increasing involvement in medical work from 1930 onwards. My conchological interest, however, brought me in contact with the pathologist Professor A. E. Boycott, F.R.S., who, as well as being interested in carrying out research on the genetics of mollusca, e.g., into the inheritance of left-handedness (of the shells' spire), acted as recorder for the Conchological Society in relation to the distribution of mollusca in the U.K. I remember only too well sending him specimens of what I thought (erroneously) was a rare species and receiving from him a reply the short rejoinder: "No, A. E. B.".

In the mid-1930s I switched to collecting lepidoptera (of which I had started a collection when my father was alive) and I have maintained this interest ever since. First, this was confined to British butterflies and moths but subsequently this had been extended, parallel with the increasing opportunities I had for foreign travel, mostly to medical meetings and/or lecture tours. These trips, undertaken in the 1950s, 1960s and 1970s have taken me to many parts of Europe, and to N. and S. America, South Africa, India and Singapore, Australia and New Zealand; and I have usually managed to sample (although obviously only most superficially) the local butterfly fauna. In this way I have learnt, however, a good deal about the distribution of butterfly species on a world-wide basis and of the remarkable similarities (and dissimilarities) between the butterfly fauna in different parts of the world. At home, in Wimbledon, in my garden I have run a mercury-vapour moth trap more or less continuously since 1955 and have kept a diary of the catches, and have noted interesting changes in the prevalence of different species over the years.

My father had been married twice: his first wife died, I believe of tuberculosis, leaving him with three small daughters. He met my mother at the South-West (London) Choral Society: she was a member of the choir and he played the viola. Music had, in fact, an important influence on my childhood. I was surrounded by it. My mother was a good amateur pianist, particularly in respect of reading music and at accompanying, and a good deal of chamber music was played in the drawing room in our house in Putney. I learnt the piano – I was taught by one of my father's two sisters, but never achieved much, although music, particularly piano and chamber music, has given me life-long pleasure. One of my three half-sisters, Annette, was quite a good pianist and taught the piano, and my single full sister played the 'cello.

After natural history, particularly shells, and music, the third influence which I remember seemed to dominate my childhood was shortage of money. My father never really had enough money to bring up his five children and have a maid, nurse and cook, as he wished; he was always 'pinching and screwing' and I recollect having to have second-hand books etc. at school, and even sometimes second-hand clothes e.g., school uniform, which was a source of considerable humiliation.

My father's intention had been that when I left school I should help him in his business, which was virtually a one-man show. In the event he died when I was 15 before this became a practical possibility and I, for my part, having started biology at school, wished if possible to make a career for myself in a natural science subject rather than in accountancy. I had then no thought of medicine. It was my father's death which indirectly led to this. He had died rather suddenly, probably from a myocardial infarct, and the problem then arose as to what to do with his cabinets and collections. My mother was seriously short of money. My father had lent money – which he borrowed on the security of his two sisters' estate – to finance a business associate in a speculative venture. This proved to be a failure, and when the debt was paid my mother had virtually no capital and no income. All she was left with was a fairly large Victorian house. She managed to survive by taking in lodgers and my father's sisters paid for my schooling and when I left school I subsequently supported myself at London University from scholarships.

My mother wished to sell my father's collections, primarily to raise money. Two of his conchological friends, a Mr. J. E. Cooper and Lt. Col. A.J. Peile (a retired regular soldier) generously undertook to catalogue and lot his shells and cabinets so that they could be sold at Auction. (Stevens' Auction Rooms of King Street, Covent Garden, London, then regularly carried out auctions of natural history material.) The question of my future came up for discussion and Col. Peile remarked that his cousin – Sir Andrew Balfour – had done well; he had eventually become Director of the London School of Tropical Medicine and Hygiene. Why did not I think of medicine as a profession? I had certainly never thought of this, but the idea when suggested strongly appealed to me. I wrote to all the London Medical Schools for their prospectuses, with the idea of attempting their entrance scholarships. In the meanwhile, I started to work hard at school at botany, zoology, chemistry and physics and managed to pass the Higher Certificate and to obtain exemption from the intermediate London B.Sc. My gaining the Sambrooke Scholarship to King's College Hospital, London (KCH), the first of the London Medical School Entrance Scholarships I attempted, meant that I was able to start the second M.B. preclinical course at King's College, London (KCL) in October 1930, and to obtain the London County Council Scholarship support which was so essential for my maintenance.

I owe Col. Peile a great great deal; in fact much more than for making the suggestion that I might study medicine. He introduced me to microscopy. He was an expert on the radulae of mollusc, the ribbon of teeth with which the mollusc rasps away its vegetable food. Radulae are formed of chitin which is

insoluble in strong alkalis and by dissolving virtually all the body of the snail or sea-shell away in caustic potash it is possible to leave the radula intact and to make very beautiful and permanent preparations of the radulae. Col. Peile showed me how to do this and to stain them and I started to make a collection of radulae with great enthusiasm. I bought a second-hand Watson Service microscope – I think for £13:10:0, and later when at KCL an old rocker microtome also, and some other simple equipment for a home laboratory. I cut sections of a variety of animal and vegetable tissues and learnt a good deal about staining techniques, too. In 1933, when I became attached to a medical firm at KCH my home laboratory work took off in a different direction. I became fascinated with the blood and its diseases and I started to make blood films of the blood of all the patients with blood diseases that I could lay my hands on. I stained their films at home.

Looking back at the simple laboratory work that I carried out at home, it seems to me now that I was fulfilling a desire to do things with my own hands, as well as satisfying an inborn collector's instinct. My opportunity to make a comprehensive collection of blood films led me to wish to find out all I could about the changes in blood cell morphology which accompanied disease. (Later, I became to believe that the best possible morphological atlas of blood diseases was a personal collection of blood films. I used to preach this to trainees, but, I think, few followed my example.)

I became enthusiastically interested in medical work and seized when at KCH all the opportunities available to do practical work, either clinical or laboratory. I volunteered to do casualty work at weekends and on public holidays and was a very regular attendant at post-mortem examinations. I taught myself how to learn. My system was to abstract books and papers and to set out in summary form the points in the article I was reading which I did <u>not</u> know – such lists became admirable aide-memoires prior to exams. I found routine lectures a waste of time as a rule, partly, I think, because I knew already a good deal of what the lecturer was talking about and lost interest. However, I recollect one lecture of outstanding interest still. This was because it contained a great deal which was to me new and novel and was excellently illustrated. This was a special lecture given by Dr. A. Gilpin, a young physician, on congenital aneurisms of the cerebral arteries – the subject, I believe, of his recently obtained M.D. thesis.

I started subscribing to the Lancet in 1933, soon after starting clinical work, and its weekly arrival was eagerly awaited. I still subscribe, although I no longer find its content so exciting. Perhaps because of an unusual ability to learn, perhaps because of enthusiasm and interest, partly no doubt because I had to earn my living, I was successful in winning a whole series of prizes at KCL and KCH, including the Hughes prize for Anatomy, the Todd medal for Medicine, the Ware prize for Clinical Pathology, the Legg prize for Surgical Pathology, and the Senior Scholarship. With the proceeds of the Senior Scholarship I was even able to pay back to my aunts (my father's sisters) money they had lent me for my final exam fees.

I qualified MRCS, LRCP and MB BS, London in 1935 and was lucky enough to pass the MRCP in 1936 a few days after my 24th birthday. (I had passed the Primary FRCS soon after my 2nd MB at KCL). My first medical post was a locum surgeon job in Addenbrooke's Hospital, Cambridge and in 1935 and 1936 I held the house posts at KCH of House Physician to Dr. Firth, Dr. Gilpin and Dr. East, Resident Biochemist (to Dr. Lawrence and Dr. McCance) and Resident Pathologist.

Dr. Gilpin took an unusual interest in me; he encouraged my even then existing interest in blood diseases and allowed me to work in his small laboratory – he was hospital morbid anatomist and did the post-mortems as well as being a physician. (KCH at that time was years behind in medical organisation. They had

good clinicians but no full time medical scientist – with one shining exception, Dr. R. C. McCance.) I was thus able to cut sections of bone marrow and of spleens etc. It was at about this time that I bought a copy of the 2nd edition of Dr. Janet Vaughan's book The Anaemias and this certainly fuelled my interest in blood diseases.

It was while acting as resident biochemist, and as resident pathologist, that I learnt how to collect blood from patients. At that time blood sugar and blood urea tests were performed on capillary blood obtained by ear prick. This necessitated two 0.2 ml samples for each test and the post provided unrivalled practice in their collection! This was in the days when the laboratory staff – which in fact virtually meant the resident pathologist or resident biochemist – went to the wards to collect blood samples for blood counts etc. from in-patients and often from out-patients, too. One learnt to make blood films from capillary blood on the spot – by the cover-slip technique and this, too, provided valuable training; and blood for haemoglobin (Hb) estimation and red- or white-cell counts had to be collected similarly from the ear into separate pipettes. The idea of sending blood to the laboratory in a bottle or tube previously provided with an anticoagulant had not yet reached KCH, nor had the idea of examining the bone marrow by marrow puncture or the measurement of packed cell volume (PCV) and the calculation of absolute values.

The pathology staff at KCH was in 1935-1936 little more than vestigial. There was a general path lab, off which there were two small rooms. The larger of the two was the sanctum of the hospital pathologist, Dr. E. ff. Creed; the smaller – a very small room – belonged to the assistant pathologist. The third and junior member of the team, the resident pathologist – myself for instance – had two feet or so of bench space in the general lab. Dr. Creed mostly concerned himself with private practice – simple lab tests on urine, sputa, and the preparation of vaccines, but he also acted as a general consultant – he was in fact knowledgeable and undertook the Wassermann reactions one or twice a week. The assistant pathologist reported on surgical biopsy material and was mainly responsible for the bacteriological work; the resident pathologist did the haematological work, some bacteriological work too, and collected blood samples. He also gave blood transfusions throughout the hospital, that is to say grouped the patients (A, B, O only), bled Red-Cross blood donors and administered the blood. It was all hard work but of very great interest and I thoroughly enjoyed the experience and found it exciting. I remember vividly administering to a patient with a streptococcal septicaemia the first dose of Prontosil rubrum given at KCH and her miraculous response.

Dr McCance – who later as Prof. R. A. McCance, FRS became Professor of Experimental Medicine at Cambridge – was in my time undertaking pioneer work in salt metabolism, extra-renal uraemia and iron metabolism. I was involved in following up the haematological change which developed in a patient – a Salvation Army lady – who had polycythaemia vera whom McCance and his chief assistant Miss Elsie Widdowson (also elected FRS many years later) had decided to treat with acetylphenylhydrazine – then in vogue as a haemolytic agent and effective in reducing haemoglobin levels – and to follow the metabolic consequences of the haemolysis as completely as they could. One objective of the study was to find out the fate of the iron liberated from the haemolysed red cells. The lady was overdosed (the cumulative property of the chemical being not appreciated sufficiently) with dramatic effects on her metabolism and haemoglobin level. I noted the important effect the drug had on her blood picture and published a photomicrograph of her blood films (which I had kept) in the first edition of my book The Haemolytic Anaemias published in 1954. I have often quoted this study as an example of how much can be learnt from the detailed (and accurate) study of a single patient if the right questions are asked and the

appropriate techniques employed to study them. This study also further stimulated my interest in blood diseases – which had, of course, too, been strongly fostered by my experience as resident pathologist.

In 1936 I obtained a Salah marrow puncture needle and carried out the first puncture at KCH; and I was able to take the needle with me when I spent 6 months in Manchester in the Dept. of Haematology at the Royal Infirmary there. This department was headed by Dr. John Wilkinson, and Dr. Martin Israels was his first assistant. I introduced the technique of marrow puncture to them, and it was not long before one of their patients thought to be suffering from achrestic anaemia was clearly shown in fact to have monocytic leukaemia! (Dr. Israels was so impressed with the potentiality of marrow puncture that he later published a monograph on the subject An Altas of Bone-Marrow Pathology [1948]. I often felt that I had been a parent of this book!)

By 1936 I had decided to make pathology or least laboratory work my career, if I could. I had thought at one time of trying to embark on research on cancer. I remember wondering how I might start and a colleague at KCH, Dr. Philip Evans, later an eminent paediatrician, who had a contact at the Lister Institute in London, made some tentative enquiries on my behalf. It was suggested to me – I think by the then Director. Prof. Ledingham – that as a first step I should take up virology and that as a preliminary to this I should take the Diploma in Bacteriology course at the London School of Tropical Medicine and Hygiene. This was no doubt excellent and prescient advice. But I did not wish to take it, because it meant remaining a pupil and paying fees which I could not afford and I wished to marry! I decided instead to apply for a Medical Research Council (MRC) scholarship, then at £ 200 per annum, with the aim of taking up haematology. I was lucky enough to be successful in my application, and I remember having an interview with Sir Edward Mellanby, the then Secretary of the MRC himself. I did not make any attempt to ask anyone to intervene in any way on my behalf – but I subsequently found out that Mellanby had telephoned McCance, who had clearly not spoken unfavourably. The question of where I should work was next raised. I suggested Dr. Janet Vaughan at the British Postgraduate Medical School (BPMS). I had not met her but I had her book. The MRC agreed to this and then mentioned for a second 6 months the possibility of working with Dr. Engelbreth-Holm, of Denmark, then carrying out research into avian leukaemia, or, as an alternative, Drs. Wilkinson and Israels in Manchester. I elected to go to Manchester, because I wished to be near my fiancée. I have often speculated, if I had worked with Dr. Engelbreth-Holm, whether I would have ultimately ended up working in experimental cancer research after all, rather than on human haematological problems.

I spent the first 6 months on 1937 at the BPMS. Dr Janet Vaughan, Senior Lecturer in Clinical Pathology, who had been a disciple of Cecil Price-Jones (of Price-Jones curve fame), was interested in red-cell size measurements and had published a paper on red cell characteristics in acholuric jaundice (hereditary spherocytosis [HS]). It was suggested to me that I investigated how laboratory technique affected the results of the osmotic fragility test and also how anaemia affected the results. A paper was published with Dr Vaughan on the topic in 1938 (1).

Dr. Vaughan's department was unique at the time (at least in the UK) in that, although part of the Dept. of Pathology, it was devoted almost entirely to the laboratory aspects of haematology, including blood transfusion, and although Dr. Vaughan saw a few patients from time to time, she had no beds of her own. She was one of the first clinical pathologist to be a specialist 'laboratory haematologist' in the UK, and her

appointment set a pattern which was to be followed rather closely for the next 20-30 years, after which time more and more haematologists assumed clinical responsibilities with a consequent reduction in their former laboratory role.

I learnt a great deal during the 6 months I spent at the BPMS – including the importance of controls and statistical analysis, the value of high quality microscopes (binocular instead of monocular) and the importance of keeping the blood films of all patients as long as possible, in theory indefinitely. Above all I learnt of the importance of enthusiasm on the part of a departmental head and of getting things done without delay and of punctuality.

The current approach was predominantly morphological, although attempts at the assessment of the rate of red-cell destruction were made from urobilinogen excretion measurements. On the other hand, too little thought was given to the reason for, and the mechanisms behind the morphological abnormalities that were recorded and measured. For instance, microspherocytosis was looked upon as a characteristic feature of "acholuric jaundice" and that was that! It was a time, too, of missed opportunities for the carrying out of significant research. For instance, Winifred Ashby had described her method of measuring the life-span of transfused red cells as far back as 1919: we could have applied this to the "acholuric jaundice" cases then under investigation in Janet Vaughan's Dept. (The potential value of Ashby's method was not 'rediscovered' until the approach of World War II necessitated urgent studies into the preservation of blood in vitro for transfusion purposes. Its value and relative accuracy was then clearly demonstrated. Subsequently the technique was improved and it was used with great effect in the investigation of cases of haemolytic anaemia [6,7]).

I spent the second half of 1937 in Dr. Wilkinson's Dept. at the Manchester Royal Infirmary. Drs Wilkinson and Israels had beds and were essentially clinicians, but a disused ward had been converted into a laboratory. Their interest was primarily in pernicious anaemia and obscure anaemias not responding to stomach or liver extracts, then referred to as achrestic anaemia. When I had arrived on the scene they had just diagnosed a case of paroxysmal nocturnal haemoglobinuria (PNH), and it was suggested that I investigated this patient. I was aware that Dr. Hale Ham had been investigating a similar case in Boston, having been told of this patient by Dr. W. B. Castle when he visited Janet Vaughan at the BPMS while I was working with her. I thus knew that the Boston patient's haemolysis appeared to be affected by acid-base balance, for alkali therapy had produced some benefit. I set out to see whether in the case of the Manchester patient I could demonstrate any effect on haemolysis in vitro by alteration of pH. I was able to establish that the patient's red cells would undergo lysis in vitro in normal human serum or heparinised plasma if the pH was adjusted to acid side of neutrality. It was concluded that the patient's red cells had become sensitive to a potential lysin present in normal sera (2); the possibility of this 'lysin' was in fact complement (as subsequent studies have demonstrated) was not discussed. It is interesting in retrospect to recall that the suggestion was then made that the lysin might be a "natural heterolysin" or "group-specific isolysin" to which the patient's red cells had become "antigenetically suitable". This is the sort of mechanism now regarded as a possible explanation for the "false-positive" acidified serum tests in the rare congenital dyserythropoietic disorder HEMPAS (122,142).

In 1938 I returned to KCH, having been appointed to the Will Edmonds Clinical Research Fellowship, at that time a private fund; it was subsequently administered by the Royal College of Physicians. I held this Fellowship until the beginning of World War II in September 1939. I worked in a small side room adjacent to the main path lab at KCH. My research aim was to investigate several families with acholuric jaundice (HS) who were under the care of Dr. A. Gilpin. I studied the effect of splenectomy on osmotic fragility and demonstrated some interesting changes, in particular, a transient increase in fragility immediately after the

operation and the subsequent loss of the tails of very fragile cells which had been a conspicuous feature in most of the cases before splenectomy (5). The transient increases were attributed to the expulsion of fragile blood from the spleen into the peripheral circulation as the result of compression and manipulation of the organ during its removal. The loss of the tails of fragile cells was thought to be due to the removal of an organ which increased the fragility of the cells which circulated through it.

The availability of several spleens as the result of therapeutic splenectomy provided the opportunity for perfusion experiments designed to see whether the striking congestion of the spleen pulp which was obvious in histological sections could be the consequence of a vascular abnormality, i.e., caused perhaps by an absence of direct pathways from arteriole to venous sinuses. Large ova (fowl red calls were used as test objects but they appeared in the efflux of cannulated splenic veins as quickly in the acholuric jaundice spleens as in control spleens, suggesting that there was in fact no obstruction to direct arteriolar-venous pathways. However, it was found that whereas control spleens could quickly be freed of red cells by perfusion with saline via splenic arteries, many red cells remained in the spleen pulp in the acholuric jaundice spleens after a similar period of perfusion. It was concluded that the observation supported the view that there is in-vivo stagnation of blood in the spleen pulp in acholuric jaundice and that much of the pulp might be a backwater outside the main current of the blood stream. A modern interpretation of these findings would be that many of the red cells failed to escape from the spleen, despite the perfusion pressure, because so many were present and because their spherocytic shape and unusual membrane rigidity (which is characteristic of HS) prevented their ready passage through the stomata in the venous sinuses (5).

While working at KCH in 1938-39 I had repeatedly observed that the blood of acholuric jaundice (HS) patients underwent spontaneous lysis (autohaemolysis) in vitro much more rapidly than did normal blood. The cause of this could not be established, but it was shown to be independent of the presence of plasma or serum (and of the number of leucocytes present), i.e. it was determined by an abnormality of the red cells (4). These observations were expanded and developed after World War II and were the basis of the autohaemolysis test (37).

On the onset of the War I remained in London in KCH where I was responsible for the blood transfusion and clinical pathology service at the hospital. In January 1940 I was moved to a central Emergency Medical Services (EMS) laboratory which had been set up in Epsom College, south of London, where I worked as a general pathologist until January 1943 when I joined the Royal Army Medical Corps (RAMC) as a specialist in pathology. In 1941 and 1942 I had the opportunity to collaborate with Dr. P. L. Mollison, then at the South London Blood Transfusion Depot at Sutton, on the application of the Ashby technique to the study of cases of haemolytic anaemia. It was clearly established that in HS transfused normal red cells survived normally (6), an important observation which demonstrated that, although spherocytic haemolytic anaemias might be the results of the action of haemolysins, as had been demonstrated by Dameshek and Schwartz in 1936 and 1940, this could not explain the spherocytosis and haemolytic anaemia of HS. A further PNH patient was also studied at that time: additional observations were made on the effect of pH on lysis in vitro (8), and it was clearly shown that transfused normal red cells survived normally (7). Studies were also continued on a patient who was one of three children in the same family who suffered from aplastic anaemia (Fanconi's anaemia). This patient had been shown in 1939 to have red cells which behaved in vitro as did PNH cells. Remarkably, he eventually recovered both from the marrow aplasia and also from the PNH abnormality. An account of this patient was published in 1944 (9) and further details

given in 1961 (69). These papers, it is believed, give the first description of the now well known association of PNH with marrow aplasia – and also of recovery from well-authenticated PNH.

From 1943 to 1946 I served in the RAMC in Command Centre Services (CCSs) and in General Hospitals and undertook a wide range of diagnostic pathological work; I also served as blood transfusion officer. In 1944-45 I was attached to the Canadian Army and with a Canadian Officer and a mobile laboratory studied blood loss in battle casualties in Belgium and Holland. We employed Evans blue dye for the estimation of plasma volume and the Ashby technique to study the survival of transfused blood (10) and to estimate from this the proportion of the circulating blood which was the patient's, after his blood volume had been restored by transfusion. This allowed us to calculate the approximate volume of blood lost as a result of wounding. It was clearly shown that men might recover having lost half or more of their original blood volume.

In March 1945 I was posted to a General Hospital in Belgium where I acted as pathologist. It was to this hospital that all cases of suspected diphtheria were transferred, and I thus gained some experience in the typing of C. diptheriae.

In June 1945 I was posted to Italy, en route for the Far East. Soon after I arrived in Italy (Naples) I contracted infective hepatitis and by the time I had recovered the war with Japan was over and the Far East posting was cancelled. I remained as pathologist to a General Hospital in Italy until the end of December 1945 and from there I was posted to Palestine and ultimately to the Central Pathological Laboratory in Cairo. I obtained a Class B release from the RAMC in September 1946 to take up the appointment of Senior Lecturer in Clinical Pathology at the BPMS in London, the post that Janet Vaughan had previously held. The vacancy was occasioned by her appointment as Principal of Somerville College, Oxford. I remained at the BPMS, subsequently renamed the Royal Postgraduate Medical School (RPMS), until my retirement at the end of September 1977, a period of just over 31 years. In 1950 I was made Reader in Haematology in the University of London and in 1957 Professor of Haematology, and the name of the department I was in charge of was changed from that of Clinical Pathology to Haematology.

The Department of Haematology at the RPMS, although an academic department within the Division of Pathology with an importance teaching and research commitment, was nevertheless responsible for the routine haematological and blood transfusion work of Hammersmith Hospital, the hospital associated with the RPMS. This arrangement, which set the pattern for other academic departments of haematology subsequently established – for the Chair at the RPMS was the first to be set up in the U.K. – had both advantages and disadvantages. The advantages, however, outweighed the disadvantages, for the arrangement meant that all the haematological work of the hospital passed through the department, with a consequent relatively large amount of material and problems potentially available for teaching and research; the sole but important disadvantage was that the small staff of the department was chronically overloaded with routine work, much of it relatively uninteresting, to the detriment of its teaching and research programme.

The department, from very small beginnings, gradually grew in size and reputation and by the 1960s it had become a large one, certainly the largest in the U.K., in which research on a wide range of haematological subjects was pursued. In 1970 the Medical Research Council set up a Leukaemia Unit within the department with a consequent large increase in research and teaching in this field.

At first the Department had no beds of its own and patients referred to me and other members of the haematological staff had to be admitted into beds allocated to staff members of the Dept. of Medicine. This often worked well, for it provided expert general medical care for the patients. On the other hand, there were obviously disadvantages. Not infrequently a bed for a patient with a haematological problem was difficult to find, the physician perhaps having other patients on his waiting list of more interest to him or who in his judgement required a higher priority for admission. Occasionally, too, once the patient had been admitted there was conflict between the views of the physician or his staff and those of the haematologists as to how the patient should be best treated.

In 1960 this problem was alleviated by the creation of a joint appointment between the Dept. of Haematology and Medicine for a clinical haematologist, a physician with a major interest in haematological problems. Dr. M. C. Brain was the first appointee and subsequently Dr. E. Gordon-Smith. A small number of beds were allocated to the clinical haematologist and he, too, provided a consultative clinical service for patients who had a blood disease problem in other clinicians' beds. This arrangement worked well and when the MRC Leukaemia Unit was established Hammersmith Hospital built an "Anaemia Ward*" of eight beds in separate rooms, and out-patients facilities, too, for blood-disease patients were provided. The Anaemia Ward has proved to be of great value in the management of patients severely ill with leukaemia or aplastic anaemia.

*This ward was later named the "Sir John Dacie Ward".

My personal view on the organisation of haematological services in a hospital were first summarized in papers published in 1960 (59) and 1962 (74). In these papers were set out how I then thought they should be best organized and the concept and responsibilities of a department of haematology. The importance of the close collaboration between laboratory-trained and clinically-trained haematologists was stressed. In district general hospitals the senior staffing arrangement to aim at was considered to be a consultant of each species of haematologists whose complementary experience and training would provide a wide range of expertise. In recent years the training of haematologists in the U.K. has been based on the concept that the Consultant in haematology should be able to take charge of the diagnostic haematological laboratory as well as being competent to take full responsibility for the clinical care and treatment of patients with serious blood diseases. This is in my view a difficult task, but with the increasing part that non-medical technical staff and automated equipment can play in the running of a laboratory service it is inevitable, and not unreasonable, that the role of the medically qualified haematologist will become more and more clinical.

Research

During the 31 years that I worked at the RPMS my personal research effort was concentrated on the haemolytic anaemias. In fact, I continued more of less where I had left off before the interruption cause by the war and service in the RAMC, taking advantage of patient problems as they presented. My main contributions have been on PNH, on hereditary spherocytosis (HS) and elliptocytosis (HE) and in-vitro autohaemolysis, on the non-spherocytic hereditary haemolytic anaemias, on the unstable haemoglobinopathies, on auto-immune haemolytic anaemias, the auto-antibodies and the antiglobulin test, and on haemolytic anaemias of mechanical origin. Throughout I have attempted to throw light, mainly by laboratory studies, on the mechanism by which the life-span of the red cells has been shortened and the

significance of any major alterations in red-cell morphology present in relation to the increased haemolysis.

I have been fortunate to have had throughout most of the studies I have carried out the assistance of RPMS staff colleagues and of a series of postgraduate research fellows. This has been, I believe, of mutual benefit; and many of the research fellows now hold or have held senior positions in haematology or medicine in many countries of the world.

Paroxysmal Nocturnal Haemoglobinuria (PNH)

In relation to PNH the most important observations were as follows:-

Saline-washed normal red cells were shown to survive well in PNH patients and the transfusion of such cells was shown not to lead to febrile reactions or increased haemolysis of the patient's own red cells (13).

The survival of PNH red cells in normal recipients was shown to be impaired and a correlation was demonstrated between the cell's in vivo survival and the lytic action of the recipient's serum in-vitro (15); the shape of the elimination curve of PNH red cells in normal recipients – at first rapid and then much less rapid – paralleled time-lysis curves obtained in vivo and indicated the presence of a proportion of cells resistant to haemolysis (15).

The great sensitivity of PNH red cells to haemolysis by iso-antibodies was confirmed and the failure of anti-D and anti-M sera to lyse PNH cells was demonstrated (18).

PNH red cells were shown to be lysed by the sera of many patients suffering from auto-immune haemolytic anaemia and thus to be valuable reagents in the detection of haemolytic antibodies; treatment of PNH cells with the enzyme trypsin was shown to make them extremely sensitive reagents (27).

PNH cells were shown on incubation to liberate material with thromboplastic activity to a greater extent than did normal red cells and this 'non-haemolytic thromoboplastic activity' was thought possibly to be important in relation to thrombosis in vivo (54).

The sensitivity of PNH red cells to lysis by high-titre anti-I was explored: this was shown to parallel lysis in acidified serum. It was shown, too, that some cells sensitive to lysis in one test might undergo lysis in the other test, indicating that a proportion of the cells resistant to lysis in one test were, nevertheless, abnormal (62).

The reported low values for red-cell acetylcholine esterase in PNH were confirmed, and it was shown that the reduction in activity was correlated with the degree of sensitivity of the cells to lysis in acidified normal serum or by high-titre anti-I serum. It was shown, however, that even in severely affected patients some of the cells had a normal enzyme activity.

Studies were published on the variation in the clinical severity of PNH and also further data on its relationship with marrow hypoplasia (69, 79, 99); these indicated that in 30% of patients, at least, the PNH change is preceded by marrow hypoplasia, i.e. aplastic anaemia.

In 1963 (79) it was suggested that the PNH change might be due to a somatic mutation affecting marrow stem cells which gave rise subsequently to defective progeny which nevertheless had some biological advantage. Attention was drawn to the possibility of cure in PNH.

In a later paper (152) it was suggested that a likely explanation for the cures (10-15% of patients) could be that PNH clones lose viability with time, perhaps as the result of ageing. The patient's recovery might then depend upon whether he or she has surviving normal stem cells capable of repopulating the marrow. It was suggested, too, that the frequency with which overt PNH followed marrow aplasia was due to the hypoplasia providing "an unusually favourable opportunity for the PNH clone and that in the absence of hyperplasia the close has difficulty in establishing itself in competition with normal haemopoietic cells" (152).

In 1965 and 1966 further studies were published on the sensitivity of PNH red cells to lysis by complement (89, 92, 96, 97). These established without question the existence in the patients' blood of two populations of red cells, i.e. very sensitive cells and cells of much less sensitivity than normal; also that the severity of the patients' clinical presentation was positively correlated with the proportion of very sensitive cell present.

Data was published on neutrophil alkaline phosphatase (NAP) activity in PNH and it was shown that the lowest scores were found in the most severely affected patients, as judged by the acidified-serum test (90).

The complement sensitivity of AET-treated red cells (PNH-like red cells) was shown to be greatly increased. As was to be expected, however, only one population of cells could be demonstrated with respect to sensitivity, in striking contrast to naturally-occurring PNH cells in which two populations are the rule (100).

PNH red cells were shown to be more sensitive than normal red cells to the sulphydryl inhibitors PMB and PMBS; this was attributed possibly to the existence of an abnormality in the proportion of sulphydryl groups located in the outer surface of the PNH red cell membrane (130).

Hereditary spherocytosis (HS) and elliptocytosis (HE)

Early studies carried out in 1930-1939 (5, 6) have already been referred to. In 1953 an account was published of some cases of "atypical congenital haemolytic anaemia" (30). In this series of patients were two families in which a mild form of HS was diagnosed; in one of the patients a curious morphological appearance of the red ells – "pincered cells "– was described. A male infant suffering from severe haemolytic anaemia dating from birth was also described. This was considered to be a variant of familial (hereditary) elliptocytosis. His anaemia was relieved by splenectomy and, subsequently, his blood film was characterized by the presence of numerous fragments of red cells as well as microspherocytes. This rare disorder is now quite well known and recently has been labelled "pyropoikilocytosis". A fuller description of this unusual case was given in the 1st and 2nd editions of The Haemolytic Anaemias (1954; 1960).

Autohaemolysis

Further studies on the used of the autohaemolysis test in the laboratory differentiation of cases of hereditary haemolytic anaemia were published in 1953 and 1954 (30, 37). Accelerated haemolysis was described not only in HS and HE but also in several cases of non-spherocytic haemolytic anaemia not responding to splenectomy. It was noted that whereas in HS adding extra glucose to incubating blood usually markedly retarded haemolysis, in two non-spherocytic patients glucose was of no benefit (a Type II result). From this and other evidence (i.e. the demonstration of impaired glucose utilization) it was concluded that "the greatly increased lysis of these cells in-vitro, and probably also in vivo, is related to their defective glucose utilization" (37). This was the first suggestion that the basis of certain cases of

hereditary haemolytic anaemia was a defect in red cell metabolism. The measurement of the rate of autohaemolysis, and the effect of glucose on the result, is still used as a screening test in the laboratory differentiation of hereditary haemolytic anaemia despite its admittedly non-specific nature (87). The value of a refined technique in the diagnosis of pyruvate kinase (PK) deficiency was described in 1968 (109).

The unstable haemoglobinopathies

In January 1950 I found that large Heinz bodies were permanently present in practically all the red cells of a small child who had been splenectomized for a congenital haemolytic anaemia of unknown type. No explanation for their presence was forthcoming at the time. Subsequently, however, it was established that the patient was forming an unstable haemoglobin, which was described by Steadman et al (1970) as Hb-Bristol. An account of this case was originally given by Cathie in 1952. In 1962 Grimes and Meisler describe, from the Dept. of Haematology at the RPMS, that solutions of haemoglobin from a similar case were unstable to heating at 50°C. (This patient was described later as suffering from Hb-Hammersmith (102).) Further studies on the unstable haemoglobin of this patient were published in 1964 (84), when it was reported that about half of the haemoglobin being synthesized was unstable. Five patients belonging to another family in which an unstable haemoglobin had been demonstrated were also investigated and it was established that Heinz bodies were only to be found in fresh peripheral blood in the one patient who had been submitted to splenectomy. It was noted, however, that in the other unsplenectomized patients small Heinz bodies formed in their blood if it was incubated at 37°C in vitro, e.g., for 24-48 hours. In the patient in this family who had undergone splenectomy the presence of numerous granules in the littoral cells of her spleen unaccompanied by haemosiderin, suggested that in vivo the spleen was, in fact, removing Heinz bodies from red cells, probably without destroying them, during their passage from pulp to sinuses. By 1971 White and Dacie, in a review (136), reported that at least 30 different types of unstable haemoglobin had by then been described and that they represented a whole family of abnormal haemoglobins which had one feature in common, namely that the nature and position of the amino acid substitution led in each case to instability of the haemoglobin molecule.

The autoimmune haemolytic anaemias and auto-antibodies

Papers on the haemolytic activity of auto-antibodies were published in 1949 (17) and in 1950 (20) when the importance of pH for the demonstration of haemolysis in vitro by cold auto-antibodies (high-titre cold auto-agglutinins) was demonstrated.

In 1950 it was reported, too, that incomplete forms of cold auto-antibodies could be demonstrated, by means of the antiglobulin reaction, in many normal sera (21). (These antibodies were later demonstrated to have anti-H specificity). In 1951 descriptions were given of three patients suffering from the cold haemagglutinin syndrome (chronic haemolytic anaemia, Raynauds' phenomena and haemoglobinuria). The haemolytic potentiality of the antibodies and the effect of pH on lysis, was demonstrated in vitro (26).

In 1951, too, de Gruchy and I published an account of a detailed study of the sera of 19 patients with warm- or cold-antibody types of auto-immune haemolytic anaemia. Enzyme-treated (trypsinized) red cells and PNH red cells were found to be valuable reagents in the demonstration of antibody activity. The wide patient-to-patient variation in the antibody patterns was thought to be a reflection of the individuality of the patients' responses to the stimuli causing the formation of the auto-antibodies. Haemolysins were considered to play a part in the production of the increased haemolysis but not to be the only cause. The

evidence of responses to splenectomy suggested that the spleen acted as a remover of altered or damaged cells rather than as an important source of antibody.

In 1951, too, some significant observations were reported on the differences in behaviour of antibody-sensitized red cells to agglutination by antiglobulin sera to which various amounts of human γ–globulin had been added. Whereas the addition of small amounts of γ–globulin always inhibited the agglutination of red cells sensitized by anti-D antibodies and also that of the red cells from some patients with autoimmune haemolytic anaemia (AIHA), in other AIHA patients inhibition was less marked and cells sensitized with cold antibodies were only inhibited if very large amounts of γ globulin were added. It was concluded that the antibodies such as anti-D were γ globulins and that the cold antibodies might not be or that, alternatively, the antiglobulin sera were reacting with a component of fresh serum (not a γ–globulin) adsorbed with the antibody (24). Later work demonstrated conclusively that the 'non γ' type of reaction was due to a reactivation with complement adsorbed as the result of red-cell-antibody interaction (47, 53).

In 1956 substantial differences in the agglutinability of human red cells by high-titre cold antibodies were described (45) and in 1957 it was reported that in a paper electrophoretic study of serum proteins in cases of auto-immune haemolytic anaemia abnormal peaks were regularly demonstrable in the γ region with sera containing cold agglutinins at very high titres (48). These protein peaks were shown to be formed by the cold antibody macromolecular protein (49). In 1960 details of some experiments were published which demonstrated that adsorption of complement was a much more important mechanism of haemolysis that was intense auto-agglutination (61). In collaboration with D. L. Brown and P. J. Lachman a series of experiments were carried out on rabbits to determine the role of fixed C3 in the non-lytic destruction of red cells. These experiments showed that red cells which had adsorbed C3 (EC43 (5)) became attached to phagocytic cells and that, while some were immediately ingested, with time unphagocytosed cells returned to the circulation as spheroctes (131).

Haemolytic anaemias of mechanical origin

Microangiopathic haemolytic anaemia In 1962 it was reported that a peculiar blood picture, referred to as microangiopathic haemolytic anaemia, characterized by the presence of numerous distorted and fragmented red cells, was associated with the presence of pathological changes in small blood vessels, e.g., intraluminal hyaline thrombi. This type of blood picture was found in thrombotic thrombocytopenic purpura and various other types of haemolytic-uraemic syndrome and also in cases of disseminated carcinoma (75). Subsequent experimental work established that the haemolysis resulted from the traumatic fragmentation of red cells caught up by and attached to fibrin strands in loose thrombi (106, 107, 111, 112). An increased rate of fibrinogen was demonstrated in human cases (110).

Haemolytic anaemia following cardiac surgery In 1961, for the first time, a patient was described in whom continuous intravascular haemolysis followed cardiac surgery (70). His blood picture was, too, characterized by the presence of numerous red-cell fragments. At a second operation a defective mitral valve was found to be allowing a jet of blood to be playing on the artificial interauricular Teflon septum which had been inserted at the first operation. Covering up a bare patch in the septum on which the jet of blood was playing resulted in immediate cessation of the intravascular haemolysis. This thus appeared to be due to interaction between red cells and the bare Teflon, i.e. to be of traumatic origin.

Teaching

Throughout my appointment at the RPMS I was responsible for organizing the Department of Haematology's teaching programme. The most important commitment was an annual 6-week course (in later years extended to 10–11 weeks) as part of the Dept. of Pathology Course for the London University Diploma in Clinical Pathology. In the late 1960s and 1970 an annual postgraduate 'June Course' in 'Advances in Haematology' was held which proved popular, often with attendances exceeding 100. In addition to teaching in the U.K., I have participated in courses organised in Universities abroad, as well as giving research or review lectures from time to time in many places of the world, e.g., in most European countries, North America (Canada, the United States of America, Mexico), South America (Argentina, Chile, Peru, Venezuela), South Africa, India, Singapore, Australia and New Zealand.

Appointments and Honours

Overseas and International

Honorary Doctorates of the Universities of Uppsala (1961) and Aix-Marseille (1974)

Honorary Fellowships of the Royal College of Pathologists of Australia, of the Royal College of Physicians of Canada, and of the College of Medicine of South Africa

Honorary Memberships of Societies of Haematology, including the International Society

The Henry M. Stratton Award (Paul Ehrlich Medal) and Lectureship of the International Society of Hematology (1968)

President of the 5th Congress of the European and African Division of the International Society of Haematology (London, Sept 1975)

In the U.K.

Honorary Membership of the British Society for Haematology; President in 1964

First Editor of the British Journal of Haematology (1955-1962) then Chairman of the Editorial Board of the Journal until 1980

Foundation Lecturer of the Association of Clinical Pathologists and recipient of the Dyke Medal (1976)

Oliver Sharpey Lecturer (1962) and Croonian Lecturer (1973) of the Royal College of Physicians

Davidson Lecturer (1967) of the Royal College of Physicians of Edinburgh

Emily Cooley Lecturer of the American Association of Blood Banks (1979)

President of the Royal College of Pathologists (1972-75); Kettle Lecturer 1970

President of the Royal Society of Medicine (1977); President of Sections of Experimental Medicine (1958) and Pathology (1963)

Chairman of the Medical Advisory Panel of the Leukaemia Research Fund (1974-1982)

Chairman of the MRC Working Party on Leukaemia in Adults (1970-1978); Chairman of Leukaemic Steering Committee (1978-1982)

Fellow of the Royal Society (1967); Council 1979-81

Knight Bachelor (1976)

Professor Sir John Dacie's publications (references 1-148 from the 3 volumes [1938-1978] presented on retirement)

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