GUSTAV VICTOR RUDOLPH BORN
29 July 1921 — 16 April 2018
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Elected FRS 1972

BY ROD FLOWER FRS*

The William Harvey Research Institute, Queen Mary University of London,
Charterhouse Square, London EC1M 6BQ, UK

Gustav Born (Gus) made fundamental contributions to the study of blood platelets and their role in thrombogenesis. Working first on the biochemistry of these cells and their granules, Gus later devised an extremely effective method to measure their dynamic responses when stimulated to aggregate by pro-thrombotic stimuli. ‘The Born Aggregometer’, as it is known, revolutionized the study of platelets and the diagnosis of platelet-related disorders. Gus was elected a Fellow of the Royal Society in 1972 for his work and subsequently awarded the Royal Medal. He received numerous other accolades and awards for his contributions to cardiovascular medicine.

FAMILY AND EARLY LIFE

The Göttingen years (1921–1933)

At the time of Gus’s birth (29 July 1921), the Born family had lately arrived in the medieval town of Göttingen in Lower Saxony, Germany. He was the youngest of three siblings: his two sisters, Irene and Margaret (known as Gritli), having been born in Berlin in 1914 and 1915, respectively.

Gus’s father Max Born (FRS 1939), the distinguished mathematician and physicist, had recently been appointed as Director and Professor of Theoretical Physics at the University of Göttingen. This was an institution which, since the late eighteenth century, had established

*Email: r.j.flower@qmul.ac.uk

Note: Additional material relating to the life of Gus Born is supplied in an online Appendix, which is available at https://royalsocietypublishing.org/doi/suppl/10.1098/rsbm.2019.0026.

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itself at the epicentre of German science and where Max had established a world-renowned
department attracting many outstanding students and colleagues, some of whom later
become Nobel Laureates (as did Max himself). Gus’s mother was Hedwig Born (née
Ehrenberg), a literary figure of some stature and a talented poet.

Both Max and Hedwig came from ancient and distinguished families. Max could trace his
family back to the eighteenth century through successive generations of European Jews, many
of whom were prominent academics, physicians, industrialists or patrons of the arts. His father
was Gustav Born (in whose honour he named his son), a prominent embryologist. Hedwig
(known as Hedi) was half-Jewish and could trace her family history back to the fourteenth
century. Her ancestors (mainly academics) included historians, philosophers and theologians
as well as (on her non-Jewish side) the protestant reformer, Martin Luther.

Their Göttingen home became a meeting place for the leading intellectual and cultural
figures of the day. These included Max’s colleagues from within the physics community, such
as Albert Einstein (ForMemRS 1921; a particular family friend), Max Planck (ForMemRS
1926), Niels Bohr (ForMemRS 1926), Paul Dirac (FRS 1930), Pascal Jordan, Wolfgang Pauli
(ForMemRS 1953) and Werner Heisenberg (ForMemRS 1955; one of Max’s students and
treated by him as a ‘second son’), and several famous musicians of the day (the family were
talented amateurs), artists and writers. Another family friend that the Borns met through their
love of music was Albert Schweitzer, the great humanitarian.

Hedi (‘a curiously complex woman’, Gus once remarked) spent much of her life in her
bedroom surrounded by her books and busied herself writing, so Gus and his sisters were
brought up by the daughters of a Lutheran pastor who was a family friend. Hedi was an
indefatigable letter writer who corresponded with many of the leading cultural and artistic
figures of her day. She was also an accomplished (and published) poet whom Einstein (who
was fond of her) rated very highly, even comparing her in stature to the young Goethe.

During his childhood therefore Gus was surrounded by scientists, intellectuals and artists.
A family photograph shows him as a young child sitting on Einstein’s knee, and he
recalls whiling away many happy hours making paper airplanes from Heisenberg’s discarded
mathematical notes.

It was in Göttingen that Gus began his early schooling at the primary school, Albani
Volksschule (1926–1928), later attending the secondary modern school or Oberrealschule
(1929–1933).

Refugee scientists

The settled domestic and professional lives of the Born family came to an abrupt end in 1933
with the final collapse of the Weimar republic, the appointment in January that year of Hitler
as Reichschancellor and the ensuing collapse of constitutional democratic government. As
Hitler’s fascist party gained power, a wave of antisemitism swept the country. Disruptions at
the university caused by Nazi students increased, a Nazi rector was appointed to ‘oversee’ the
university and a systematic purge of ‘Jewish’ staff began.

Even though he was a non-observant Jew and anyway had converted to Lutheranism on
his marriage to his protestant wife, Max was nevertheless identified as being ‘Jewish’. He was
sent away on indefinite leave of absence, thereby losing in one cruel blow his world-famous

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1 Gus later commented ‘My parents tried to make [my childhood] as normal as possible’ (see https://www.youtube.com/watch?v=1At6lyPeFfw).
institute, his students and colleagues\(^2\). Family friends, including Einstein (who was already abroad), alarmed by the direction that events were taking, urged Max to leave Germany ‘immediately’ before it was too late. Max concurred, and in May 1933 Gus and his parents left Göttingen by train to travel to the Italian Dolomites. They moved into a small apartment in Selva (now close to a famous skiing resort) which they had rented for family holidays (Gus’s sisters were both studying in Switzerland).

Many family friends, including the Plancks, the Schrödingers and the Schnabels, visited them, as did Ernest Rutherford FRS who arrived one day in great style in a huge Daimler (personal communication, 2019). Max had first met Rutherford while studying in Cambridge before the war and in fact Rutherford had visited Max in Göttingen just the previous year, so they were already old friends.

Clearly, the family could not remain in Selva indefinitely, but, because of his eminence in the field, Max received many offers from academic institutions around the world (including Paris, Brussels and Belgrade). Ultimately, however, he decided upon Rutherford’s offer of a lectureship in Cambridge, and in October that year Gus and his father (together with their dog) boarded the night train to Calais, from where they could cross the channel to England. Hedi had preceded them to Cambridge and negotiated the purchase of a house in Hills Road. Once he had settled, Max was appointed Stokes Lecturer and elected a Fellow of Caius College\(^3\).

Gus continued his schooling in the town, attending the Perse School in 1933–1936 as a Foundation Scholar. His main interests during these early days were biology, English, and history. Both his sisters subsequently found husbands in Cambridge. Irene married Brinley Newton-John, a talented linguist who subsequently worked in intelligence during the war years, while Gritli married the physicist Maurice Pryce (FRS 1951).

The tenure of the Stokes Lectureship was three years, but, just as this phase of his life was coming to an end, Max received more job offers, this time from Moscow and the University of Edinburgh. The latter institution invited him to apply for the Tait Chair in Natural Philosophy, which had become vacant when the previous incumbent (Charles Darwin’s grandson, Charles Galton Darwin FRS) was appointed Master of Christ’s College, Cambridge. It was an offer that appealed to Max, and so the family moved once again, establishing their home in a rather traditional grey stone house in Edinburgh.

The family grew to love Scotland and its people and were delighted with the musical and artistic life of Edinburgh. Gus continued his studies here, attending the Edinburgh Academy (1936–1938). Once again, his main academic interests at the time included biology and history, but also music. Gus took up the flute and subsequently became a very proficient player, certainly of professional standard\(^4\). In 1938 he gained five passes in his Scottish Higher Leaving Certificate (English, mathematics, science, history and German).

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\(^2\) Max subsequently received a letter from Hitler briefly thanking him for his contributions and ‘releasing’ him from his university and academic duties (see https://theconversation.com/hitlers-letter-to-the-father-of-quantum-mechanics-3772).

\(^3\) Max and Hedi did return very briefly to Germany in 1935 to close up their Göttingen home, burning many of their superfluous possessions in the garden.

\(^4\) Years later, his friend, the English composer Kenneth Leighton (1929–1988), wrote a flute serenade for him which was subsequently published and recorded. A snippet from this can be heard in an interview with Gus by Sir Harry Kroto FRS and Edward Goldwyn at http://www.vega.org.uk/video/programme/92.
Undergraduate years; 1938–1943

The prospect of war was looming, and, at the urging of his father, Gus enrolled as an undergraduate medical student at Edinburgh University in 1938. Recollections of his undergraduate days included those of his anatomy lecturer Dr E. P. Jamieson, whom he recalled as being notorious for his skull cap and for his pedantry. Intellectually, Gus found himself drawn to pathology, especially inflammation and immunity, and he developed a real passion for physiology (taught by Professor Ivan de Burgh Daly (FRS 1943)). He also particularly admired the ‘brilliant lectures’ delivered by the pharmacologist Alfred Joseph Clark FRS 1931 and remarked how Clark’s book *The mode of action of drugs on cells* had captured his imagination.

The war years; 1943–1947

With the formal outbreak of war in 1939, medical schools were under pressure to increase production of doctors to support the armed forces, and Gus relates how the top twenty students in his year decided to forgo vacations in order to qualify in four-and-a-half rather than the usual five years. He therefore graduated with an MBChB in January 1943, aged 22. The usual house jobs followed, including one in the Western General Hospital with the distinguished surgeon James Learmonth. This experience left a deep impression on Gus, as well as an abiding aversion to clinical medicine as a long-term career choice.

In 1938 the Born family had become naturalized British subjects, and so, upon the final completion of his medical studies, Gus was liable for military service and he duly enrolled as a medical officer in the Royal Army Medical Corps (RAMC). Perhaps understandably, it was a time of heightened pro-nationalist sentiment in the UK and Gus was concerned that the name ‘Born’ might sound rather too ‘Germanic’. To avoid any problems, he decided to change his surname to Buchanan—the name of some close family friends in Edinburgh. By adding the first names ‘George Vernon’, he was able to retain his initials (G.V.B.).

It might be thought that the RAMC could have deployed a fluent German speaker to good advantage somewhere in the European theatre but, when his posting came through, the newly commissioned ‘Captain Buchanan’ was surprised to discover that he was to be dispatched to India. Here, he worked first as a physician for two years and then, after taking a clinical pathology course organized by the Army in Poona, as a clinical pathologist. He eventually headed up laboratories in Kalimpong-Darjeeling (West Bengal) and later Meerut, about 50 miles from New Dehli in Uttar Pradesh.

Nuclear devastation; 1945

His next posting, in December 1945, was to alter the course of his life. Gus was in Bombay (present-day Mumbai) on 6 August 1945 when the allies dropped a uranium bomb on the city of Hiroshima in Japan. After a second attack on Nagasaki the Japanese government sued for peace and Gus was appointed as one of two pathologists sent to accompany the British occupation force. He was stationed at an Army Base Hospital in Hiro on the Inland Sea four miles from Hiroshima. The area nearby was heavily contaminated. Images of the appalling

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5 ‘Why don’t you do medicine like your grandfather? Then, you won’t have to kill anyone and, you are less likely to be killed yourself.’

6 Gus relates some reminiscences of his medical student days in an interview with Harry Kroto and Edward Goldwyn at [http://www.vega.org.uk/video/programme/92](http://www.vega.org.uk/video/programme/92).
aftermath of the atomic blast stayed with him all his life. ‘In the rubble’, he said many years later, ‘people stood at the roadside, desperate, thin and starving, and very quiet. Thousands were still dying from the effects of radiation . . . . It was a vision you could never forget.’ (Seabrook 2009) Across the Inland Sea, Gus could see a sixteenth-century Shinto peace shrine. ‘I was struck by the contrast’, he said, ‘with the thousands of people dying three or four months after the bomb, with no food and medicine.’

His patients included large numbers of Japanese civilian casualties, many thousands of whom died from haemorrhages. Gus knew that this was secondary to thrombocytopenia caused by radiation-induced damage to the bone marrow and its consequent inability to produce platelets. It brought home to him the importance of these tiny cells and aroused a lifelong interest in platelet biology. ‘It occurs to me’, Gus wrote later, reflecting on the grotesque medical legacy of the bomb, ‘that I must be one of the few, perhaps the only one, still alive to have met Albert Einstein . . . the discoverer of $E = mc^2$ and witnessed the first, historically disastrous consequence of that equation in the destruction of Hiroshima by the first atomic bomb, a consequence deeply deplored by Einstein’ (personal communication, 2009). He might have added, rather ruefully considering his father’s deeply held pacifist convictions, that both Robert Oppenheimer, who played such a large part in the development of the Manhattan Project, and Klaus Fuchs, who betrayed its secrets, were both doctoral students of his father.

The final surrender of Japan marked the formal end of World War II, and Gus was finally demobilized in 1947. With the termination of hostilities, Gus no longer felt it necessary to use his nom de guerre; to inform his friends of this change, he sent telegrams and letters which simply read ‘Born again’.

James Learmonth had promised Gus a job in Edinburgh after the war, but when this did not materialize, Gus had to make alternative plans. While he did so, he continued his education with the aid of a government grant awarded to demobilized military staff. He attended (1947–1948) an Advanced Biochemistry Course run at University College London by Professor F. G. Young (FRS 1949; a pioneer researcher in diabetes) and took the opportunity to learn some experimental pathology in the Department of Professor Roy Cameron FRS.

Gus was particularly keen to work at the William Dunn School of Pathology in Oxford with Howard Florey FRS. Although the antibacterial properties of penicillin had been discovered in 1928 by Alexander Fleming (FRS 1943), it was Florey’s brilliant work with Ernst Chain (FRS 1949) and Norman Heatley on the manufacture and purification of the compound in the 1940s that led to the introduction into the clinic of one of the most important medicines of all time. Gus wrote to Florey and was overjoyed to be accepted as a research student. He was awarded an MRC Studentship for training in research methods commencing in 1948 (figure 1). Unfortunately, his euphoria was short-lived. His allotted supervisor was Dr W. E. ‘Kits’ van Heyningen (known as Van H) but for some reason that personal chemistry essential to creative intellectual partnerships just never developed. His relationship with Van H began badly and deteriorated thereafter.

For his PhD project Gus was given the topic ‘Bacteriolytic enzymes produced by moulds’, which, despite being of obvious relevance given the recent discovery of penicillin, Gus found to be of little interest. Even more distressing was the fact that an account of the very work that Gus was doing was published by a Belgian group before he could complete his thesis and he therefore had little to show for his three years’ work.
However, he did manage to produce one paper (1)* which subsequently was to have a crucial impact on his career. Entitled ‘The extracellular bacteriolytic enzymes of a species of Streptomyces’, the paper made reference to a method for measuring the bacteriolytic action of enzymes by monitoring the turbidity of the solution. ‘Bacteriolytic activity was estimated by measuring the percentage lysis, i.e. percentage decrease in turbidity of a standard suspension of autoclaved Bact. coli, caused by the lytic preparation in 15 min. under defined conditions’. This was a technique that he employed to great effect later in his career.

One personal event that lifted his spirits during this, otherwise rather depressing, phase of his life was his marriage in Edinburgh in 1950 to the psychoanalyst (Wilfrida) Ann Plowden-Wardlaw. The couple had three children: Max (Russell; born 1951), Sebastian (John Paul; born 1953), and Georgina (Emma Mary; born 1955).

Gus eventually graduated with a DPhil in June 1951 with a thesis entitled ‘Lysis caused by micro-organisms’. He had endeavoured to maintain an interest in science through these rather bleak years by reading widely but as he later, rather candidly, wrote, his relationship with his supervisor had ‘broken his spirit’ such that at one point he considered completely abandoning his hopes of a research career. Overall, it was not a propitious start to an academic life.

One day, however, Florey called him into his room to say that he was aware of the situation and that he should not lose confidence. He arranged a two-year position as research officer for Gus in the Medical Research Council (MRC) Toxicology Unit in Carshalton, Surrey, which was directed by John Barnes, a former student of Florey. Here, Gus studied the mechanism of pulmonary oedema in the rabbit. Gus later described Barnes as an excellent scientist and a charming, benevolent person. However, little of real note emerged in the way of publications from this period of his life despite Gus being given complete academic freedom by the benevolent Barnes. Luckily his academic fortunes were about to change.

* Numbers in this form refer to the bibliography at the end of the text.
Gustav Victor Rudolph Born

Oxford again; 1953–1960

In 1953 Gus’s friend Geoffrey Dawes (FRS 1971), then Senior Research Officer at the Nuffield Institute for Medical Research at Oxford University, invited him to join a team working on changes in the fetal circulation of the sheep at birth. This was a productive period and Gus participated in important discoveries concerning the autonomic control of the fetal circulation and the mechanism of closure of the ductus arteriosus. This work resulted in several important publications (5, 8, 9).

During the same period, he was appointed Departmental Demonstrator in the Department of Pharmacology at Oxford University, which was then under the charismatic leadership of J. H. ‘Josh’ Burn FRS. Here, Gus contributed to teaching the innovative practical classes using living tissue devised by Edith Bülbring (FRS 1958), and also joined her researching the physiology and pharmacology of smooth muscle (4, 7). Gus also worked with Hugh Blaschko (FRS 1962) on mechanisms of the cellular uptake, storage and release of biogenic amines in adrenal medulla and elsewhere. This was, he later remarked, a ‘postdoctoral initiation into pharmacological research’ (29).

The late 1950s was therefore a fertile time for Gus. Working alone or with Blaschko and others, he published a number of influential papers dealing with the relationship between 5-hydroxytryptamine (5-HT) and platelets. He observed, for example, that the amount of 5-HT in platelets was proportional to the amount of ATP (adenosine triphosphate) (11), suggesting that—as was the case with the adrenal gland (6)—this amine was stored in intracellular granules. In another important paper he studied the uptake of 5-HT by platelets (10), demonstrating that this amine was not just ‘bound’ to platelets (as was still believed by many) but was taken up in a process that suggested an active transport mechanism. These important observations formed the foundation of his later work.

Gus’s time in Oxford was important for personal as well as professional reasons and he remained in contact with many of the scientists he met there throughout his life. One proved to be of especial significance: it was here that Gus met John Vane (FRS 1974), who was studying pharmacology in Burn’s department. The two immediately hit it off, and although they published little together (their two joint publications concerned histamine and its effect on gastric acid secretion (2, 3)), John soon became Gus’s closest scientific associate and the two became lifelong friends (figure 2).

The Institute of Basic Medical Sciences; 1960–1973

The Institute of Basic Medical Sciences was created in 1950 in conjunction with the British Postgraduate Medical Foundation in order to provide an autonomous academic unit within the Royal College of Surgeons of England (RCS; then in the process of restructuring after being damaged by bombing in World War II) which could qualify for grant funding from the (then) University Grants Committee. The overall academic aim was to provide surgeons studying for the FRCS qualification with a sound scientific training.

Originally, the Institute, which was housed in refurbished nineteenth century neoclassical premises in Lincoln’s Inn Fields, comprised departments of pathology, anatomy and physiology, but the college also resolved to set up a department of pharmacology which would later be attached to the Institute. This was to be followed by a department of biochemistry. As Dr Y. S. ‘Mick’ Bakhle, who joined the department in 1965, recollects, ‘[Many expressed
surprise] that pharmacology, so obviously clearly and closely associated with Physicians, should be a subject of any interest to Surgeons . . . It is very likely that this strange, non-standard, academic structure contributed to the strange, non-standard results and opinions that emerged from the work of this Department during its 30+ years existence.’

The Chair of Pharmacology was originally held by W. D. M. Paton (FRS 1956), but he had recently moved to Oxford. When Gus was elected to the vacant chair in 1960, the acting head was ‘by a wonderful happy coincidence’ (Taylor 2008) his Oxford friend John Vane. Initially, the department was located on the top floor of the Examination Hall in nearby Queen Square, but in 1962 it moved to occupy the top floor of the main RCS building in Lincoln’s Inn Fields.

‘A career in pharmacology’

When Gus joined the department, it was not yet a mature research unit. ‘To begin with’, Gus remarked, ‘we didn’t have much going on—so we decided to make a film!’ This was intended to provide after-dinner entertainment for an upcoming meeting of the British Pharmacological Society at the College. Possibly because Gus had a weakness for humorous films such as those of the Marx Brothers genre, the film, A Career in Pharmacology, was a spoof of an eponymous booklet issued by the American Society for Therapeutics and Experimental Therapeutics in a worthy attempt to publicize career opportunities in the discipline. What started originally as a slightly off-beat suggestion eventually engulfed the department and consumed everyone’s time and energy⁷ (see online Appendix, §1).

⁷ John Thompson, who was then a senior lecturer in the department, has deposited detailed notes on this episode in the archives of the Wellcome Trust, which also has a copy of the film (see https://search.wellcomelibrary.org/iii/encore/record/C__Rb1646239?lang=eng).
Gus’s chair and research programme was supported by a large charitable endowment made to the RCS by the motor racing magnate Tony Vandervell. The University of London formalized this arrangement with the creation of a ‘Vandervell Chair in Pharmacology’. Gus was also appointed Honorary Director of the Thrombosis Research Group of the Medical Research Council.

With new premises and some guaranteed financial support, Gus was set to embark upon his most productive period. He immediately resumed his work on platelets and tackled the subject on a broad front, investigating further the mechanisms of uptake, storage and release of 5-HT in platelets using techniques he had learned while working with Blaschko.

The platelet ‘aggregometer’

When appropriately activated, platelets form—together with components of the blood clotting system—a haemostatic plug at sites of injury in blood vessels, thereby limiting blood loss. They do this by sticking together in a clump, a process generally referred to as ‘aggregation’ (also called ‘agglutination’ or ‘floculation’). Investigations into platelet behaviour at the time were hampered by the lack of a suitable quantitative method to measure this aggregation response. Measurement of platelet adhesion to glass \textit{ex vivo} had long been used, notably by Helen Payling-Wright (1941), who had joined Gus in his new department, but this was not a satisfactory substitute. Although many investigators still used this approach, it was increasingly replaced by direct optical observation of the cells in platelet-rich plasma (known as ‘PRP’\textsuperscript{8}), using a strong hand lens or by examination of a sample on a microscope slide. Several groups had shown that the addition of thrombin or adenosine diphosphate (ADP) to such a solution caused the platelets to aggregate, and this could be scored, thereby providing a semiquantitative estimate of the phenomenon (e.g. O’Brien 1961, 1962a).

It was against this background that Gus made his greatest contribution to experimental pharmacology: the invention of the ‘aggregometer’, which, in turn, launched the whole field of platelet ‘aggregometry’ (a term that he subsequently came to hate). Here, his familiarity with methods for measuring the turbidity of bacterial lysates that he had developed during his unhappy postgraduate years came into its own\textsuperscript{9}. Instead of laboriously estimating the amount of platelet aggregation with a hand lens, Gus had the simple—though inspired—idea of using a conventional laboratory absorptiometer to measure, dynamically and quantitatively, the changes in optical density during the aggregation process. Several technical issues had to be surmounted before this method could be usefully employed: for example, the platelet suspension had to be continuously stirred, necessitating the addition of a magnetic stirring rod small enough to fit into the transparent centrifuge tube used as an absorptiometer cuvette. Once inserted, changes in the optical density could then be read from the galvanometer, providing a dynamic picture of the aggregation response.

This simple arrangement worked extremely well, and Gus demonstrated the device at a meeting of the Physiological Society (12) and reported the technique the same year in a brief letter in \textit{Nature} in 1962 (13) (figure 3). In his first experiments Gus showed that the addition of ADP caused an immediate fall in optical density and during this time the platelets were observed to aggregate, generally in a reversible fashion.

\textsuperscript{8} Produced by low-speed centrifugation of citrated whole blood during which the platelets remained in the upper plasma phase while the red blood cells sedimented to the bottom.

\textsuperscript{9} He later joked that ‘it was the only good thing to come out of my DPhil at Oxford’ (see https://www.youtube.com/watch?v=1At6lyPeFfw).
Figure 3. Above: The first bespoke aggregometer produced in the workshop of the Royal College of Surgeons. Below: The first optical record of platelet aggregation by adenosine diphosphate (ADP) from Gus’s original paper (13). The tracing shows the increase in transmitted light, recorded as downward deflection on the y-axis, caused by the addition of ADP to platelet-rich plasma in the cuvette; x-axis, 5 min time units. ADP was added at time zero: (A) 2.5×10⁻⁷ M; (B) 5×10⁻⁷ M; (C) 1×10⁻⁶ M; (D) 2.5×10⁻⁶ M. Note the reversible aggregation seen at the lower concentrations. (Reprinted with permission from *Nature*.)

A more formal study appeared the following year, in which Gus, together with Michael Cross, characterized the system more closely (14). They reported that there was a linear relationship between the optical density and the number of platelets in the suspension, that ADP and calcium could initiate aggregation, that the initial rate of increase in aggregation was proportional to the logarithm of the ADP concentration, and that the rate of stirring affected the speed at which the aggregation occurred.

In most cases the ADP-induced aggregation was reversible, which Gus attributed to the breakdown of ADP in the plasma. In addition to ADP they also tested AMP (adenosine monophosphate), ATP, adenosine, and a range of other nucleotides and nucleosides, some of which were found to inhibit ADP-induced aggregation. Several other subsequent papers
appeared from the group further exploring this phenomenon, for example (16). In an important extension of their *in vitro* observations, Gus and Michael Cross also demonstrated that ADP injected intravenously into animals caused a transient fall in blood platelets (15).

The ability to follow the aggregation response in ‘real time’ led to several other important findings. As Gus had noted, one of the fascinating features of the aggregation response to some stimuli (such as ADP) was that it was preceded by a rapid isovolaemic ‘shape change’ in the platelets which, by scattering light, caused a transient increase in the apparent optical density of PRP. Later, Gus and his colleagues researched this phenomenon in some detail (18, 20), sometimes using a photocell detector set at right-angles to the incident light beam so as to measure scattered rather than transmitted light. They observed that the magnitude and velocity of this response was proportional to the logarithm of the ADP concentration and conformed to Michaelis–Menton kinetics. This, together with its speed of onset, was interpreted (correctly) by Gus and his colleagues as evidence that ADP acted at discrete and specific membrane receptors (now, of course, known to be the P2Y1 receptor).

Some years later, working in Cambridge with Paul Lattmer and Frank Michal, Gus demonstrated that the changes in transmission during this change of platelet shape could be accounted for by conventional light scattering theory (25).

In the case of aggregation induced by ADP, the shape change was followed by a biphasic aggregation response. This important observation was originally made using the aggregometry technique by Macmillan & Oliver (1965). ‘Michael Cross and I’, wrote Gus later (27), ‘had failed to notice, or to think about, the anomalous deformations in our manually plotted aggregometer tracings (this was before the availability of continuous recording), which turned out to be the optical manifestation of the platelet release reaction.’

The biochemistry of this phenomenon was of paramount interest. Gus’s colleagues, including David Mills, Gordon Roberts and Duncan Thomas (Mills et al. 1968; Mills & Thomas 1969), discovered that this ‘second phase’ of ADP-induced aggregation (later dubbed the ‘release reaction’) was accompanied by a substantial release from the platelets of stored ADP and ATP, 5-HT and ‘lysosomal’ enzymes such as acid phosphatase and β-glucuronidase. These mediators then served further to accelerate the aggregation response, in what became known as the ‘platelet feedback response’.

Increasing emphasis in the department on human platelet aggregation brought with it a major logistical issue: where to obtain sufficient fresh human blood. In the original papers, samples were often taken from colleagues, but, as demand increased, Gus turned to other sources. By chance, the building next door to the RCS was Her Majesty’s Land Registry Office, and on the other side of Lincoln’s Inn Fields were the offices of the Equity & Law Life Assurance Society company. These two institutions provided Gus with an almost inexhaustible supply of volunteer blood donors. As Mick Bakhle put it (see Atherton et al. 2019), ‘Using his customary charm, Born recruited, over the years, many hundreds of these willing donors who used to arrive, two or three on each working day, to donate 200 ml of their blood in return for a cup of tea and a biscuit.’ The selfless participation of these volunteers was a major factor in the success of the whole platelet project.

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10 Michael Cross, together with other prominent researchers in the field, tragically died in 1964 in an air crash on his way to a platelet meeting at Oak Ridge National Laboratories in the USA. Gus set up a memorial lecture in his honour.
Inhibitors of aggregation

One of the most important applications of the aggregometry technique was the search for inhibitors of platelet activation. ‘If it can be shown’, Gus had written, rather presciently, in the closing paragraphs of his original 1962 paper (13), ‘that ADP takes part in the aggregation of platelets in blood vessels it is conceivable that AMP or some other substance could be used to inhibit or reverse platelet aggregation in thrombosis.’

Using older techniques, several authors had screened a variety of common drugs and metabolic inhibitors (cf. O’Brien 1962a) as inhibitors of platelet adhesion or aggregation, but Gus’s aggregometer proved to be a breakthrough technology in this respect. His group had already shown that ATP and adenosine were weak inhibitors and that 2-chloroadenosine was much more effective (16). But the most important drug to emerge from this search was, rather surprisingly, aspirin.

The original observations suggesting that aspirin might have an effect on platelets can be traced back to the 1950s (Craven 1953), but it was not until over a decade later that this effect was ‘re-discovered’ by Weiss & Aledort (1967), who showed that 1.3 g/day aspirin administered to volunteers inhibited the activation of platelets by connective tissue fragments. The crucial observation that aspirin could block the ‘second phase’ of platelet aggregation induced by ADP, originally made by O’Brien (1968), had huge implications for the treatment of cardiovascular disease. It was very gratifying to Gus that many of the studies in this area relied heavily on his aggregometry technique.

In 1971, at the other end of the RCS corridor, John Vane and his colleagues demonstrated that aspirin was an inhibitor of the prostaglandin-forming cyclooxygenase in vitro and in vivo (Collier & Flower 1971; Ferreira et al. 1971; Vane 1971). The subsequent discovery that platelet cyclooxygenase also generates a potent prostaglandin-derived pro-aggregating substance, (now known as) thromboxane A2 (Hamberg & Samuelsson 1974), when these cells are stimulated, completed this conceptual circle and paved the way for the widespread use of aspirin as a ‘cardiovascular drug’—now its chief therapeutic role.

Oddly, completely independently of Gus’s work and John Vane’s experiments (although obviously drawing inspiration from them), another paper from the RCS group appeared in the same issue of Nature as Vane’s original report. Bryan Smith in Gus’s group and Anthony ‘Jim’ Willis in John’s group demonstrated that platelets in blood samples taken from volunteers who had received 1.2 g aspirin 60 min before venepuncture did not release prostaglandins when stimulated to do so with thrombin (Smith & Willis 1971). Their original hypothesis, that aspirin inhibited platelet phospholipase, turned out to be incorrect, but this was an important paper which showed for the first time that aspirin (and indomethacin) administration could inhibit platelet prostaglandin production ex vivo.

Summarizing all this work at a later date (27), Gus wrote ‘It is gratifying that the feedback hypothesis of platelet aggregation turned out to be explanatory of the remarkable effectiveness of antiplatelet drugs of the aspirin type in the prevention of heart attacks and strokes.’

Further development of the aggregometer

The original, and rather ‘Heath Robinson’, laboratory apparatus with which Gus had begun his platelet work had been gradually improved, often at the suggestion of his colleagues. Coupling a chart recorder eliminated the tedious necessity of recording the optical density manually, and a thermostatically controlled heating system was devised to maintain the sample at 37°C.
Before long, a free-standing bespoke ‘aggregometer’ (see figure 3) was fabricated by the departmental electronics engineer Ziggy Sabikowski, and, as the power of the technique was appreciated by the scientific community and the technique became more popular, requests for aggregometers began to flood in. The activity became a minor source of departmental income although Ziggy (later assisted by Len Cracknell) was hard-pressed to keep up with the demand. Ultimately, the concept was taken up commercially by several companies, who now supply the bulk of the instruments used around the world in research laboratories and hospitals.

Later, variant forms of the aggregometer appeared, such as the ‘lumi-aggregometer’ (Feinman et al. 1977) which enabled simultaneous quantitation of released ATP as well as light transmission, thus providing another insight into the mechanism of the aggregation response. Eventually, the single cuvette of the original aggregometer was replaced by a multi-well plate system enabling many samples to be read simultaneously, and concentration–response curves to be constructed with speed and accuracy, using a plate reader (Armstrong et al. 2009; Chan et al. 2011). A totally different type of aggregometer, able to measure aggregation in whole unseparated blood, was also devised (Cardinal & Flower 1980).

Gus was often asked why he had not patented the aggregometer. In answer he cited Florey’s response when asked the same question about penicillin: ‘Can one patent sunshine?’ It reflected a conviction, common in that era, that scientific advances with medical implications should not be exploited for commercial gain. But there was a sour footnote to the aggregometer story. Having been brought up in the tradition that one should be totally open about one’s work, Gus was always happy to demonstrate his new technique to anyone who wanted to see it. One of those was John O’Brien, an established platelet researcher and haematologist from Portsmouth, and so Gus duly invited him to the lab for a demonstration. When, in a short addendum (‘Some results from a new method of study’) to a paper on platelet adhesiveness, O’Brien reported some data obtained using an almost identical device (O’Brien 1962a, O’Brien 1962b), Gus was outraged. O’Brien acknowledged the priority of Gus’s paper, commenting that it had appeared ‘since this paper was prepared’, but Gus felt that an unwritten code of scientific etiquette had been breached. It caused a rift between the two men and Gus continued to be exercised by the episode until his last days.

But notwithstanding this unpleasantness, the aggregometer story was a huge success and could be said to mark the beginning of a new era of platelet biology. Within a few years both of the original papers became citation classics, and by January 2017 the Nature paper had been cited 5010 times and the Journal of Physiology paper (with Michael Cross) 2480 times. Gus was elected to the Royal Society in 1972 for this work.

In vivo studies

Observations of platelet responses to ADP and other aggregating agents in vitro led to many useful insights: but were they physiologically relevant? Gus’s early paper with Michael Cross suggested that they were (15) but Gus wanted to pursue this by examining platelet behaviour at the level of the microcirculation. Other groups attempting to address this question had relied upon experimentally induced ‘injuries’ to microvessels in order to study the activation of platelets. Reproducibility was the major issue here, as the site of injury had to be changed every time. To tackle this problem, Gus instead used micro-iontophoretic application of ADP

11 Although he also sometimes joked ‘Florey had morals so I could remain poor!’
to the outside of microvessels of the hamster cheek pouch. Working on this painstaking project with Gus was a PhD student, Nicola Begent. As this technique was already in use in a lab in the USA, ‘Obviously the most practical way of my making progress with the method’, she recalled later, ‘was for me to go to the States and learn all about it—then return home and set it up. So off I was sent and thus began a long struggle with a very experimental technique, which eventually paid off with a paper in Nature! Gustav was immensely supportive when we tried unproductive deviations and progress was slow—but the encouragement and joy when things went well and results began to appear were great!’

Their observations showed that ADP applied to the outside of the microvessels induced intravascular platelet thrombi that formed reproducibly at the same location without obvious damage to the endothelium. The resulting recordings, made under difficult conditions on 16 mm ciné film, made extraordinary viewing: following ADP application, clouds of platelets suddenly appeared from the flowing blood and coalesced rapidly at the site of application, the rate depending on blood flow and the presence or absence of inhibitors (17)\(^{12}\).

In a further extension of this technique, Begent & Born (19) compared a number of known inhibitors of platelet aggregation \textit{in vitro} using the aggregometer and \textit{in vivo} using this new technique. In light of subsequent developments in the field, the most striking result was that aspirin not only exerted an inhibitory effect on aggregation \textit{in vitro} but also prevented the accumulation of platelets following the iontophoretic application of ADP. As it turned out, this demonstration was of some practical importance, as regional administration of aggregation inhibitors was found to prevent thrombosis formation in artificial organs (24).

\textit{Rolling leukocytes}

‘Once seen, never forgotten’ was Florey’s comment upon the appearance of the microcirculation when viewed through a microscope. Gus had noticed that, whereas the endothelium seemed comparatively undamaged by his iontophoretic applications of ADP, there was nevertheless evidence of enhanced granulocyte adhesion to the site. This observation formed the basis of another long-term project aimed at quantifying granulocyte behaviour in the microcirculation. Working with him on this venture was a new PhD student, Anne Atherton (née Dawson). Their first paper used the microcirculation of hamster cheek pouch or the mouse mesentery as experimental preparations and once again, in that pre-digital world, the results were recorded on film. Gus noted the difference between the behaviour of adhering platelets and rolling granulocytes and demonstrated that chemotactic substances increased the tendency for these latter cells to roll along the endothelium (21).

‘The rolling of granulocytes is presumably governed by two forces,’ they wrote in a later paper (22), ‘the shear force of the flowing blood and an adhesive force between the surfaces of granulocytes and vascular endothelium. Our results suggest that, within limits, the proportionality between the velocities of blood flow and rolling cells is due to shear force, the adhesive force being similar for all the cells. The results suggest that this adhesion force per granulocyte is of the order of \(10^{-5}\) dynes’.

Gus was intrigued by the mechanism which normally prevented cell adhesion to endothelium. Since endothelial cells were rich in sialoproteins and carried a strong negative charge, he speculated that the basis for this could be an electrostatic repulsion. Gus

\(^{12}\) A demonstration of this can be seen in the interview with Harry Kroto and Edward Goldwyn at http://www.vega.org.uk/video/programme/92 (see the online Appendix, §10, for further links).
demonstrated that an infusion of sialic acid inhibited platelet adhesion but not that of
granulocytes, whereas an injection of neuraminidase, which removes sialoproteins, caused
a rapid fall in circulating granulocytes (23).

The significance of this work was that it provided a novel technique for quantitating
the rolling and adhesion of granulocytes in vivo. In subsequent years, the elucidation of the
biology of adhesion molecules became a major endeavour in its own right and led to the
detailed understanding that we now have of the way in which granulocytes ‘roll’, ‘stick’, and
subsequently migrate between and through endothelial cells during episodes of inflammation.
Most subsequent investigations used techniques very similar to those pioneered by Gus and
his team in those early experiments, which, as Gus subsequently observed, contributed to an
understanding of adhesive reactions prior to the discoveries of specific adhesion molecules. It
was a project that Gus returned to several times in later years.

A time of change

If this sketch of Gus’s career has concentrated excessively upon this phase of his life at
the RCS, it is because it was his most creative and productive. ‘The atmosphere’, he later
said of the department, ‘was singularly innovative and productive, so that we published an
extraordinary amount of valuable research which has stood the test of time. I was certainly
happiest during that time.’ (Taylor 2008).

Quite apart from the work that Gus and his team produced, John Vane’s group discovered
the mode of action of aspirin and the non-steroidal anti-inflammatory drugs and provided
the experimental and theoretical basis for what later became one of the major groups of
cardiovascular medicines, the angiotensin-converting enzyme inhibitors. In addition to this
string of major discoveries, several departmental staff and postgraduate students went on to
win major international prizes (including two Lasker Prizes and a Nobel Prize) or to achieve
eminence in academia or industry (see online Appendix, §2).

One major factor in its success was undoubtedly the relationship between Gus and John.
‘We had been friends’, wrote Gus some 30 years later, ‘for nearly ten years and our friendship
continues to this day. There was an easy-goingness; we were very open and totally at ease
with everybody, and if anybody was difficult, we soon cured them.’

During these years, there had been major changes in Gus’s personal life. His marriage to
Ann had been dissolved in 1960 and, in 1962, he had married Faith (Elisabeth) Maurice-
Williams, a graduate in medicine from University College London, with an interest in
microbiology but who later became a general practitioner. The couple had two children, Carey
(Lucy Natasha; born 1965) and Matthew (Stephen; born 1968).

But by 1973 there were developments in the air. John Vane was offered the position of
R&D Director at the Wellcome Foundation (at that time a pharmaceutical company with a
large UK research facility in Beckenham, Kent) and almost simultaneously Gus was offered
the Shield Chair in Pharmacology at Cambridge, which had stood vacant since the departure
of Arnold Burgen FRS in 1971.

ACADEMIC APPOINTMENTS AND RESEARCH TOPICS 1973–2018

The University of Cambridge; 1973–1978

It was a tough decision to leave the RCS. ‘I didn’t want to stay on without him [John]’ Gus
said later but the deciding factor in his decision seems to have been a remark made by a
fellow refugee pharmacologist, Wilhelm Feldberg FRS, over dinner one night. ‘Gus,’ Feldberg admonished, ‘as a refugee boy, one does not turn down chairs at Oxford or Cambridge!’ (Taylor 2008).

Nevertheless, it was with mixed feelings that Gus made the decision to leave the rather special department that he and John Vane had created. In 1973 he was appointed to the Shield Chair and subsequently elected a Professorial Fellow of Gonville and Caius College.

But these turned out to be difficult years for Gus. His family remained in London and he found the constant travelling back and forth to Cambridge rather stressful. On Monday mornings, students often reported seeing him anxiously hitchhiking up Hills Road from the railway station, evidently running behind schedule.

Despite many setbacks and, what Gus perceived as a general lack of interest in his work by many of his colleagues, he continued to manage the department with his customary élan. Noel Cusack recalled an amusing incident which took place about this time:

Gustav had the ability to put a positive complexion on almost any event . . . for instance, one of his post-doctoral scientists in the Cambridge laboratory would practise the classical cello in the early evening, and one could enjoy the lovely sounds floating through my adjoining wall. However, this performance was not universally appreciated. On one occasion, someone came storming down from a laboratory on the floor above complaining to Gustav, ‘Someone in your group is playing a cello!’ To which Gustav replied mildly, ‘Playing it well I hope’, thus completely defusing the situation and taking it in a new direction.

But Cambridge was not Gus’s happiest appointment. He had to deal with a heavy administrative and teaching load, neither of which he had during his time at the RCS. When he was approached about a Mastership of a college, he declined the invitation—‘I’m a researcher not an administrator’, he said later (Taylor 2008).

**Kings College London; 1978–1986**

It must, then, have been something of a relief when he was offered the Chair of Pharmacology at King’s College London after what seems to have been a somewhat informal interview with the Principal, Sir Richard Way.

Now closer to his family home, he was much more settled in his new labs in The Strand, and, together with several colleagues who had followed him from Cambridge, he forged ahead with his research programme. Gus eventually retired from Kings College, aged 65, in 1986.

**The William Harvey Research Institute; 1988–2018**

By chance, John Vane had also retired from the Wellcome Foundation that year and had been offered space on the Charterhouse Square campus of (what was then) St Bartholomew’s Hospital Medical School. John had ambitions to grow this operation into something larger and more influential and he invited some of his old scientific friends and colleagues to join him.

Gus explained how he heard the news in 1988. ‘The phone rang’, he said, ‘and it was John (Vane). He said why not come and join me here. Let’s start again and have some fun!’

Within a couple of years John, together with Gus, Erik Ånggård, Derek Willoughby, David Tomlinson, the author, and (later) Iain MacIntyre (FRS 1996) formed an independent registered medical charity named The William Harvey Research Institute (WHRI) in honour of the eponymous ground-breaking physician/anatomist who had worked at Barts at one point during his career. Gus named his new labs the Department of Pathophysiology, and, together
with some of his former colleagues from King’s College and some new PhD students, he embarked upon a yet another phase of his career (figure 4).

**Research topics 1973–2018**

The programme of work Gus had initiated during his time at the RCS continued to occupy his attention during his subsequent academic career, and space does not permit an account of all these endeavours in this article. Broadly speaking, they comprised further studies on platelet physiology and biochemistry and an extension of his *in vivo* work on atherogenesis and cardiovascular disease. Later, at King’s and in his WHRI laboratory, Gus also branched out into new fields such as parasitology and cancer. His main collaborators during these years included his old friend Peter Richardson (FRS 1986), Peter Görög, Eduardo Cardona-Sanclemente, Shahida Shafi and Rudolfo Medina, and new PhD students including Claire Ludlam and Hayley Farmer (see online Appendix, §3).

**Retirement (2000–2018)**

Gus finally ‘retired’ from the WHRI aged almost 87 years. Even then, he remained active, lecturing and commenting or writing on scientific matters.
A highlight of these latter years came in July 2011, when his colleagues at the WHRI organized a Festschrift in honour of Gus’s 90th birthday (figure 5). It was a nostalgic event and Gus was reunited with many old colleagues, students and co-workers. His own talk, at the end of the symposium, brought a wonderful perspective on his life and work.

Throughout his long and distinguished career Gus had made seminal contributions to our knowledge of platelet physiology, biochemistry and pharmacology, ranging from a study of individual cells to a study of their behaviour in vivo in health and disease. By any measure it was an extraordinary achievement, and this is echoed in the accolades and awards he received (see online Appendix, §4). His death in April 2018 aged 96 was a great loss not only to his immediate circle but also to the wider scientific community. Many friends and colleagues joined the family to pay tribute to him at his funeral on 29 April 2018 and subsequently at a Musical Memorial for him held at St Peter’s College, Oxford, in November that year. Gus was particularly fond of Bach ‘for the great peace that he can bring’ as he once put it, and for his memorial he had selected two of his favourite pieces, including Erbame dich, mein Gott from the St Matthew passion. A peaceful note indeed to mark the end of his extraordinary life.

PERSONALITY AND CHARACTER

The family man and the colleague

Gus was devoted to his family—past and present. He was deeply attached to his parents and sisters and seemed to have an especially strong bond with his father. When Max died in 1970,
Gus was profoundly affected, and he assumed the role of guardian of his father’s reputation. His home was his personal citadel and refuge. The cornerstone of this edifice was his wife Faith, who organized and regulated his personal life, cared for the children from both her own marriage and his previous marriage to Ann, and selflessly supported him throughout his career (at the expense of her own). Gus was hugely proud of his extremely talented children, grandchildren and other close relatives and never lost an opportunity to talk of their achievements (see online Appendix, §§5 and 6).

Throughout his professional life, Gus worked with, and nurtured, many outstanding students and scientists. In one of his more autobiographical papers he lists 38 colleagues who were particularly important in his platelet studies. After his death, many of these former colleagues, co-workers and students wrote to share their recollections and memories. Amongst the most frequent comments were those relating to his kindness, generosity, loyalty and his willingness to share scientific credit (see online Appendix, §7 for these comments in full).

Gus had a number of foibles that were occasionally frustrating, but which were generally a source of wry amusement to his colleagues. Take his sartorial sense, for example: not everyone would feel as comfortable as Gus appeared to be while wearing white trainers with an otherwise sober dark suit.

But it was his love affair with the telephone for which he was best known. No mere caprice this, it was a *grande passion*, and it seemed to many as though telephony was his preferred mode of communication. At scientific meetings, Gus could usually be found hovering near call boxes fossicking in his bag and ransacking his pockets for the appropriate change, or wedged into a conference centre phone booth engaged in a deep conversation. His family, friends and colleagues grew accustomed to receiving unexpected calls from around the world, and, as Gus seemed to have an almost preternatural (albeit unintentional) ability to pick the most inconvenient times, these usually arrived at odd hours in the early morning or late evening.

Such calls did not always originate from distant locations: his technicians, who always arrived in the lab before him, were often summoned to the phone in the secretary’s office to take lengthy instructions for the day’s experiments. Upon hanging up, staff were sometimes disconcerted by the almost immediate appearance of Gus himself, who had apparently rung from a telephone box just around the corner from the lab.

Oddly, when the era of the mobile phone arrived Gus was seemingly uninterested, preferring always the reassuring solidity of the heavy desktop handsets and the enduring assurance of the hard-wired land line (see online Appendix, §8).

**The scientist in society**

Max Born was a highly principled man and a committed pacifist. He was a founder member of Pugwash, an organization committed to the abolition of weapons of mass destruction throughout the world, and he often spoke about the special responsibilities borne by scientists within a society.

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13 On one occasion, he wrote to the novelist and playwright Michael Frayn concerning what he saw was an error in his play ‘Copenhagen’ about the birth of modern physics.

14 One of his nieces, the daughter of Irene Newton-John, was the renowned singer Olivia Newton-John, and he would later joke that during his life he was known first as the son of a famous scientist and then as the uncle of a famous film star.
Gus evidently inherited these concerns and was active in a number of areas, including environmental issues. No doubt recalling his family’s flight from Göttingen during Hitler’s rise to power and his father’s subsequent status as a refugee academic in the UK, the necessity of preserving intellectual freedom, particularly in countries where academics were persecuted for their beliefs, was a topic close to Gus’s heart. He campaigned for, and wrote about, these subjects often and was particularly proud to be associated with CARA (Council for At-Risk Academics). It was this organization, then chaired by Rutherford, that brokered Max Born’s Cambridge lectureship when he fled to the UK. Gus strongly supported this organization. He wrote a paper detailing his family’s own experiences of exile for CARA’s 75th anniversary.

Gus was not a religious man despite his father’s Jewish ancestry or, through his mother’s family, his connection to Martin Luther. He believed passionately in the Enlightenment ideal and the pursuit of the rational. He often commented and wrote on science policy and was an eloquent advocate for curiosity-driven research and the importance of learning, often warning against the rising tide of irrationality, particularly that pernicious brand peddled by religious extremists.

Gus was also concerned about the teaching of science to the young. Quoting Einstein (‘I am not exceptionally clever, only incurably inquisitive’), Gus believed that it was this attitude that characterized the best scientists. He was concerned that it was all too easy for children to become blasé about the technological and medical marvels that are today taken for granted by society and that this could extinguish the feeling of wonder and curiosity. These were qualities which he possessed, and which drove him on throughout his career.

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A list of historical resources I have consulted is available in the online Appendix (§10). Unless otherwise referenced, Gus’s quotes are taken from or from personal letters or conversations.

The portrait was taken in 1986 and is copyright © Godfrey Argent Studio.

AUTHOR PROFILE

Rod Flower was recruited to his first job in science following an interview with Gus Born and John Vane at the Royal College of Surgeons and he remained friends with the two men throughout their lives. He continued working with John Vane for 11 years at the Wellcome Foundation in Kent before moving to the University of Bath to take up the

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15 He was a prominent figure in the ‘Anti-Concorde’ movement in the 1970s founded by Richard Wiggs, which sought to restrict the ‘sonic booms’ caused by these aircraft.
16 ‘Refugee scientists in a new environment’, delivered 21 October 2009. Available at https://www.cara.ngo/professor-gustav-born-frs-1921-2018/.
17 He sometimes wondered if there would have been a scientific revolution if it was not for the Protestant Reformation initiated by Luther, which did much to free the world of religious dogma.
Chair of Pharmacology in 1985. In 1990, he moved to St Bart’s Hospital Medical School and together with John Vane, Gus Born and other colleagues he founded the William Harvey Research Institute, serving as Institute Director from 2000 to 2004. Flower has spent the majority of his scientific career working on mediators of inflammation and anti-inflammatory drug mechanisms but has also worked in the platelet field. Flower also has in an interest in ‘science and security’ issues. He was elected to fellowship of the Academy of Medical Sciences (1999) and the Royal Society (2003), was president of the British Pharmacological Society (2000–2003) and has published over 350 papers and books. He has been awarded several national and international awards for his research.

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