Frederick Akbar Mahomed

Michael F. O'Rourke

Frederick Akbar Mahomed was an Englishman of mixed Indian and Irish descent who made substantial contributions to the study of high blood pressure in a short professional life from 1872 to 1884. He was strongly influenced by the previous work of Richard Bright on kidney disease at his own hospital (Guy's Hospital in London) and by the contemporary pathological studies of Gull and Sutton on arteriolar changes in persons with high blood pressure. In detailed clinical studies, he separated chronic nephritis with secondary hypertension from what we now term essential hypertension. He described the constitutional basis and natural history of essential hypertension and pointed out that this disease could terminate with nephrosclerosis and renal failure. His clinical studies were done without the benefit of a sphygmomanometer but with the aid of a quantitative sphygmogram that he had initially developed while a medical student. He described characteristic features of the pressure pulse in patients with high blood pressure and in persons with arteriosclerosis consequent on aging. These pressure wave changes have recently been verified and explained. He contributed to a number of other advances in medical care, including blood transfusion and appendectomy for appendicitis. He initiated the Collective Investigation Record for the British Medical Association; this organization collected data from physicians practicing outside the hospital setting and was the precursor of modern collaborative clinical trials. Mahomed died from typhoid fever, almost certainly contracted from one of his patients, at age 35 at the height of his career. (Hypertension 1992;19:212–217)

By any reckoning Frederick Akbar Mahomed must be regarded as one of the most important pioneers in the field of arterial hypertension. He was the first to separate clearly chronic glomerulonephritis from essential hypertension, the first to describe the natural history of essential hypertension, and the first to develop a quantitative sphygmogram so that high blood pressure could be recognized during life. He was the first to describe and explain how high blood pressure and arterial degeneration alter the contour of the arterial pulse and how the pulse changes in contour between central and peripheral upper limb arteries in humans. He was the first to initiate the precursor of modern multicenter cooperative trials.

Despite these achievements, history has not treated Mahomed well, and few recognize him today. Credit for the concept of "essential hypertension" (and the other names this condition has been called) is usually given to Huchard (of Paris), von Basch (of Vienna), and Albutt (of London), all of whose contributions were made many years after those of Mahomed.1–4 Some reasons for such neglect emerge from consideration of the man, his instrument, and the atmosphere of medical and scientific practice in the late 19th century.

The Time and the Place

In 1827 Richard Bright of Guy’s Hospital, London, described the association between “dropsy” and kidney disease.5 He described a series of autopsy cases of contracted kidney from patients who during life had evidence of dropsy (probably cardiac failure), hardness of the pulse (high blood pressure), and albuminuria. He attributed the clinical features to renal disease. In 1836, he noted a high incidence of hypertrophy of the left ventricle of the heart and of cerebral hemorrhage at autopsy in addition to renal contraction in a further clinical series.6 He considered the possibility that

either the altered quality of the blood (from chronic renal disease) affords irregular and unwanted stimulus to the organ (the heart) immediately, or that it so affects the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system.

Bright’s ideas were generally accepted, but it was not possible to classify conveniently all patients with dropsy, hardness of the pulse, and left ventricular hypertrophy as having Bright’s disease, since many had no or only intermittent albuminuria, hematuria,
FIGURE 1. Frederick Akbar Mahomed.

or other evidence of renal disease. Prominent in pathological research on the subject were Gull and Sutton of the London Hospital. These two men, a scientifically oriented physician and a pathologist, respectively, had found evidence of "arteriocapillary fibrosis" throughout the body in patients who died at different stages of Bright's disease, as well as in the granular contracted kidney, and they queried the primary role of the kidney in the whole process.7

Gull's remarks, recorded from a lecture given with a pathological specimen at Guy's Hospital in 1872 before a large audience that almost certainly included Mahomed as a medical student, were as follows:

It is always dangerous to rest in a narrow pathology; and I believe that to be a narrow pathology which is satisfied with what you now see before me on this table. In this glass you can see a much hypertrophied heart and a very contracted kidney. The specimen is classical. It was, I believe, put up under Dr Bright's own direction, and with a view of showing that the wasting of the kidney is the cause of thickening of the heart. I cannot but look upon it with veneration, but not with conviction. I think, with all deference to so great an authority, that the systemic capillaries, and, had it been possible, the entire man, should have been included in this vase, together with the heart and the kidneys; then we should have had, I believe, the truer view of the causation of the cardiac hypertrophy and of the disease of the kidney.

The specimen in question was apparently one of Bright's own recorded cases, which has subsequently been reexamined histologically and is now regarded as being a case of essential hypertension with secondary nephrosclerosis.1,8

The Man

Frederick Henry Horatio Akbar Mahomed was a colorful character in many ways (Figure 1). His grandfather was an Indian surgeon, who established a Turkish bathhouse in Brighton and later became "shampoo surgeon" to the Prince of Wales and two English kings. His grandmother was Irish and his father ran a boxing and fencing academy near Brighton. As a child he described himself as restless, excitable, passionate... not remarkable for any brilliancy... with a tendency towards mathematics... fond of mechanical toys and apt in constructing them.

He entered Guy's Hospital from the Sussex County Hospital in 1869 at age 20. As a medical student his career was distinguished, and he won a series of prizes, including the Pupils Physical Society Prize for his work on improving, evaluating, and using a new sphygmogram.9 He took up an appointment at the Central London Sick Asylum and then at the London Fever Hospital, from which he published his second paper on "the pre-albuminuric stage of Bright's disease."10 Here he came under the influence of Sir William Broadbent, who became a strong supporter and friend, who attended Mahomed in his final illness, and whose book on the pulse contains abundant illustrations from his pupil.11 Broadbent was Physician-Extraordinary to Queen Victoria, had a practice of enormous proportions, and was regarded as one of the best clinical teachers of his day.

Mahomed was elected MRCP in 1874 and received an MD from the University of Brussels in the same year. In 1876 his first wife died, leaving him with two children. He yearned to join the staff of his old hospital, and in 1877 was appointed medical registrar at Guy's Hospital. Knowing that a degree from a British university was required for promotion and wanting to advance quickly, he decided to continue his position at Guy's Hospital while traveling by train to Cambridge every second night (so as to satisfy all requirements, including residence, of Cambridge University) and returning to London for work the following morning. He was elected FRCP in 1880 but initially could not afford the entry fee and had to defer. The MB Cambridge was conferred in 1881 for a thesis entitled "Chronic Bright's Disease Without Albuminuria."12 The same year he realized his ambition and was appointed assistant physician at Guy's Hospital. He married his deceased wife's sister in 1881, and they had three more children. He contracted typhoid fever in 1884, probably from one of his patients at Guy's or the London Fever Hospital,
Hypertension  Vol 19, No 2  February 1992

8. On the sphygmographic evidence of arterio-capillary fibrosis.

By F. A. Mahomed, M.D.

The pulse, ranks the first among our guides; no surgeon can despise its counsel, no physician shut his ears to its appeal. Since, then, the information the pulse affords is of so great importance and so often consulted, surely it must be to our advantage to appreciate fully all it tells us, and to draw from it all that it is capable of imparting. Our sense of touch, however highly educated, is manifestly liable to error and it is to our more reliable sense, that of sight, we appeal, when possible, for confirmation. It is by the aid of this more accurate sense we should study the pulse in its marvellous changes of character and form, as recorded by the sphygmograph.

After a description of pulse contour in normotensive subjects during fevers and in heart disease, Mahomed turned his attention to patients with Bright's disease. He wished to quantify the "hardness of the pulse" that is a characteristic feature of this condition and so described by Bright. Using the sphygmogram he determined the characteristic features of the pulse of high tension, which he enumerated as follows:

1. Pressure above 1 ounce and sometimes as high as 10 ounces is employed to develop the pulse-tracing to its greatest extent.
2. The percussion wave is usually well marked and distinctly separated from the tidal.
3. The dicrotic wave is very small and often scarcely perceptible; the vessels, however, are full during the diastolic period, and collapse slowly.
4. The tidal wave is prolonged and too much sustained.

... The most constant of these indications is the prolongation of the tidal wave; any one or all of the other characters may under certain conditions be absent.

Mahomed qualified these remarks by noting that a similar pulse may be seen in arterial degeneration, accompanying age. He noted:

A similar result can be produced experimentally in a schema of the circulation by making the contraction of the ventricle laborious and prolonged, as it would necessarily be under these conditions of high tension. This feature is often indicated in the tracing merely by a slight rounding of the tidal wave, and any such tendency must be looked upon with suspicion; implicit confidence, however, cannot be placed in it, as a similar appearance is produced by degeneration of the arteries, but if due to this cause a very slight pressure is sufficient to extinguish the pulse, while a pulse of high tension generally requires a more considerable one.

Mahomed described such a pulse developing after scarlet fever and preceding onset of hematuria and albuminuria in acute Bright's disease (acute glomerular...
cause in one case may be the result in another; thus, general disorder may cause high arterial pressure and this, in its turn, kidney changes; while on the other hand, kidney changes may be primary and acute and they may in their turn produce impurity of blood and this general pressure. But whether we read the tale backwards or forwards it is the same tale in the end.

Mahomed went on to describe the natural history of this condition:

These persons appear to pass on through life pretty much as others do and generally do not suffer from their high blood pressures, except in their petty ailments upon which it imprint itself ... As age advances the enemy gains access of strength ... the individual has now passed forty years, perhaps fifty years of age, his lungs begin to degenerate, he has a cough in the winter time, but by his pulse you will know him ... Alternatively headache, vertigo, epistaxis, a passing paralysis, a more severe apoplectic seizure, and then the final blow.

One recognizes in this description the asymptomatic patient with gradual development of left ventricular failure, graduating to transient cerebral ischemic episodes, and then a cerebrovascular accident, and eventually death from cardiac failure or myocardial infarction as “the final blow.” Mahomed stressed that such patients could be identified from the character of their radial pulse.

Referring to the work of Gull and Sutton, Mahomed stated:

Of this I feel sure, that the clinical symptoms and the pathological changes resulting from high arterial pressure are frequently seen in cases in which very slight, if any, disease is discoverable in the kidney. The observations provide strong evidence of Gull and Sutton’s work. It appears to me that these clinical, and their pathological, observations must stand or fall together; that one is the pathological, the other the clinical aspect of the same condition.

It is surprising that these clear-cut statements were overlooked by later researchers. Certainly they were presented before the cream of the English profession and published in journals of high repute. His publications were abundant and varied (see “Appendix”). They were not, however, collected into a single monograph appearing under Mahomed’s name, and he never had the authority of the highest professional standing. In seeking a reason one sees a few possible explanations for his work being overlooked. Mahomed personally was considered an overenthusiastic champion of his cause, a man whose views could reasonably be doubted or discounted. His observations depended on use of the sphygmogram, which few were able to use with the same facility; the sphygmogram was overtaken by the cuff sphygmomanometer, which introduced a new order to the field, and Mahomed died early in a junior position and left no acolytes.

Recent work has renewed interest in Mahomed’s sphygmographs.

The disease may commence as an acute infection and afterwards become chronic ... what has been the ulonephritis). He noted a similar pulse with high mean pressure in persons with lead poisoning, gout, after alcoholic bouts, and with other different conditions where the kidney was not directly involved. Gradually he came to doubt the accuracy of the concept attributed to Bright that all such cases of high arterial tension are due to renal disease. His clinical views were swayed by the microscopic pathologic findings on arteriocapillary fibrosis made by Gull.

He recognized the disease as different from that which Bright had described but including many of the cases usually grouped under the heading of Bright’s disease. He pointed out that in almost three quarters of cases exhibiting features of Bright’s disease with high arterial pressure, left ventricular hypertrophy, and dropsy, there was no or only intermittent albuminuria or other evidence of renal disease. He noted that these patients died suddenly (presumably from myocardial infarction, cardiac failure, or cerebrovascular hemorrhage). He was unwilling, however, to introduce a new name lest this “rid Bright of half his glory”; but his description of this condition, its uncertain cause, and its relation to renal disease was contained in unequivocal language.

The disease may commence as an acute infection and afterwards become chronic ... what has been the
FiguRe 4. Pressure recordings taken at short intervals apart with a Millar micromanometer in the ascending aorta (above) and brachial artery (below) under control conditions (left) and after administration of 0.3 mg nitroglycerin (right) sublingually in a middle-aged human with arterial degeneration. Prominent tidal wave in the brachial artery is even more prominent in the ascending aorta and adds almost 20 mm Hg to the pulse amplitude in late systole. Amplitude of this tidal wave is reduced by nitroglycerin. Although this simply alters the contour of the pressure wave in the brachial artery without altering systolic pressure, it causes a drop of almost 20 mm Hg in the ascending aorta. Reproduced from Reference 18.

plain change in contour of the arterial pulse and hypertension (and in arteriosclerosis), with the prominent tidal wave and absent diastolic wave explained on the basis of early wave reflection.14 The studies of Freis et al,15 and our own studies16 on the aging process simply rediscovered what Mahomed had described almost 100 years before (Figures 2–4). Our own work, too, on wave transmission to the upper limb in humans was accurately anticipated by Mahomed’s tracings first published in 1872.

Mahomed’s greatest contribution was in describing what is now known as essential hypertension, noting its natural history and separating this condition from hypertension caused by chronic glomerulonephritis, but he made other major contributions in this and other fields. He described the constitutional predisposition to essential hypertension and its tendency to run in families. At a time when tuberculosis was considered to have a constitutional predisposition, he used multiple photographic portraits to present a contrary opinion. He made a major contribution to the surgical management of appendicitis and was one of the first to use direct blood transfusions in humans. He described a syphilitic causation of thoracic aortic aneurysms. His last contribution was the precursor of modern cooperative clinical trials. He successfully proposed development of a Collective Investigation Record by the British Medical Association to prepare information on morbidity and mortality of diseases seen by physicians practicing outside hospitals. He became the first Secretary of the Record, with an old mentor and friend, Sir George Humphrey, as President, and was enthusiastically pursuing this throughout Britain and in Europe at the time of his premature death.

Frederick Akbar Mahomed was a visionary. His views command fresh appraisal today as his writings are reevaluated, remote from personal and professional prejudices, and in an atmosphere where pulsatile phenomena are gaining new respect. Mahomed’s life is an example of what a youthful mind can achieve when combined with an enthusiastic and indomitable spirit and when opportunities are given by those in high places for a gifted mind and hand to flourish.

Acknowledgments
I acknowledge with thanks the assistance provided by librarians Brenda Heagney from the Royal Australasian College of Physicians, Sydney; Geoffrey Denison from the Royal College of Physicians, London; and Andrew Baster from Guy’s Hospital, London.

Appendix

Bibliography of Frederick Akbar Mahomed

1872 The physiology and clinical use of the sphygmograph. Medical Times Gazette 1: 63, 128, 220, 250, 340, 427, 569 and 2: 72, 143, 324
1873 The physiology and clinical use of the sphygmograph (cont.). Medical Times Gazette 2: 141, 222, 478, 693
1874 The etiology of Bright’s disease and the prealbuminuric stage. Med Chir Trans 57: 197
1875 Case of intestinal obstruction produced by the abnormal remains of a foetal vessel. Trans Pathol Soc 26: 117
A contribution to the clinical history of scarlatinial convalescence. Practitioner 15: 21
1877 On two cases of adenoma hepatis. Trans Pathol Soc 28: 144
Two cases of syphilitic disease of the lungs. Trans Pathol Soc 28: 339
Aneurysm of the aorta in a syphilitic subject. Trans Pathol Soc 28: 344
The sphygmographic evidence of arterio-capillary fibrosis. Trans Pathol Soc 28: 394
On the pathology of uraemia and the so-called uraemic convulsions. Lancet 1: 10, 42, 136
Some indications for the diagnosis and treatment of aortic aneurysm (lecture). London
1879 On chronic Bright’s disease and its essential symptoms. Lancet 2: 46, 76, 149, 261, 399, 437
Some of the clinical aspects of chronic Bright’s disease. Guy’s Hospital Reports 39: 363
1880 Suggestions concerning the scientific work of the Association. Br Med J 1: 30, 74
1881 Remarks on a case with the paradoxical temperatures. Lancet 2: 790
The pathology and aetiology of myxoedema. Lancet 2: 1078
Chronic Bright’s disease without albuminuria. Guy’s Hospital Reports 40: 295
With Sir F. Galton) An enquiry into the
physiognomy of phthisis by the method of composite photography. *Guy's Hospital Reports* 40: 475

1882 Two cases of direct transfusion of blood for hemorrhage in typhoid fever. *Trans Clin Soc Lond* 15: 50


1883 Thrombosis of the pulmonary artery (paravaginal and dermoid cysts). *Trans Pathol Soc* 34: 72

Primary cancer of the undescended testis, cancer of the thymus, vacuolation of liver and kidneys. *Trans Pathol Soc* 34: 182

Case of malignant anthrax (charbon) with anthracoid affection of intestine, stomach and lung. *Trans Pathol Soc* 34: 294


(With C.H. Golding-Bird) Two cases of pulsatile tumour at the root of the neck. *Guy's Hospital Reports* 41: 83


1884 Albuminuria and the symptoms which indicate its gravity. *Guy's Hospital Reports* 42: 201

Obituaries and Other Biographical Notes

1884 *Br Med J* 2: 1099, 1165, 1206, 1261

*Medical Times Gazette* 2: 763

*Goodhart JF, Jacobson WHA: Guy’s Hospital Reports* 43: 1


*Med Mag Lond* 1: 1060


References


6. Bright R: Tabular view of the morbid appearances in 100 cases connected with albuminous urine with observations. *Guy’s Hospital Report* 1846;1:380–400


8. Mann WN: *Guy’s Hospital Report* 1946;95:73


10. Mahomed FA: The etiology of Bright’s disease and the prealbuminuric stage. *Med Chir Trans* 1874;57:197–228


Frederick Akbar Mahomed.
M F O'Rourke

Hypertension. 1992;19:212-217
doi: 10.1161/01.HYP.19.2.212

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1992 American Heart Association, Inc. All rights reserved.
Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/19/2/212

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Hypertension can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Hypertension is online at:
http://hyper.ahajournals.org//subscriptions/