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Alfred Baring Garrod and the beginnings of modern rheumatology

Alfred Baring Garrod e o começo da reumatologia moderna

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Alfred Baring Garrod y los inicios de la reumatología moderna

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Palabras Clave: RELACIÓN MÉDICO-PACIENTE, ENFERMEDADES REUMÁTICAS, REUMATOLOGÍA

"En esta Entrevista con la Historia hablaremos con Alfred B. Garrod, quien hizo importantes contribuciones en el estudio de la gota y la nomenclatura de las enfermedades reumáticas introduciendo el término de artritis reumatoide utilizado en la actualidad, entre otros aportes a la medicina siendo uno de los precursores de la reumatología moderna. "

Alfred Baring Garrod

Alfred Baring Garrod (1819-1907) (Foto 1) nació en Ipswich. Hijo de Robert Garrod, quien era un granjero conocido de la región. Alfred decidió estudiar Medicina y fue aprendiz de Charles Chambers Hammond en el Hospital de su ciudad; posteriormente, se trasladó al University College Hospital donde calificó para M.B en 1842 y M.D en 1843. Se casó en 1845 con Elizabeth Ann, hija de Henry Colchester de Ipswich, con quien tuvo cuatro hijos y dos hijas. Uno de sus hijos fue el también médico Archibald Garrod. (1,2)

Logró convertirse en un profesor y médico reconocido por su importante contribución a nuestro conocimiento sobre las causas de la gota y haber contribuido a redefinir varias enfermedades reumatológicas siendo quien acuñó el término “artritis reumatoide” para la enfermedad que conocemos así en la actualidad.

En estaEntrevista con la Historiaabordaremos su legado y las investigaciones que lo llevaron a ser considerado uno de los padres de la reumatología moderna.

¿Cómo fueron sus inicios?

Comencé mi carrera como asistente clínico en el departamento de química del University College Hospital de Londres a los 28 años. Mis funciones incluían el análisis de fluidos corporales y otras muestras enviadas al laboratorio.

Ya luego, en 1847, fui nombrado médico asistente, seguido en 1851 como Profesor de Terapéutica y Medicina Clínica en el University College Hospital de Londres. Participé activamente en los asuntos del hospital y en el establecimiento del museo de la Materia Médica.

En 1862 dejé el University College Hospital y me uní al King's College Hospital como médico y profesor. Abandoné el trabajo hospitalario activo en 1874, pero continué siendo médico consultor en el mismo hospital. (1,2)

Hablemos de su legado…

Creo que es amplio. Fue precisamente en 1847, estando de médico asistente, cuando hice el descubrimiento de la presencia de ácido úrico en la sangre de pacientes gotosos, presenté los hallazgos en una conferencia el 8 de febrero de 1848, demostrando un incremento del ácido úrico en los pacientes con gota que no se observa en los casos de reumatismo agudo o de la enfermedad de Bright (cómo se denominaba la glomerulonefritis en general) (3,4).

La historia la recuerda así (4):“Se tomaron 1.000 granos de suero para su examen, y se evaporaron hasta la sequedad en capas finas en un baño de agua.

A continuación, se pulverizó y se trató con alcohol rectificado, se hirvió durante unos diez o quince minutos, se volvió a tratar de la misma manera y se conservaron las soluciones de alcohol para su examen. Después de lavarse de nuevo con alcohol, el suero seco se agota mediante agua destilada hirviendo, repitiéndose la operación dos o tres veces y mezclándose las soluciones acuosas. Cuando se evaporó una pequeña cantidad de este fluido con la adición de ácido nítrico, y después se mantuvo sobre el vapor de amoníaco, se dio una clara evidencia de la existencia de ácido úrico por la producción del hermoso tinte púrpura del murexido o púrpura de amoníaco. La solución acuosa se evaporó hasta que se volvió ligeramente espesa y, una vez fría, se aciduló con ácido clorhídrico puro. Al reposar durante algunas horas, se depositaron cristales de ácido úrico, que luego se recogieron, se lavaron con alcohol y se pesaron".

Por otro lado, también logré desarrollar una prueba, el "Thread Test" (o prueba del hilo), un método semicuantitativo para medir ácido úrico en suero u orina. Mostré que había pequeñas cantidades de ácido úrico alrededor del hilo en el suero de personas normales y pacientes con gota y también demostré los depósitos de urato en el cartílago articular de los pacientes con gota (3,4). Y discutí que el ácido úrico se encontraba en personas sanas, como en pacientes con gota y especulè sobre la presencia del ácido úrico en la orina, de los pacientes con gota tofácea crónica y alcancé a plantear el papel de los riñones en la excreción del ácido úrico y otros solutos. (3,4)

De hecho, debo contar que escribí que "el urato de soda depositado puede considerarse como la causa, y no el efecto, de la inflamación gotosa". Esta fue la primera prueba química jamás realizada para diagnosticar una enfermedad reumática (5).

Estos experimentos fueron pasados por alto durante más de medio siglo hasta la publicación de un artículo fundamental de McCarty y Hollander (6), que mostró que los cristales del líquido sinovial de pacientes con gota estaban compuestos de urato monosódico

Y, finalmente, en mi publicación de 1876 describo el uso de la colchicina para el tratamiento de la crisis aguda y el papel de su profilaxis (4) una observación extraordinaria para esa época.

¿Cuál fue su aporte en cuanto a la artritis reumatoide?

Es probable que la artritis reumatoide haya estado presente como enfermedad durante mucho tiempo, ciertamente desde la época de Sydenham (7) , pero se había confundido con términos como gota reumática, reumatismo crónico, reumalgia, reumatismo escorbútico, etc.

Entonces fue cuando escribí sobre el término "artritis reumatoide" en mi cuaderno en el otoño de 1858.

En ese momento pensaba "aunque no estoy dispuesto a agregar más nombres, no puedo evitar expresar el deseo de que se pueda encontrar uno para esta enfermedad, no implica ninguna relación necesaria entre ella y la gota o el reumatismo". Por lo tanto, sugerí que fuera una enfermedad diferente: "Quizás la artritis reumatoide respondería al objeto por el cual el término implica una afección inflamatoria de las articulaciones, no muy diferente al reumatismo (pero) en algunos de sus carácteres difieren materialmente de él".

Fue en 1859 que propuse el nombre de artritis reumatoide para sustituir definitivamente las diversas denominaciones que tenía hasta entonces (gota reumática, reumatismo nudoso y artritis reumática crónica) (4,8) , término que se aceptó en Gran Bretaña y otros países de Europa y con el cual se conoce la enfermedad en la actualidad.

El nombre de “Artritis Reumatoide” que persiste hasta la actualidad es un gran legado aunque no exento de controversia. Rechacé en ese momento el reumatismo crónico de Heberden y la gota reumática de Fuller y elegí el nombre de "artritis reumatoide" para la enfermedad, y proporcioné ilustraciones. Lo dividí en formas agudas, crónicas e irregulares de tipo generalizado y localizado.

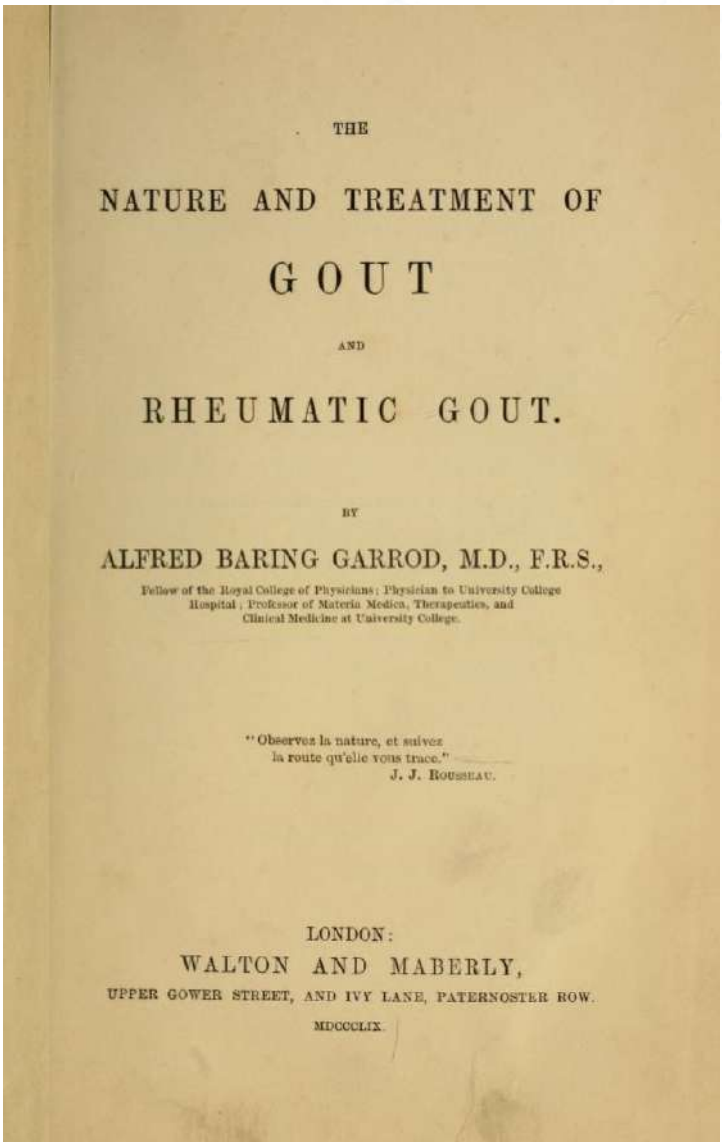
Usted también publicó libros. Háblenos de eso y lo que allí se plantea.

Essentials of Materia Medica and Therapeuticsse publicó en 1855, tuvo 13 ediciones (9) y mi libro clásico,The Nature and Treatment of Gout and Rheumatic Gout, publicado en 1859, en el que diferenció a los pacientes con gota y artritis reumatoide. (10).

En el capítulo XV de mi libro describí con gran precisión la enfermedad que conocemos hoy en día, la diferencié de la gota y la fiebre reumática (ver figuras 2 y 3). Además, detallé en ilustraciones las deformidades características y denoté el carácter grave de la enfermedad respecto a las otras, debido a su difícil control y a su curso clínico incapacitante. Sin embargo, tengo que anotar que, a pesar de la exhaustiva descripción clínica de la AR, no hice referencia al compromiso articular desde el punto de vista histopatológico.

Figuras 2 y 3

Carátula deThe Nature and Treatment of Gout and Rheumatic Gout y cuadro con diagnóstico diferencial entre gota y artritis reumatoide en el capítulo 15 del mismo.



THE

NATURE AND TREATMENT OF

GOUT

AND

RHEUMATIC GOUT.

BY

ALFRED BARING GARROD, M.D., F.R.S.,

Fellow of the Royal College of Physicians; Physician to University College Hospital; Professor of Materia Medica, Therapeutics, and Clinical Medicine at University College.

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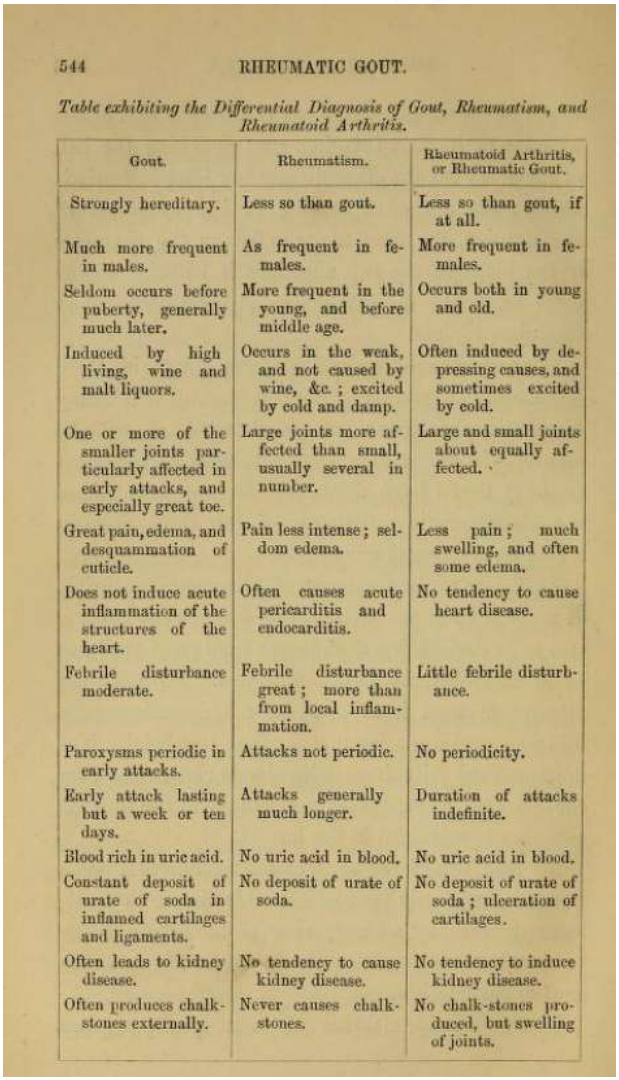

544 RHEUMATIC GOUT.

Table exhibiting the Differential Diagnosis of Gout, Rheumatism, and Rheumatoid Arthritis.

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Strongly hereditary.	Less so than gout.	Less so than gout, if at all.
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Seldom occurs before puberty, generally much later.	More frequent in the young, and before middle age.	Occurs both in young and old.
Induced by high living, wine and malt liquors.	Occurs in the weak, and not caused by wine, &c.; excited by cold and damp.	Often induced by depressing causes, and sometimes excited by cold.
One or more of the smaller joints particularly affected in early attacks, and especially great toe.	Large joints more affected than small, usually several in number.	Large and small joints about equally affected.
Great pain, edema, and desquammation of cuticle.	Pain less intense; seldom edema.	Less pain; much swelling, and often some edema.
Does not induce acute inflammation of the structures of the heart.	Often causes acute pericarditis and endocarditis.	No tendency to cause heart disease.
Febrile disturbance moderate.	Febrile disturbance great; more than from local inflammation.	Little febrile disturbance.
Paroxysms periodic in early attacks.	Attacks not periodic.	No periodicity.
Early attack lasting but a week or ten days.	Attacks generally much longer.	Duration of attacks indefinite.
Blood rich in uric acid.	No uric acid in blood.	No uric acid in blood.
Constant deposit of urate of soda in inflamed cartilages and ligaments.	No deposit of urate of soda.	No deposit of urate of soda; ulceration of cartilages.
Often leads to kidney disease.	No tendency to cause kidney disease.	No tendency to induce kidney disease.
Often produces chalk-stones externally.	Never causes chalk-stones.	No chalk-stones produced, but swelling of joints.

Fuente: *The nature and treatment of gout and rheumatic gout byGarrod, Alfred Baring*, Sir, 1819-1907Tomado de Digitized by the Internet Archive in 2011 with funding from Open Knowledge Commons and Harvard Medical School. Disponible enhttps://archive.org/details/naturetreatmento00garr

En su tratado sobre la artritis hace diez proposiciones. ¿Podríamos hablar de algunas?

- Los niveles altos de ácido úrico están presentes (y son esenciales) antes, durante y entre los ataques de gota.
- Los niveles altos de ácido úrico pueden ser asintomáticos
- Los riñones están involucrados tanto en las primeras etapas de la enfermedad con un defecto funcional específico de incapacidad para excretar ácido úrico como en etapas tardías de la enfermedad con el desarrollo de cambios estructurales. Postuló que la hiperuricemia era el resultado del aumento de la producción o de la incapacidad de los riñones para excretar ácido úrico de manera eficiente.
- Solo en la gota verdadera hay depósito de urato de sodio en los tejidos inflamados.

Además de los ya mencionados, ¿qué otros reconocimientos tiene?

Me convertí en miembro del Royal College of Physicians en 1856 y miembro de la Royal Society en 1858. Dicté las famosas conferencias 'Goulstonian' en 1858 y conferencias 'Lumelian' en 1883. Fui vicepresidente del Royal College of Physicians y serví en 1860 como presidente de la Sociedad Médica de Londres.

En 1887 fui nombrado caballero por la reina Victoria con motivo de mi primer jubileo. Fui el primer destinatario de una medalla fundada en memoria del doctor Moxon por el Real Colegio de Médicos. Además, fui miembro honorario de Berliner Gesellschaft für Innere Medizin, en Berlín, y miembro activo del comité de la Farmacopea Británica.

Y como dato a contarles, existe una calle en Aixles Bains en Londres, que lleva mi nombre (11).

Por todas estas observaciones, descubrimientos y planteamientos de los que dialogamos se me considera como uno de los precursores de la reumatología moderna.

Figuras

Figura 1 :*Alfred B Garrod*. Disponible en : Fotos De Desconocido - [1], Dominio público,https://commons.wikimedia.org/w/index.php?curid=33125820

Figuras 2 y 3
The nature and treatment of gout and rheumatic gout byGarrod, Alfred Baring, Sir, 1819-1907 Tomado de Digitized by the Internet Archive in 2011 with funding from Open Knowledge Commons and Harvard Medical School. Disponible enhttps://archive.org/details/naturetreatmento00garr

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Keywords: DOCTOR-PACIENT RELATIONSHIP, RHEUMATIC DISEASES, RHEUMATOLOGY

"In this Interview with History we will talk with Alfred B. Garrod, a person who made important contributions in the study of gout and the nomenclature of rheumatic diseases. He was the person who introduced the term rheumatoid arthritis used today, among other contributions to medicine. Garrod is one of the precursors of modern rheumatology. "

Alfred Baring Garrod

Alfred Baring Garrod (1819-1907) (Pic. 1) was born in Ipswich. His father, Robert Garrot, was a known farmer in the region. Alfred decided to study Medicine and apprenticed himself to Charles Chambers Hammond at the local Hospital. Later, he moved to University College Hospital where he qualified for M.B in 1842 and M.D in 1843. In 1845 he married Elizabeth Ann, daughter of Henry Colchester of Ipswich, with whom he had four sons and two daughters. One of his sons, Archibald Garrod, was also a doctor. (1,2)

He became a renowned professor and doctor thanks to his important contributions to our knowledge about the causes of gout and to the redefinition of various rheumatological diseases. He was the one who coined the term "rheumatoid arthritis" for the disease which we know as such today.

In this**Interview with History** we will talk about his legacy and the studies that led him to be considered one of the fathers of modern rheumatology.

Tell us about your beginnings

I started my career as a clinical assistant at the Chemistry Department of University College Hospital in London when I was 28 years old. My responsibilities included the analysis of body fluids and other samples send to the laboratory.

Later, in 1847, I was appointed assistant doctor and as professor of Materia Medica and Therapeutics at University College Hospital in London, in 1851. I participated actively in the affairs of the Hospital and in the establishment of the Museum of Medical Knowledge.

In 1862 I left University College Hospital and joined King's College Hospital as a doctor and professor. I left the active hospital work in 1874 but continued as a consultant medical officer at the same hospital. (1,2)

Let's talk about your legacy...

I think it was an extensive legacy. It was indeed in 1846, when, being an assistant doctor, I discovered the presence of uric acid in the blood of patients with gout, I presented the findings at a conference on 8 February 1848, demonstrating an increase of uric acid in patients with gout, that is not observed in patients with acute rheumatism or Bright's disease (As the glomerulonephritis was called then). (3,4)

This is how he recalls the story (4): "1,000 grains of serum were taken for examination and evaporated to dryness in thin layers in a water-bath.

It was then powdered and treated with rectified spirit, boiled for about ten or fifteen minutes, re-treated in the same way, and the spirit solutions preserved for examination. After washing again with spirit, the dried serum was exhausted by boiling distilled water, the operation being repeated two or three times and the watery solutions mixed. When a small quantity of this fluid was evaporated with the addition of nitric acid, and then held over ammonia vapor, distinct evidence of the existence of uric acid was afforded by the production of the beautiful purple tint of murexide or purpurate of ammonia. The watery solution was evaporated until it became slightly thick and, when cool, was acidulated with pure hydrochloric acid. On standing for a few hours, crystals of uric acid were deposited, which were then collected, washed with alcohol and weighed."

On the other hand, I also developed the “Thread Test”, a semiquantitative method to determine uric acid in serum or urine. I demonstrated that there were small quantities of uric acid around the thread in the serum of normal persons and patients with gout and I demonstrated as well urate deposits in the articular cartilage of patients with gout (3,4). And I argued that uric acid was found in healthy persons as well as in patients with gout and suggested the presence of uric acid in the urine of patients with chronic tophaceous gout and raised the issue of the role of the kidneys in the excretion of uric acid and other solutes (3,4).

Indeed, I have to say that I wrote that “deposited urate of soda may be looked upon as the cause and not the effect, of the gouty inflammation”. This was the first chemical test ever performed to diagnose a rheumatic disease (5).

These experiments were overlooked for more than half a century until the publication of a seminal paper by McCarty and Hollander (6), which showed that crystals in the synovial fluid of patients with gout were composed of monosodium urate.

And finally, in my 1876 publication I describe the use of colchicine for the treatment of acute crisis and the role of prophylaxis (4), an extraordinary observation for that time.

What was your contribution as regards rheumatoid arthritis?

It is likely that rheumatoid arthritis had been present as a disease for a long time, certainly since the time of Sydenham (7), but the nomenclature had been confused with terms like rheumatic gout, chronic rheumatism, rheumalgia, scorbutic rheumatism, etc.

It was then when I wrote about the term “rheumatoid arthritis” in my notebook in the autumn of 1858.

At that time, I thought "although I am unwilling to add any more names, I cannot help to express the wish of finding one for this disease, it implies no necessary relation between it and gout or rheumatism". I therefore suggested that it was a different disease: "Perhaps rheumatoid arthritis would answer the object by which the term I imply an inflammatory affection of the joints, not unlike rheumatism (but) in some of its characters differing materially from it"

It was in 1859 when I proposed the name of arthritis rheumatoid to categorize it as a different condition and definitively replace the various names it had until then (rheumatic gout, knotty rheumatism, and chronic rheumatic arthritis) (4,8). The term was accepted in Great Britain and other European countries, and it has remained ever since.

The name "Rheumatoid Arthritis" is a great legacy, although not without some controversies. I rejected the chronic rheumatism of Heberden and the rheumatic gout of Fuller and chose the name "arthritis rheumatoid" for the disease and provided illustrations. I divided it into acute, chronic, and irregular forms of generalized and localized type.

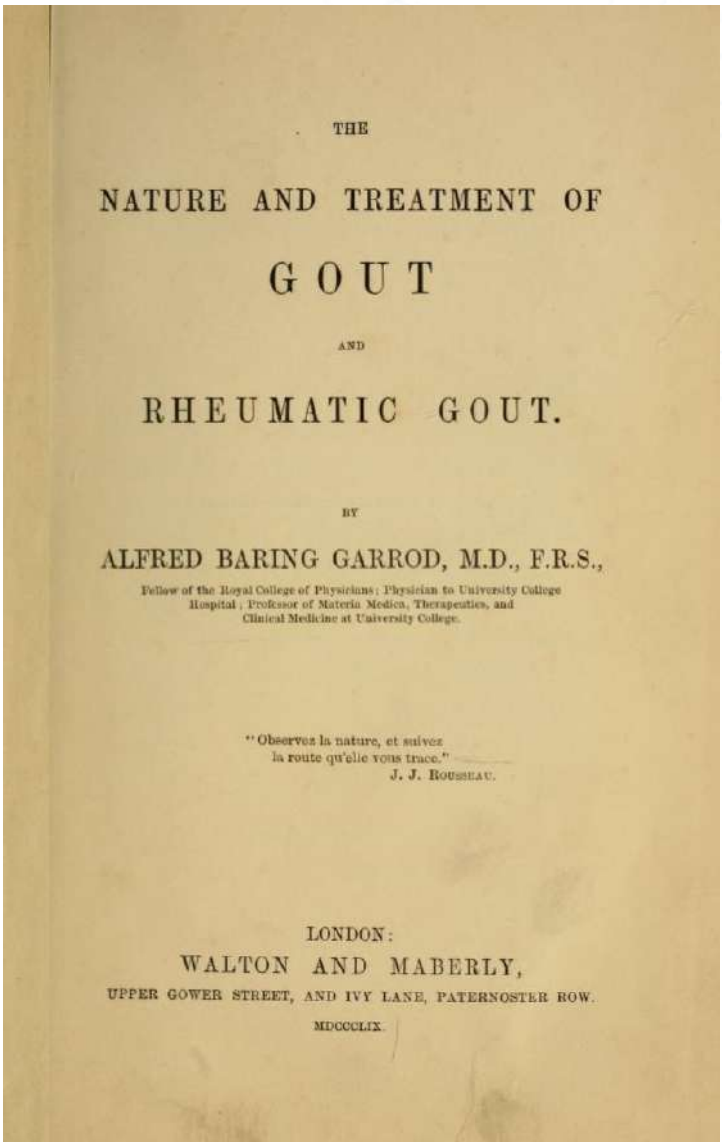
You also published books. Tell us about that and what is raised there

Essentials of Materia Medica and Therapeutics was published in 1855 and went through 13 editions (9), and my classic, The Nature and Treatment of Gout and Rheumatic Gout, published in 1859, in which I differentiate patients with gout and patients with arthritis rheumatoid.(10)

In Chapter XV of my book, I described with great precision the disease we know today, delineating it from gout and rheumatic fever (see Figures 2 and 3). In addition, I illustrated the characteristic deformities and denoted the serious character of the disease with respect to the others, due to its difficult control and disabling clinical course. However, I must note that, despite the exhaustive clinical description of RA, I did not refer to joint involvement from the histopathological point of view.

Figures 2 and 3

*Cover sheet of The Nature and Treatment of Gout and Rheumatic Gout*and table showing the differential diagnosis between gout and rheumatoid arthritis included in Chapter XV of the book.



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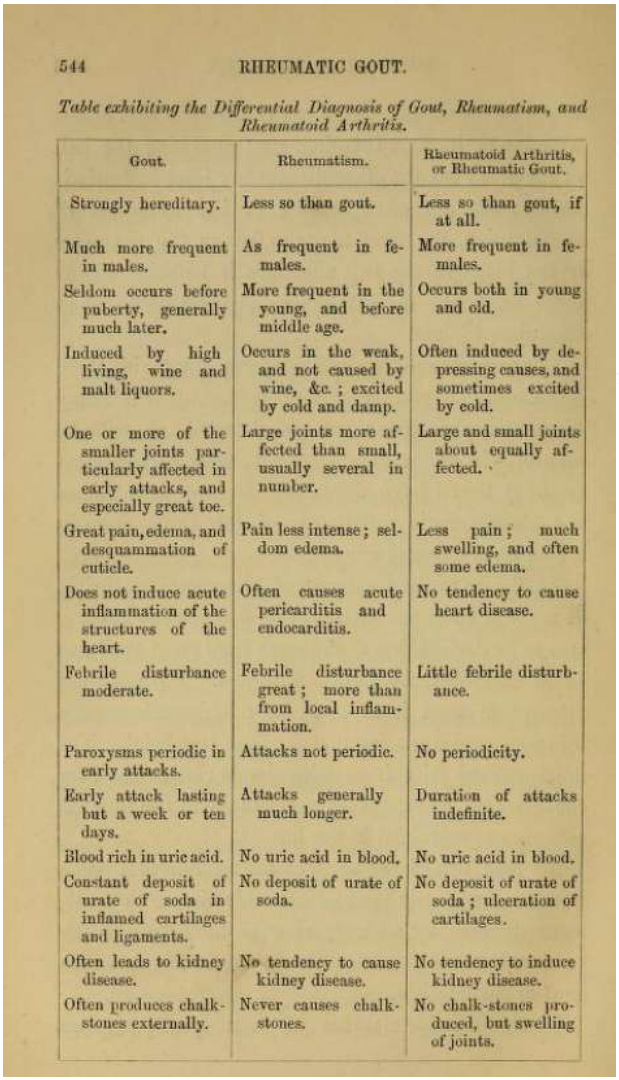

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Often leads to kidney disease.	No tendency to cause kidney disease.	No tendency to induce kidney disease.
Often produces chalk-stones externally.	Never causes chalk-stones.	No chalk-stones produced, but swelling of joints.

Source: The nature and treatment of gout and rheumatic gout byGarrod, Alfred Baring, Sir, 1819-1907Tomado de Digitized by the Internet Archive in 2011 with funding from Open Knowledge Commons and Harvard Medical School. Available athttps://archive.org/details/naturetreatmento00garr

In your treatise on arthritis, you make ten propositions. Could we talk about some of them?

- High uric acid levels are present (and essential) before, during and between attacks of gout.
- High uric acid levels can be asymptomatic.
- Kidneys are involved in both, the early stages of the disease with a specific functional defect of inability to excrete uric acid and late in the disease with the development of structural changes. I proposed that hyperuricemia was the result of either increased production or inability of kidneys to excrete uric acid efficiently.
- Only in the gout there is deposition of urate of soda in the inflamed tissues.

In addition to those already mentioned, what other awards do you have?

I became a fellow of the Royal College of Physicians in 1856 and a fellow of the Royal Society in 1858. I delivered the famous "Goulstonian" Lectures in 1858 and "Lumenian" Lectures in 1883. I was Vice-President of the Royal College of Physicians and served in 1860 as President of Medical Society in London.

In 1887, I was knighted by Queen Victoria on the occasion of her first Jubilee. I was the firs recipient of a medal founded in memory of Dr. Moxon by the Royal College of Physicians. I was also an honorary member of Berliner Gesellschaft für Innere Medizin in Berlin and an active member of British Pharmacopoeia Committee.

And as a curious fact, a street has been named after me in Aix les Bains in London (11).

Thanks to all the studies, discoveries and statements we discussed, I am considered one of the precursors of modern rheumatology.

Figures

Figure 1

Alfred B Garrod.Available at: Unknown author- [1], Public domainhttps://commons.wikimedia.org/w/index.php?curid=33125820

Figures 2 and 3
The nature and treatment of gout and rheumatic gout byGarrod, Alfred Baring, Sir, 1819-1907 Retrieved fromDigitized by theInternet Archive in 2011 with funding from Open Knowledge Commons and Harvard Medical School. Available athttps://archive.org/details/naturetreatmento00garr

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Alfred Baring Garrod y los inicios de la reumatología moderna

ARTIGOS E REPORTAGENS ESPECIAIS

Alfred Baring Garrod e o começo da reumatologia moderna

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Palavras chaves: RELAÇÃO MÉDICO-PACIENTE, ENFERMEDADES REUMÁTICAS, REUMATOLOGÍA

"Nesta Entrevista con la Historia falaremos com o Alfred B. Garrod, que fez importantes contribuições para o estudo da gota e da nomenclatura das doenças reumáticas, introduzindo o termo artrite reumatóide hoje utilizado, entre outras contribuições para a medicina, sendo um dos precursores da reumatologia moderna."

Alfred Baring Garrod

O Alfred Baring Garrod (1819-1907) (Foto 1) nasceu em Ipswich. Filho do Robert Garrod, um conhecido fazendeiro da região. O Alfred decidiu estudar Medicina e foi aprendiz do Charles Chambers Hammond no Hospital da sua cidade; posteriormente, ele se mudou para o University College Hospital, onde se qualificou para M.B em 1842 e M.D em 1843. Em 1845 ele se casou com Elizabeth Ann, filha do Henry Colchester de Ipswich, com quem teve quatro filhos e duas filhas. Um dos seus filhos era o Archibald Garrod, também médico.(1,2)

Tornou-se professor e médico reconhecido pela sua importante contribuição para o nosso conhecimento das causas da gota e por ter contribuído para redefinir várias doenças reumatológicas, sendo quem cunhou o termo "artrite reumatóide" para a doença que conhecemos hoje.

Nesta Entrevista con la Historia abordaremos o seu legado e as pesquisas que o levaram a ser considerado um dos pais da reumatologia moderna.

Como foi o seu começo?

Comecei a minha carreira como assistente clínico no departamento de química do University College Hospital, em Londres, aos 28 anos. As minhas funções incluíam a análise de fluidos corporais e outras amostras enviadas para o laboratório.

Mais tarde, em 1847, fui nomeado médico assistente, seguido em 1851 como Professor de Terapêutica e Medicina Clínica no University College Hospital, em Londres. Estive ativamente envolvido nos assuntos do hospital e no estabelecimento do museu da Matéria Médica.

Em 1862, deixei o University College Hospital e ingressei no King's College Hospital como médico e professor. Deixei o trabalho ativo do hospital em 1874, mas continuei como médico consultor no mesmo hospital.(1,2)

Vamos falar sobre o seu legado ...

Eu acho que é amplo. Foi justamente em 1847, como médico assistente, quando fiz a descoberta da presença do ácido úrico no sangue de pacientes gotosos, e apresentei os achados em uma conferência no dia 8 de fevereiro de 1848, demonstrando um aumento do ácido úrico nos pacientes com gota que não é observada no reumatismo agudo ou na doença de Bright (como era geralmente chamada de glomerulonefrite)(3,4).

A história o lembra assim(4): "Foram retirados 1.000 grãos de soro para o exame e foram evaporados à secura em camadas finas em banho-maria.

Posteriormente, foi pulverizado e tratado com álcool retificado, fervido por cerca de dez a quinze minutos, tratado novamente da mesma maneira, e as soluções de álcool foram preservadas para o exame. Depois de ser novamente lavado com álcool, o soro seco é drenado por água destilada fervendo, sendo a operação repetida duas ou três vezes e misturando as soluções aquosas. Quando uma pequena quantidade deste fluido foi evaporada com a adição de ácido nítrico, e então mantida no vapor de amônia, a evidência clara da existência do ácido úrico foi dada pela produção do belo corante púrpura de murexide ou púrpura de amônia. A solução aquosa foi evaporada até ficar ligeiramente espessa e, uma vez fria, acidificada com ácido clorídrico puro. Em repouso por algumas horas, cristais de ácido úrico foram depositados, os quais foram coletados, lavados com álcool e pesados.

Por outro lado, também consegui desenvolver um teste, o "Thread Test", um método semiquantitativo para medir o ácido úrico no soro ou na urina. Eu mostrei que havia pequenas quantidades de ácido úrico ao redor do fio no soro de pessoas normais e pacientes com gota e também demonstrei depósitos de urato na cartilagem articular dos pacientes com gota(3,4). E comentei que o ácido úrico foi encontrado em pessoas saudáveis, como em pacientes com gota e espéculos na presença de ácido úrico na urina, em pacientes com gota tofácea crônica e consegui levantar o papel dos rins na excreção de ácido úrico e outros solutos. (3,4)

De fato, devo dizer que escrevi que “o soda urato depositado pode ser considerado a causa, e não o efeito, da inflamação gotosa”. Este foi o primeiro teste químico realizado para diagnosticar doenças reumáticas(5).

Estes experimentos foram negligenciados por mais de meio século, até a publicação de um artigo seminal do McCarty e Hollander(6), que mostrou que os cristais no líquido sinovial dos pacientes com gota eram compostos de urato monossódico.

E, por fim, na minha publicação de 1876, descrevo o uso da colchicina para o tratamento das crises agudas e o papel da sua profilaxia(4), observação extraordinária para a época.

Qual foi a sua contribuição em relação à artrite reumatóide?

A artrite reumatóide provavelmente está presente como doença há muito tempo, certamente desde a época de Sydenham(7), mas tinha sido confundida com termos como gota, reumatismo crônico, reumalgia, reumatismo escorbútico, etc.

Foi então quando escrevi sobre o termo "artrite reumatóide" no meu caderno no outono de 1858.

Na época eu estava pensando "embora não esteja disposto a adicionar mais nomes, não posso deixar de expressar o desejo de que um pudesse ser encontrado para esta doença, não implica qualquer relação necessária entre ela e gota ou reumatismo. " Portanto, sugeri que era uma doença diferente: "Talvez a artrite reumatóide respondesse ao objeto pelo qual o termo implica uma condição inflamatória das articulações, não muito diferente do reumatismo (mas) em algumas das suas características eles diferem materialmente "

Foi em 1859 que propus o nome de artrite reumatoide para substituir definitivamente os vários nomes que tinha até então (gota reumática, reumatismo nodoso e artrite reumática crônica) (4,8), termo que foi aceito na Grã-Bretanha e em outros países europeus e com que a doença é conhecida hoje.

O nome "Artrite Reumatóide" que persiste até hoje é um ótimo legado, embora não sem controvérsia. Rejeitei o reumatismo crônico de Heberden e a gota de Fuller naquela época e escolhi o nome "artrite reumatóide" para a doença e forneci ilustrações. Eu o dividi em formas agudas, crônicas e irregulares do tipo generalizado e localizado.

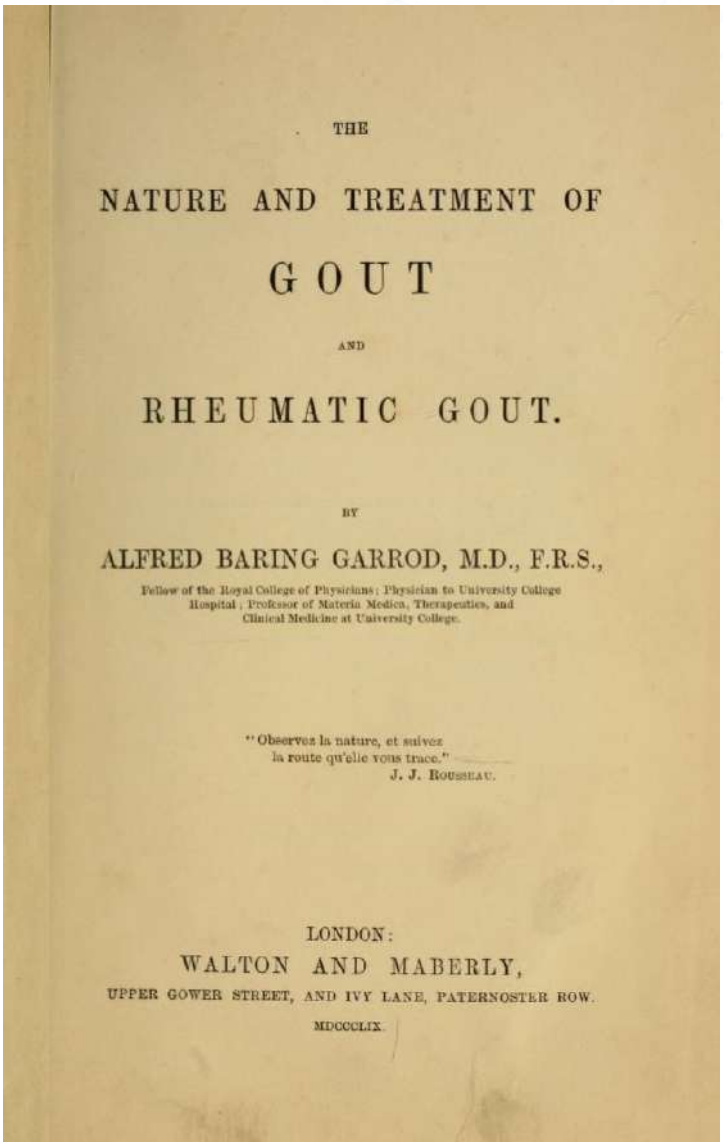
Você também publicou livros. Conte-nos sobre isso e o que está neles.

Essentials of Materia Medica and Therapeutics foi publicado em 1855, teve 13 edições(9) e meu livro clássico, The Nature and Treatment of Gout and Rheumatic Gout, publicado em 1859, no qual ele diferenciava pacientes com gota e artrite reumatóide.(10).

No capítulo XV do meu livro, descrevi com grande precisão a doença que conhecemos hoje, diferenciei-a da gota e da febre reumática (ver Figuras 2 e 3). Além disso, detalhei nas ilustrações as deformidades características e denotei a gravidade da doença em relação às demais, devido ao seu difícil controle e curso clínico incapacitante. No entanto, devo observar que, apesar da descrição clínica exaustiva da AR, não me referi ao envolvimento articular do ponto de vista histopatológico.

Figuras 2 e 3

Capa da*The Nature and Treatment of Gout and Rheumatic Gout* e tabela com diagnóstico diferencial entre a gota e a artrite reumatóide no Capítulo 15 do mesmo.



THE

NATURE AND TREATMENT OF GOUT

AND

RHEUMATIC GOUT.

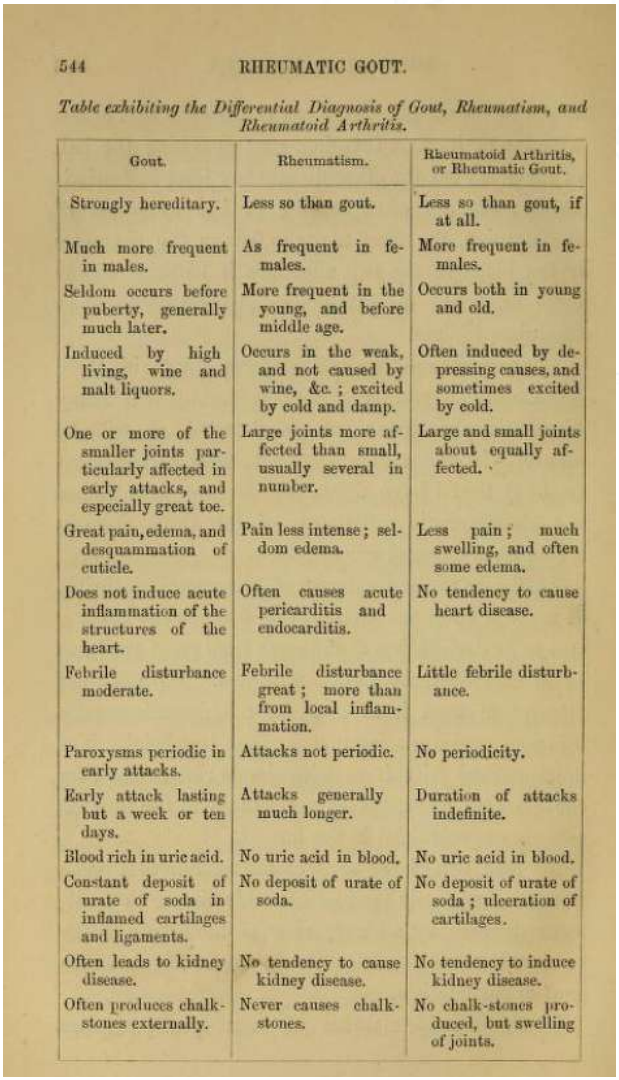
BY

ALFRED BARING GARROD, M.D., F.R.S.,

Fellow of the Royal College of Physicians; Physician to University College Hospital; Professor of Materia Medica, Therapeutics, and Clinical Medicine at University College.

"Observez la nature, et suivez
la route qu'elle vous trace."
J. J. ROUSSEAU.

LONDON:
WALTON AND MABERLY,
UPPER GOWER STREET, AND IVY LANE, PATERNOSTER ROW.
MDCCCLIX.



544 RHEUMATIC GOUT.

Table exhibiting the Differential Diagnosis of Gout, Rheumatism, and Rheumatoid Arthritis.

Gout.	Rheumatism.	Rheumatoid Arthritis, or Rheumatic Gout.
Strongly hereditary.	Less so than gout.	Less so than gout, if at all.
Much more frequent in males.	As frequent in females.	More frequent in females.
Seldom occurs before puberty, generally much later.	More frequent in the young, and before middle age.	Occurs both in young and old.
Induced by high living, wine and malt liquors.	Occurs in the weak, and not caused by wine, &c.; excited by cold and damp.	Often induced by depressing causes, and sometimes excited by cold.
One or more of the smaller joints particularly affected in early attacks, and especially great toe.	Large joints more affected than small, usually several in number.	Large and small joints about equally affected.
Great pain, edema, and desquammation of cuticle.	Pain less intense; seldom edema.	Less pain; much swelling, and often some edema.
Does not induce acute inflammation of the structures of the heart.	Often causes acute pericarditis and endocarditis.	No tendency to cause heart disease.
Febrile disturbance moderate.	Febrile disturbance great; more than from local inflammation.	Little febrile disturbance.
Paroxysms periodic in early attacks.	Attacks not periodic.	No periodicity.
Early attack lasting but a week or ten days.	Attacks generally much longer.	Duration of attacks indefinite.
Blood rich in uric acid.	No uric acid in blood.	No uric acid in blood.
Constant deposit of urate of soda in inflamed cartilages and ligaments.	No deposit of urate of soda.	No deposit of urate of soda; ulceration of cartilages.
Often leads to kidney disease.	No tendency to cause kidney disease.	No tendency to induce kidney disease.
Often produces chalk-stones externally.	Never causes chalk-stones.	No chalk-stones produced, but swelling of joints.

Fonte: *The nature and treatment of gout and rheumatic gout by Garrod, Alfred Baring*, Sir, 1819-1907 Tomado de Digitized by the Internet Archive in 2011 with funding from Open Knowledge Commons and Harvard Medical School. Disponível em https://archive.org/details/naturetreatmento00garr

No seu tratado sobre artrite, você faz dez proposições. Podemos conversar sobre algumas?

- Níveis elevados de ácido úrico estão presentes (e são essenciais) antes, durante e entre os ataques de gota.
- Níveis elevados de ácido úrico podem ser assintomáticos
- Os rins estão envolvidos tanto nos estágios iniciais da doença com um defeito funcional específico de incapacidade de excretar ácido úrico quanto nos estágios finais da doença com o desenvolvimento de alterações estruturais. Ele postulou que a hiperuricemia era o resultado do aumento da produção ou da incapacidade dos rins de excretar ácido úrico com eficiência.
- Somente na gota verdadeira há deposição de urato de sódio nos tecidos inflamados.

Além dos já citados, que outros reconhecimentos você possui?

Tornei-me membro do Royal College of Physicians em 1856 e membro da Royal Society em 1858. Dei as famosas palestras 'Goulstonianas' em 1858 e as palestras 'Lumelianas' em 1883. Fui vice-presidente do Royal College of Physicians e servi em 1860 como presidente da London Medical Society.

Em 1887, fui nomeado cavaleiro pela Rainha Vitória por ocasião do meu primeiro jubileu. Fui o primeiro a receber uma medalha fundada em memória do Dr. Moxon pelo Royal College of Physicians. Além disso, fui membro honorário da Berliner Gesellschaft für Innere Medizin, em Berlim, e membro ativo do comitê da Farmacopeia Britânica.

E adicionalmente, há uma rua em Aixles Bains, em Londres, que leva o meu nome (11).

Por todas estas observações, descobertas e abordagens que discutimos, sou considerado um dos precursores da reumatologia moderna.

Figuras

Figura 1 :*Alfred B Garrod. Disponível em* : Fotos De Desconocido - [1], Dominio público,https://commons.wikimedia.org/w/index.php?curid=33125820

Figuras 2 e 3
The nature and treatment of gout and rheumatic gout by Garrod, Alfred Baring, Sir, 1819-1907 Tomado da Digitized by the Internet Archive in 2011 with funding from Open Knowledge Commons and Harvard Medical School. Disponível em https://archive.org/details/naturetreatmento00garr

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