Syphilis : introductory address to the discussion on syphilis at the Australasian Medical Congress / H.B. Allen.

## Contributors

Allen, Harry Brookes, 1854-1926. Royal College of Surgeons of England

### **Publication/Creation**

[Melbourne] : Stillwell, printers, 1909.

### **Persistent URL**

https://wellcomecollection.org/works/q4ue622y

### Provider

Royal College of Surgeons

### License and attribution

This material has been provided by This material has been provided by The Royal College of Surgeons of England. The original may be consulted at The Royal College of Surgeons of England. where the originals may be consulted. Conditions of use: it is possible this item is protected by copyright and/or related rights. You are free to use this item in any way that is permitted by the copyright and related rights legislation that applies to your use. For other uses you need to obtain permission from the rights-holder(s).



Wellcome Collection 183 Euston Road London NW1 2BE UK T +44 (0)20 7611 8722 E library@wellcomecollection.org https://wellcomecollection.org Reprinted from the "Intercolonial Medical Journal of Australasia," RE OF SURBI

March 1909.

INTRODUCTORY ADDRESS TO THE DISCUSSI AT THE AUSTRALASIAN MEDICAL CO

# PROFESSOR H. B. ALLEN, M.D.,

President of the Congress.

The request that I should open this debate with a short address enables me to bring before you the conclusions that have been gradually forced upon me by experience during the past 32 years in the pathological department of the Melbourne Hospital, and in the pathological museum and laboratories of the University of Melbourne.

In the first place, the time-honoured diagnostics of syphilis leave much to be desired. The histories given by patients cannot be trusted. The common entry in case-books, " No history of Syphilis," is almost worthless. In post-mortem practice, eruptions, nodes, changes in the teeth and external lesions of the eyes are infrequent and of relatively little value. The great marks of syphilis are in the arterial walls, to which the spirochætes soon betake themselves in experimental syphilis. No other cause than syphilis can, in my opinion, account for the precocious forms of atheroma, when delicate linear opacities appear in the descending aorta at about the age of fifteen, and change from white to yellow during the next decade, or when small abruptly prominent rounded patches arise in the ascending aorta between twenty and thirty, and soon pass into the yellow stage. Apart from atheroma præcox, which belongs mainly to inherited syphilis, all cases of severe ulcerous atheroma must be attributed to tertiary syphilis; and also, at the other extreme, the slow atrophic form with closely set wrinkling of the surface, this

wrinkled atrophy being most common after middle age, but sometimes presenting itself in adolescents with inherited disease. Large thick glossy plates near the aortic valves, or widespread thickening of all the coats are always associated with other signs of syphilis. Marked atheroma in the arch in persons with low blood tension and small left ventricle is very suggestive. A strong tendency to relapse should be regarded as suspicious, for example, when calcified plates and atrophic wrinkles are found side by side with yellow patches of varying age, and with recent white thickenings, and possibly with ulcers, the intima being often stained by hæmorrhages and pigmentation from rupture of incoming vasa vasorum. The microscope shows how widespread is the thickening of the coats of the small arteries, and evidence to the same effect is given by the deep aspect of the calvarium, the larger meningeal arteries ploughing deep furrows in the bone, while the arterioles down to their smallest branches chase the surface with delicate engraving. Next in diagnostic value to the vascular phenomena, I would place a widespread fibrosis of organs, the smooth pliancy of liver and spleen and kidneys being very notable, implying a more or less profound alteration in general metabolism, a diminished resistance against the external causes of disease, and a tendency to untoward termination of morbid processes. In addition, careful attention should be given to the serous coats of heart, lungs, liver, spleen, &c., not merely with reference to gross thickening or adhesion, but even more particularly with regard to linear opacities or white patches, with branching lines running from them. Any pathologist who will systematically record the changes in the aorta, and particularly the descending aorta, the incidence of fibrosis, the markings in the serous capsules, and particularly the coexistence of these changes, will find material for serious consideration. Using such criteria, I analysed, in 1904, the records of a hundred consecutive autopsies performed and recorded by myself in the Melbourne Hospital; 34 showed clear signs of syphilis, 19 others showed doubtful signs, and I was open to suspicion (Intercolonial Medical Journal of Australasia, 20th September, 1904). For this Congress, I have again analysed a hundred consecutive cases, and find 32 syphilitic, and 30 more or less doubtful. There is no purposed selection of cases; but I examine only a fraction of the cases

dying in hospital, a fraction not truly representative, and the hospital population itself does not represent the average of the community. Hence, my results cannot be applied to the whole body of in-patients in the hospital, and still less to the community as a whole. But, whatever weight is given to my criteria, these results are of momentous significance.

If we turn to questions interesting some of the Sections of Congress, I have before the Section of Pathology set forth some definite conclusions concerning the circulatory system. Firstly, aortic aneurism is in the great majority of cases due to syphilis, and aneurism is very prevalent in Victoria, and very markedly prevalent among women, so that in 1906 there were 31 deaths recorded from aneurism in women, as against 57 deaths in men. Secondly, aortic regurgitation is mainly due to syphilis, either through chronic endocarditis or atheroma, or a combination of these conditions. Thirdly, a very large proportion of cases of mitral obstruction must be attributed to syphilis. Fourthly, syphilis frequently induces thickening of the fibrous rings at the bases of the valves. Fifthly, subject to unimportant exceptions, progressive chronic myocarditis is a syphilitic affection. Sixthly, syphilis is a potent cause of coronary atheroma, and thus contributes to the gravity of many cases of angina, or leads to myomalacia and rupture of the heart; while syphilitic arteritis at the mouths of the coronary arteries often causes cardiac ischæmia, claudication, and syncope.

Apart from dust disease and chronic tubercle, syphilis is the great cause of interstitial pneumonia, and it often implants a special character on the lesions of chronic tubercle. The anæmia of syphilis is well known, and the saying that mercury is the iron of syphilis epitomizes a world of wisdom; but it is not sufficiently recognised that even the grave forms of pernicious anæmia may be essentially syphilitic. The multiple fibroses of serous membranes are often ascribed to tubercle, but, in my experience, syphilis is by far the most frequent cause. Repeated cases of pancreatic diabetes have come under my observation in which diffuse syphilitic fibrosis has been demonstrated, or a more local syphilitic process with cicatricial atrophy. Mott unhesitatingly pronounces general paralysis and tabes dorsalis to be syphilitic affections, and increasing knowledge tends to show that a very large proportion of the more obscure affections of the nervous system have parasyphilis as one important factor.

Surgeons have constantly to remember the possibilities of syphilitic arteritis and thrombosis, and not only in senile gangrene, but also in the necroses that occur in young people, and also in Raynaud's disease, which is often partly the effect of cold on tissue badly supplied by syphilitic arteries; and in thrombosis of the superior mesenteric artery with consequent hæmorrhage and peritonitis. The so-called malignant tumours that spontaneously disappear are usually syphilitic, whether in the abdominal wall or the liver or elsewhere; and, on the other hand, a chronic syphilitic inflammation is often the forerunner of true maligant growth, of sarcoma in the testis, of carcinoma in the breast. An excised ulcer of the stomach sometimes proves to be syphilitic, but a corner of it may be already carcinomatous. In the tongue and in the larynx, mistakes may easily occur in dealing with the irregular forms of syphilis and epithelioma, and syphilis may give a special character to epithelial growths. The diagnosis of gumma of the brain from glioma, and of both from gummatous conditions of the meninges, must sometimes be of extreme difficulty.

For the obstetrician, the questions to which syphilis gives rise are of profound importance. What are the signs of syphilis in the umbilical cord and placenta? In the cord, the walls of the blood vessels are thickened and sclerosed, so that after a short stay in formalin they feel like hard cords against the finger ; and, in the fresh section, white fibrillar tissue can be seen spreading from the thickened vessels into the jelly of Wharton. The two arteries are constantly affected, though not always equally, but the vein may share fully in the process. The thickening may be most marked near the placenta, or at a distance from it. In the placenta itself there may be opaque white patches in the membrane covering the free surface, and beneath these markings there may be fibrosis extending into the placental substance, perhaps surrounding some sclerosed main branches of the blood vessels. In the early stages of placental growth, thickened vessels, local thrombosis and patchy hæmorrhage may fill the picture. White fibrosis may become general at an early stage,

with typical small hard white placenta; or it may set in later and in more patchy fashion, and be complicated by the scars of old hæmorrhage. In some of the worst cases, calcareous infiltration may be in progress near the attached surface. Not infrequently, definite gummata are found, and these may undergo mucoid degeneration at their centres. What then is the duty of the obstetrician who finds such conditions? Or, apart from examination of the afterbirth, when a woman after repeated miscarriages bears a syphilitic child, what is the duty of the attendant? The mother is always infected, though she shows no outward sign of the disease. This is certain. She can suckle her child with impunity, whereas it would be criminal to give that child to a clean wet-nurse. Spironemata have been found long afterwards in the inguinal glands of such a mother; she may after a subsequent marriage bear infected children to a clean husband; she may late in life develop gummata and other typical tertiary lesions. Is it not the duty of the accoucheur at once to treat father, mother and child? It has recently been suggested that the separation of the placenta marks the time of infection of the child, and that the six weeks following birth are the time of incubation ; but babies that die at birth or soon afterwards have already typical fibrosis in their organs. It has also been alleged that paternal conceptional syphilis is impossible, because the spironema is too large to be carried in the head of the spermatozoon. The neck of the spermatozoon is as important as the head. Colles' law seems to demand our belief in primary paternal infection of the ovum, and it is quite possible that the spironema is only one stage in a complicated life cycle of the parasite. It has even been suggested that the spironema is an involution form of a spore-bearing bacillus.

The gynæcologist must reckon with the rapid destruction wrought by gummata in the cervix and upper part of the vagina, and with the slower forms of ulceration and fibrosis—with forms too rapid for cancer, with forms too indolent, forms destroying the cervix and the vesicovaginal septum in a few weeks, forms persisting for months and years without corresponding alteration in general health. Chronic metritis, chronic salpingitis, chronic mastitis, in all of these syphilis may play a part, and may be the essential factor. In children with inherited syphilis, the ovaries may show pliant fibrosis and the commencement of cystic change.

From the standpoint of public health, it is distressing to contemplate the loss of life that inherited syphilis entails by miscarriage and by early marasmus. I am convinced that the main mortality of children from slight causes is due to inherited syphilis. In deaths from slight shock or slight injury, from administration of anæsthetics for some trifling condition, from mild diphtheria or typhoid or influenza, post-mortem examination reveals the typical smooth fibrosis of liver, spleen and kidnevs, the increased framework of the lungs, the dilated heart, and after fifteen years of age precocious atheroma will often be added. Compare the accounts of lymphatism with the picture of inherited syphilis. Think of the white pneumonia, the cirrhosis of the liver of childhood. Then remember that a large proportion of those with minor stigmata do not die early. What is the after history of their fibroid organs, their thickened arteries, their overworked hearts? To what extent may the enormous prevalence of fibrosis of organs and sclerosis of arteries in later life be attributed at least in part to the evolution of these early changes? The problem is disquieting, the answer difficult.

Such considerations lead me to believe that the prevalent marriage law for syphilitics is not sufficiently stringent, nor administered with adequate firmness. Time will mermit me to do more than quote Marshall's recent summary of the development of expert opinion in this respect. "In 1854, Diday proposed a period of 'two years at least' without symptoms before marriage (this period of two years is still regarded by Mr. Hutchinson as sufficient). In 1890, Fournier (Syphilis et Mariage, 2 me Edn.) increased the period to ' three or four years as a minimum.' Since then the marriage of syphilitics is sanctioned after four years by Prince Morrow (1904), after five years by Ledermann (1904), and Balzer (1906). The most recent dictum from Professor Gaucher-the successor of Fournier at the Saint Louis Hospital, Paris-is five years, provided no symptoms have occurred during the last two years." It need not be added that lapse of time must be reinforced by effective and continued treatment.

In this address, I have only been able to indicate certain main lines of thought; but what I have said from the standpoint of a pathologist may serve as an introduction to the ripe experience of physicians, surgeons, and specialists in various departments. I fervently trust that this debate will materially assist in quickening observation, in settling opinion, in defining practice, in regard to a subject which seems to me of the utmost importance for the well-being of the individual, and for the safety of the community.

