

FATAL SYPHILIS*†

BY

WILFRID D. NEWCOMB

From St. Mary's Hospital, Paddington, London

In these times when more and more stress is being laid on the prevention of disease, and its early recognition and cure, the *post-mortem* room is frequently derided as a place where only terminal stages are seen. A morbid anatomist can scarcely be expected to agree with this attitude in any circumstances, but in a discussion on fatal syphilis he surely comes into his own. Data obtained at necropsy can hardly be frowned upon, and this paper is based on material collected from the *post-mortem* records of St. Mary's Hospital, a medium-sized teaching hospital to which is attached a large V. D. Clinic.

The subject will be considered under two heads :

(1) Syphilis as a cause of sudden death.

(2) Frequency of syphilis in the *post-mortem* room during the last 30 years.

It has been a custom in the Morbid Anatomy Department at St. Mary's Hospital to define sudden death as those cases "brought in dead". Many of these are, of course, accidents and valueless in this connexion, but "Medical B.I.Ds", as they are usually called, make a definite and interesting group. Table I shows the frequency of various causes of death in two hundred consecutive cases brought in dead (B.I.D.), accidents being excluded. It will be seen that the cardiovascular group, accounting for almost three-quarters of the whole, is by far the largest, the percentage being much larger than those which have been published by many coroners' pathologists (Simpson, 1953). The reason is probably that on the whole "B.I.Ds" have a

higher proportion of instantaneous deaths than the groups examined by coroners' pathologists, which include unexpected as well as sudden deaths. By far the commonest cause of instantaneous death is coronary obstruction.

The cardiovascular group has been split up (Table II), and here again one group overshadows the rest, namely that called coronary atheroma. This diagnosis has been interpreted in its widest sense and includes coronary thrombosis, ischaemic fibrosis of the myocardium, cardiac aneurysm, etc. It is the commonest cause of sudden death in all published statistics.

The second group, the only other to reach double figures, is syphilis, which accounts for 12 per cent. of medical B.I.Ds. Ruptured aortic aneurysm is responsible for nearly half of these, the rest dying of heart failure, due to aortitis either occluding the coronary orifices or associated with aortic endocarditis.

The last line of Table II refers to ruptured dissecting aneurysms of the aorta of which five examples were observed. The frequency of these non-syphilitic aneurysms appears to be increasing and is now approximately equal to those due to syphilis. This agrees with the figures published by Simpson (1947).

Syphilis is thus the second commonest cause of sudden death, a fact that is perhaps not as widely known as it might be. Ruptured aortic aneurysm is well known both to novelists and playwrights, but even when eroding the sternum it rarely bursts externally as both appear to believe. Rupture usually occurs near the neck of the sac, and so is

TABLE I
ANALYSIS OF 200 CONSECUTIVE MEDICAL "B.I.Ds."

Cause of Death	Per cent.
Cardiovascular disease	74.5
Respiratory disease	10
Poisoning	7.5
Drowning	2.5
Cerebral haemorrhage	2
Other	3.5
Total	100

TABLE II
ANALYSIS OF 149 CARDIOVASCULAR DEATHS

Condition	No. of cases
Coronary Atheroma	108
(Ruptured aneurysm 11)	
Syphilis - Coronary occlusion 7	24
(Heart failure with aortitis 6)	
Valvular Disease of the Heart	12
Dissecting Aneurysms	5
Total	149

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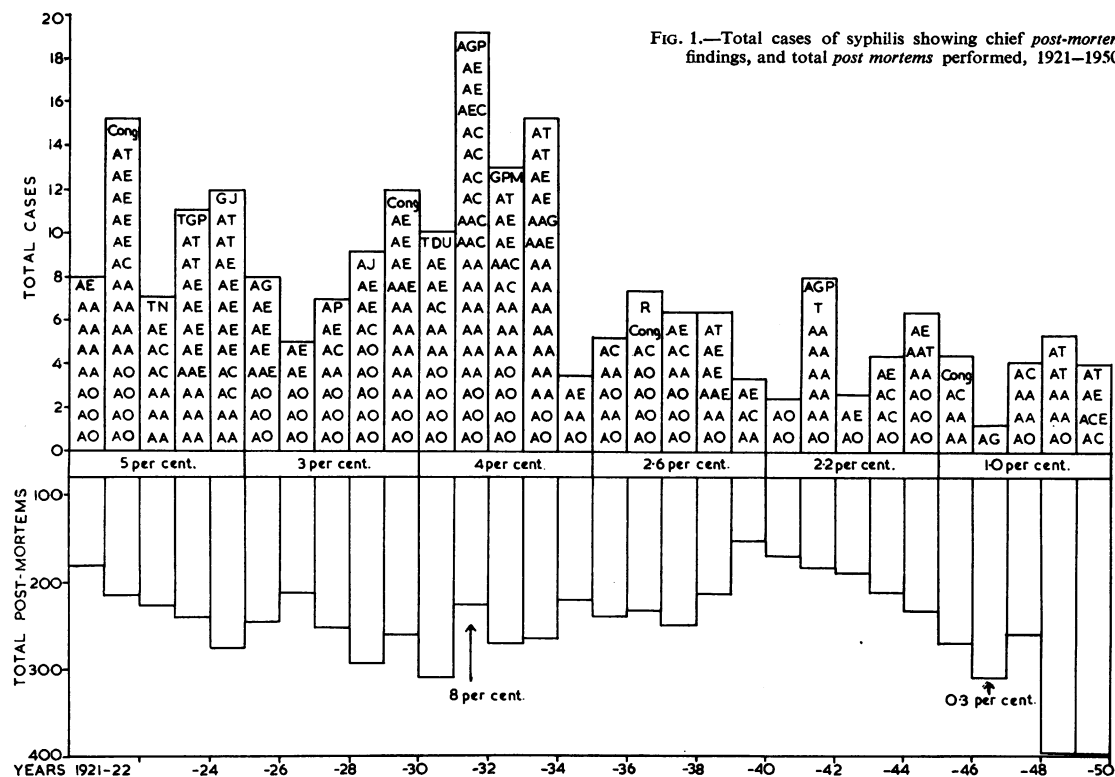


FIG. 1.—Total cases of syphilis showing chief *post-mortem* findings, and total *post mortems* performed, 1921–1950.

more common into the mediastinum than through the skin. The risk of sudden death from obstruction of the coronary ostia is well known, but some patients still die from Herxheimer reactions as the result of too vigorous treatment.

The general incidence of syphilis in the *post-mortem* room is not so great, but it is by no means negligible. During the 30 years 1921–1950 at St. Mary's Hospital, there were 7,501 autopsies, roughly 250 a year. In that time, 220 cases of syphilis were recognized in the *post-mortem* room, an average of 7.3 per annum, or approximately 3 per cent. of all necropsies. This incidence of syphilis is shown graphically in Fig. 1 where the annual number of cases of syphilis is plotted above the line and the yearly total of *post mortems* below. At first sight it seems to resemble New York water front, but the apparent windows in the skyscrapers are letters representing the chief *post-mortem* findings in the cases of syphilis.

The first A in each square of Fig. 1 stands for syphilitic aortitis, and, as would be expected, nearly all the cases diagnosed as syphilis at autopsy showed this lesion. The second letter in each square indicates other lesions as follows :

- O = syphilitic aortitis merely an incidental finding, not the cause of death.
- A = aneurysm.
- C = coronary orifice occlusion.
- E = aortic endocarditis.
- T = tabes dorsalis.
- GP = general paralysis.
- M = malaria.
- R = respiratory disease.
- Cong. = congenital syphilis.
- P = pachymeningitis haemorrhagica.
- J = jaundice.
- N = nephritis.
- DU = ruptured duodenal ulcer.

In order to show the trends more clearly the percentage incidence for each quinquennium is written just below the base line. Except for the rise in 1931–1935 there is a steady fall from 5 per cent. in 1921–1925 to just over 1 per cent. in the last quinquennium. The rise to 8 per cent. in the early 1930s culminating in 1932, which is the highest incidence in the whole period, occurs 16 to 20 years after 1914 and suggests that it corresponds to the period of maximum mortality from infection during the first world war. It is unlikely that there will be

FIG. 2.—*Post-mortem* appearance of liver in a case of syphilis.

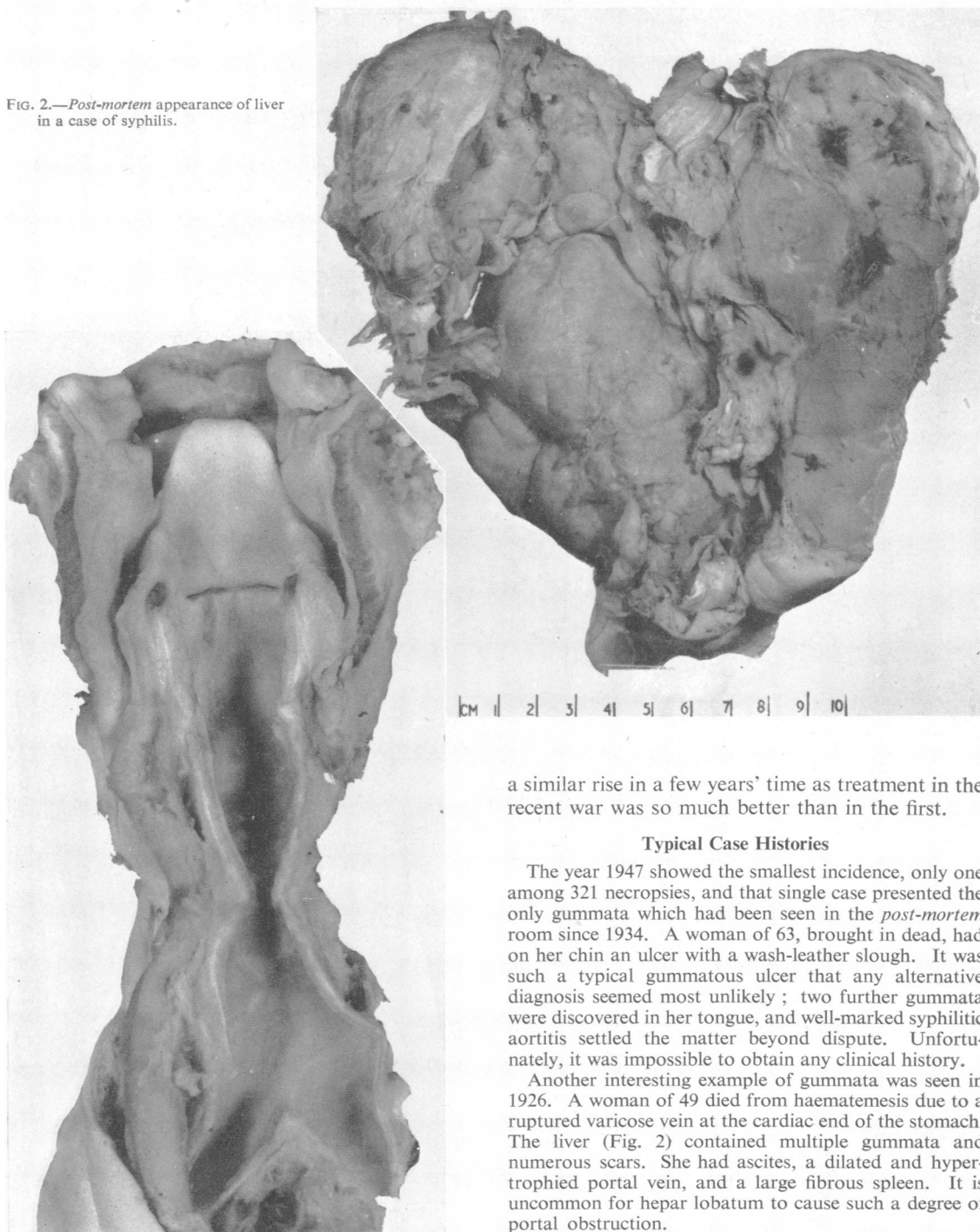


FIG. 3.—Syphilitic stenosis of the oesophagus.

a similar rise in a few years' time as treatment in the recent war was so much better than in the first.

Typical Case Histories

The year 1947 showed the smallest incidence, only one among 321 necropsies, and that single case presented the only gummata which had been seen in the *post-mortem* room since 1934. A woman of 63, brought in dead, had on her chin an ulcer with a wash-leather slough. It was such a typical gummatous ulcer that any alternative diagnosis seemed most unlikely; two further gummata were discovered in her tongue, and well-marked syphilitic aortitis settled the matter beyond dispute. Unfortunately, it was impossible to obtain any clinical history.

Another interesting example of gummata was seen in 1926. A woman of 49 died from haematemesis due to a ruptured varicose vein at the cardiac end of the stomach. The liver (Fig. 2) contained multiple gummata and numerous scars. She had ascites, a dilated and hypertrophied portal vein, and a large fibrous spleen. It is uncommon for hepar lobatum to cause such a degree of portal obstruction.

Seventeen tabetics are included during the period under consideration. The majority died from an

ascending urinary infection following cystitis, but some as the result of an acute abdominal catastrophe. One had a laparotomy for gastric crisis; nothing was found but unfortunately he died after the operation from septic peritonitis. This was, of course, before the discovery of antibiotics and the sulphonamides. On the other hand, there have been several examples of perforated ulcer in tabetics where the patient did not complain of any pain and the condition remained undiagnosed to the end. One man ate a large dinner half an hour before he died and at *post mortem* much of the food was found free in the peritoneal cavity, having escaped from the stomach through a large perforation which appeared to be about 2 days old.

Death from asphyxia from syphilitic stenosis of the trachea is a pathological rarity. Fig. 3 shows an example from a woman aged 44. The scars of gummatous ulcers in the vocal cords are not well shown in the photograph, but the stricture is obvious and, at autopsy, it was completely plugged with mucus. She was first admitted for hoarseness and dyspnoea. She was a known syphilitic and had the usual treatment with arsenicals but remained

dyspnoeic. She was re-admitted in 1937 with alleged asthma, which was treated with adrenaline but did not respond. She became more and more cyanosed until death. The lungs showed gross vesicular emphysema with oedema and congestion at the base.

I have attempted to show the trend of syphilis in the *post-mortem* room at St. Mary's Hospital for the last 30 years. The incidence now is remarkably small, and one wonders if in 30 years' time it will have become impossible to find the stigmata of syphilis at necropsy.

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