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THE AETIOLOGY OF POST-ARSPHENAMINE JAUNDICE

BY

J. BEATTIE, M.D., D.Sc.
Bernhard Baron Professor, R.C.S.

AND

J. MARSHALL, M.B., M.R.C.S.
Major, R.A.M.C.

(From the Bernhard Baron Research Laboratories, Royal College of Surgeons of England)

Damage to the liver parenchyma, with or without icterus, can occur at any stage of untreated syphilis, acquired or prenatal. Icterus is rare in early acquired syphilis, and when it appears is usually coincident with secondary manifestations, original or relapse. The liver biopsy findings in cases of jaundice in untreated secondary syphilis are indistinguishable from those seen in cases of post-arsphenamine jaundice. Liver material from untreated secondary syphilitics who show no clinical signs of liver damage is quite normal. Jaundice in untreated cases, though of some interest, is of less importance than the icterus which appears after arsenical treatment has been started. This type of jaundice, frequently referred to as "post-arsphenamine jaundice," differs from the jaundice of untreated early syphilis in certain respects. In the latter type the use of arsphenamines in treatment is, in our experience, without danger and leads to a rapid clearing of the icterus. This is not so in the case of post-arsphenamine jaundice. Although many cases have been described in which arsenical treatment has been continued through post-arsphenamine jaundice, we are convinced that such a policy is dangerous. The administration of arsenic before clinical and biochemical recovery in some such cases has produced alarming evidence of increased liver damage. Milian (1934), who believed that the delayed jaundice of arsphenamine treatment is due not to the drug but to a hepatic recurrence of syphilis, continued to treat 75 cases with arsphenamine, and claimed satisfactory results in 66. Of the remainder, 11 were intolerant and 4 died.

Goodman and Gilman (1941) thought that jaundice during arsphenamine treatment "may be due to one or a combination of the following factors—the drug, syphilis itself, or intercurrent infection. The evidence is convincing that many cases represent attacks of non-specific catarrhal jaundice occurring in patients whose livers are subjected to the added insults of syphilis and an arsenical." Mitchell (1943), reporting on jaundice in syphilitics under treatment in the Canadian Army in Great Britain, suggested that jaundice is due to "the association of two hepatoxic agents—the arsenic and the agent or toxin of infectious hepatitis in patients under arsenotherapy." These two opinions imply that the infectious hepatitis or non-specific catarrhal jaundice (other synonyms—toxic hepatitis, non-spirochaetal epidemic jaundice, simple jaundice, acute catarrhal jaundice) associated with arsenical treatment in syphilitics is identical with that which produces jaundice so frequently in the normal population and which has become so prevalent in recent years.

Two Types of Hepatitis

It is necessary to emphasize the fact that there are apparently two types of hepatitis occurring during arsenical therapy: (*a*) an early type which is usually mild and appears within the first two weeks after the first injection of the drug has been given; and (*b*) a late type which may appear at a variable time after starting treatment, but usually becomes obvious between the 12th and 17th weeks of treatment.

The early type is most commonly seen in intensive arsenotherapy (five-day or twenty-day), but it can occur during the standard treatment course of weekly or bi-weekly injections of an arsenical. The manifestations vary from an increase of urobilinogen excretion to a slight degree of actual icterus of conjunctivae or skin, but are always short-lived. It has been suggested that the cause of this condition may be a direct arsenical damage of the capillaries somewhat analogous to that found in haemorrhagic encephalitis following intensive arsenical treatment. The late type usually appears, as has been stated above, between 12 and 17 weeks after the initiation of arsenical treatment, whether by the intensive or the standard methods.

The pathological pictures and laboratory findings in these two types are different. In the early type there is an increase in the total blood cholesterol, cholesterol esters, and blood phosphates, with "bile thrombi" and cholangiolitis in liver biopsy specimens (Gutman and Hanger, 1941; Naunyn, 1919). The late type shows a marked alteration in the liver cells, ranging from swelling to necrosis with varying degrees of fibrosis (Dible and McMichael, 1943; Dible, McMichael, and Sherlock, 1943). The serum bilirubin values are higher than normal, and may be as high as 49.5 mg. per 100 ml. (in a non-fatal case in our series).

The type with which we are here concerned is the late or delayed type of jaundice which occurs in patients suffering from early syphilis who are being treated with neoarsphenamine by the routine British Army method. This antisyphilitic treatment consists of weekly injections of 0.6 g. of neoarsphenamine for ten weeks, followed by an interval of four weeks, when a similar second course is given. Third and fourth courses of treatment are given, but a four-weeks rest is interposed between each two treatment courses.

Syphilis and Arsenicals as Causal Factors

The three factors which Goodman and Gilman (1941) suggest as being responsible, either singly or in combination, for post-arsphenamine jaundice are syphilis, the arsenicals, and an attack of non-specific catarrhal jaundice. There is no pathological evidence to support Milian's view that the delayed jaundice of the type under consideration is due to syphilis of the liver (Dible and McMichael, 1943). The organic arsenicals used in the treatment of syphilis are, however, hepatotoxins. They can produce liver damage in experimental animals, and are almost certainly responsible for the early type of jaundice occurring in intensive arsenical treatment (Lloyd Jones and Maitland, 1943). If they were solely responsible for post-arsphenamine jaundice it might be expected that the incidence of jaundice would remain fairly uniform from year to year. Before the war and up to the spring of 1941 the experience of one of us (J. M.) in clinic practice showed that the incidence of jaundice was about 2 per 100 new cases of early syphilis under treatment. Although there was no change in the scheme of treatment, dosage, type, and manufacturers of the drug, the

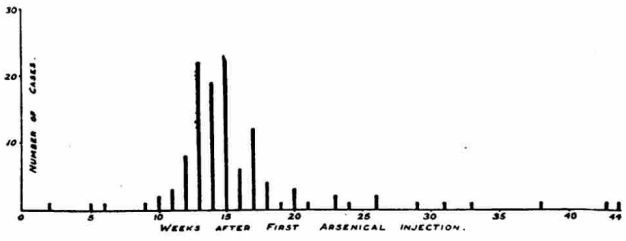
incidence rose steadily during 1941 and 1942, until in one clinic (M.I.H.) 46% of treated cases developed jaundice.

The possibility that owing to wartime conditions there might have been some alteration in the toxicity of the drug used was considered. This hypothesis was discarded early when it was discovered that other clinics whose jaundice incidence was lower were using the same drugs and in some cases the same batches of drugs from the same manufacturer. Drugs from different manufacturers and different batches of the same drug were tried in this clinic, but the incidence was not significantly affected whatever drug or batch was used. As Curtis (1942) had observed that an increase in the amount of drug used (from 7.5 to 13 g. in thirteen weeks) increased the jaundice incidence from 7–8% to 25–30%, it was thought that a reduction in the dose of arsenical from 5.85 g. to 4.05 g. over a period of ten weeks might show a significant decrease in incidence. In certain clinics where the reduced dosage was used no such decrease was observed during an adequate period of observation. When arsenoxide was given by bi-weekly injection instead of neoarsphenamine once weekly there was again no significant drop in incidence, although the total amount of arsenic was very much reduced.

The above evidence suggested that although syphilis and the arsenicals can produce liver damage they cannot be responsible alone for the increase in jaundice observed in our clinic (M.I.H.). Ruge (1927) made the observation that in the German Navy the incidence of jaundice in syphilitics bore a definite relation to the incidence of infective (non-spirochaetal) jaundice in the general population. When the incidence rose in the non-syphilitics a parallel rise occurred in the syphilitics. Stokes, Ruedemann, and Lemon (1920) noted a similar phenomenon. The present rapid rise in the incidence of jaundice among syphilitics in this country has been observed over a period when infective hepatitis was becoming increasingly common in the general population. There would thus appear to be good reason for considering the third possibility of Goodman and Gilman's—namely, an infective factor.

The Role of Infection

The complete records of 119 male cases of early syphilis who had received all their antisyphilitic treatment at one clinic (M.I.H.) were available. The time interval between the attendance at the clinic when the first arsenical injection was given and the time of onset of jaundice was determined for each patient. A graph showing the number of cases of jaundice in each week following the first injection was prepared (see Fig.). The array ranged from the 11th to the 44th week after



Graph showing number of M.I.H. cases of post-arsphenamine jaundice occurring in each week of antisyphilitic treatment. (Total cases, 119.)

the first arsenical injection. The median lay in the 15th week. Of the 119 cases, 90 (76%) occurred during the 12th to 17th weeks (inclusive).

If it be assumed that some infective factor was transmitted from patient to patient, the latent or incubation period of most of the cases would appear to be between 12 and 17 weeks. The wide limits of the incubation period and the presence of 20 cases occurring after longer periods might be explained by assuming that infection did not take place until some injection later than the first. Some of the remaining 9 cases which occurred before the 12th week may not be late or post-arsphenamine jaundice, but may be examples of the early type discussed above.

It seemed unlikely that the disease was spread by contact, as is infective hepatitis, but that the spread occurred through inoculation of an infective agent when arsenical injections were being given. Bigger (1943) has pointed out that imperfect sterilization might be responsible for leaving in the syringes infective material which could then be passed into the next person injected by the same syringe and needle. The incubation period in our cases of post-arsphenamine jaundice was probably between 80 and 100 days, which was about three times as long as that determined by Pickles (1939), Edwards (1943), and Ford (1943) for infective hepatitis in non-syphilitics in which there was no possibility of transmission by inoculation.

The length of the incubation period and the possibility that an infective agent had been transmitted by inoculation suggested that post-arsphenamine jaundice might be identical with the jaundice which occurs after the administration of human blood products or of yellow fever vaccine containing human serum (Findlay *et al.*, 1937, 1938, 1939; MacNalty, 1938; Propert, 1938; Soper and Smith, 1938; Beeson, 1943; Memorandum by Medical Officers of the Ministry of Health, 1943; Morgan and Williamson, 1943; Steiner, 1944). Although the possible incubation periods recorded by these various observers are not identical the median for each series of observations falls within or slightly before the period in which the greater number of our cases of post-arsphenamine jaundice occurred (76% of cases between the 12th and 17th weeks). While this evidence is highly suggestive it is not sufficient to prove the complete identity of post-arsphenamine jaundice with the types following the use of human blood products. It does, however, appear to separate all these types of jaundice from the epidemic form of infective hepatitis such as described by Pickles.

An attempt has been made to obtain some precise information on the method of transmission of the disease. It was suggested by MacCallum early in 1943 that if transmission of the infective factor in post-arsphenamine jaundice was due to inoculation of infective material contained in imperfectly sterilized syringes, then if each patient were given a syringe at the beginning of his treatment and received injections only by that syringe after proper sterilization the transmission of infection ought to be eliminated. It was decided to treat 10 early syphilis cases and to give each patient a new and unused syringe for his exclusive use for all his injections. (The syringes for this experiment were provided by Dr. MacCallum.) Such syringes and needles were washed and boiled both before and after each injection. Because of postings to units away from the Command only four of the ten were observed beyond 120 days after the first treatment. No case of jaundice appeared in this group in spite of the fact that the patients were in contact for about the first 14 days of treatment, and subsequently at least once each week, with men who later developed post-arsphenamine jaundice. One of the four men under observation proceeded on leave and received two arsenical injections at another clinic from syringes used to inject other patients. The syringes were not boiled after being used for previous patients, but had been rinsed with sterile water followed by 70% alcohol, and were given a further rinse in distilled water. This man developed jaundice later. The relevant data are given below:

Patient S

Date of first injection at "S" clinic	May 26, 194-
" " " at "S" clinic	Sept. 2
" " second " "	" 8
" " first symptom of liver damage	Nov. 24
" " icterus	" 27
Interval between first injection at "S" clinic and first symptom of jaundice	83 days

This series, though short when taken in conjunction with Bigger's experiments, is strongly suggestive of blood transmission. The single case of jaundice after a probable accidental infection might establish the latent or incubation period for post-arsphenamine jaundice as being about 12 weeks. It suggested, too, that patients transferred from V.D. clinics, where the incidence of jaundice was low, to M.I.H. clinic, where the incidence was high, might, if they developed jaundice, show some evidence that their infection took place at the latter clinic. Eight men were traced who had such histories. Details are given in Table I.

All these cases have time intervals between the first injection at M.I.H. clinic and the onset of jaundice which fall within the range of 12 to 15 weeks—precisely the range within which the greater number of cases of jaundice appeared in the large series of 119 patients. If it be assumed that the exact incubation period is 12 weeks from inoculation of the infective material (as occurred in the single case described above), then only a

TABLE I

Case	Date 1st As Inj.	Date 1st As Inj. at M.I.H.	Date Appearance of Icterus	Days from 1st Inj. to Icterus	Days from 1st Inj. at M.I.H. to Icterus
Fr.	26/ 3/42	13/ 8/42	4/12/42	250	110
G	17/ 4/42	22/ 8/42	9/12/42	236	109
M	2/ 8/42	16/10/42	30/ 1/43	181	106
N	12/ 1/42	18/11/42	21/ 2/43	405	101
L	29/ 9/42	20/11/42	17/ 2/43	141	89
S	7/ 8/42	3/ 1/43	20/ 4/43	256	107
F	27/11/42	25/ 2/43	4/ 6/43	190	99
D	17/10/42	26/ 2/43	7/ 6/43	233	101

portion of the patients are infected at the first injection, but the chances of becoming infected are greater with each successive injection. This would explain the shift in the median to the 15th week in the long series, the large number of cases occurring up to the 17th week after the first injection, and the sporadic cases up to the 44th week.

"X" and "Y" Diseases

The disease described by Pickles, Edwards, and Ford had an incubation period of about 28 to 30 days and was transmitted by contact with a person who was infective. In post-arsphenamine jaundice and in homologous-serum jaundice the incubation period is about three times as long and transmission is by inoculation of the infective material. These considerations suggested the possibility that there were two diseases—an "X" disease—i.e., infective hepatitis—and a "Y" disease, including post-arsphenamine jaundice and jaundice following the injection of human blood products as described above. Clinically and biochemically it has not been found possible to differentiate these diseases. When biopsy specimens from the liver are taken the pathological appearances have been shown to be common to each type of disease (Dible, McMichael, and Sherlock, 1943).

Assuming that two diseases do exist and that the infective factors are different, immunity conferred by one disease might not protect against the other. Pickles, in a private communication, is of the opinion that the immunity conferred by an attack of jaundice (infective hepatitis) is "as complete as that conferred by an attack of measles." His records contain only one instance of a possible reinfection. It would appear that there is but a remote chance of a reinfection with X disease. In our series of 105 cases of X disease no instance of a reinfection could be traced. There were no clinical relapses in this series. By analogy it might be expected that an attack of Y disease would confer similar immunity.

At one hospital (M.I.H.) 280 cases of jaundice were treated in the jaundice ward and one as an out-patient. All were syphilitics. Of this group seven developed a second attack of jaundice. The relevant data are given in Table II.

TABLE II

Case	1st Inj. As	1st Symptom of Jaundice	Interval between 1st Inj. and 1st Symptom (days)	2nd Attack of Jaundice began	Interval between Attacks (days)	Interval between Discharge from Hospital and Date of 2nd Attack (days)
H	9/12/41	26/ 1/42	48	29/ 6/42	154	137
M	9/ 7/42	9/11/42	123	1/ 3/43	112	98
B	14/ 8/42	22/ 2/43	192	29/ 7/43	157	119
L	29/10/42	8/ 3/43	130	5/ 5/43	58	50
C	12/12/42	21/ 3/43	99	22/ 5/43	62	33
E	13/ 5/43	10/10/43	150	3/11/43	24	7
Fs.	8/ 6/43	11/ 9/43	95	11/10/43	30	11

Patients E and Fs. were most probably examples of true relapse caused by untimely celebration of their discharge from hospital. There was no evidence that, either immediately before or during their stay in hospital, they had been in contact with a case of infective hepatitis or a person who subsequently developed the disease. Patients C and L had been seen at weekly intervals between their discharge from hospital and their second attack of jaundice. They were apparently quite fit during the interval and were on full duty. Arsenical injections had not been resumed. There was thus a possibility that their second attack was due to an infection with X disease after their discharge from hospital. Contact with known cases could not be proved. Cases H and M could not have been reinfections with Y disease as they had received no injections of any kind at the hospital or elsewhere until four weeks before their second attack, when arsenical treatment was resumed. Toxic hepatitis due to the arsenical itself was ruled out as there was no supporting evidence to justify this possibility. There remained a possible infection with X disease, but again an attempt to trace contacts failed. Patient B's case was exactly similar to those of Patients H and M except that he resumed arsenical treatment only two weeks before the second attack. If the two patients who had a simple relapse be omitted there are left five possible examples of infection with X disease out of a total of 281 cases. This gives a possible "infection" rate at this hospital (M.I.H.) of 1.8%.

At another hospital, B, the patients under treatment in the jaundice ward included some who had X disease (infective hepatitis) and others with Y disease (post-arsphenamine jaundice). At no time did the ward contain only one of the two diseases. During a period of six months 80 cases of Y disease and 40 of X disease were treated. While there were no relapses or reinfections in any of the cases of X disease five so-called relapses occurred in Y-disease patients. An analysis of the time of appearance of the relapses showed that they fell within a period lying between the 30th day after admission to hospital and the 30th day after discharge. As it was possible in this group to be certain of full clinical and biochemical recovery, and as all had shown no abnormal response to test doses of neoarsphenamine, the presumption is that these cases were true infections with X disease and that such infections were contacted while in the ward. The "infection" rate was therefore 6.3% as compared with 1.8% in the other hospital (M.I.H.).

In Hospital B the cases of X disease occurred exclusively in U.S. Army personnel, while the Y cases occurred almost exclusively in British Army personnel. An analysis of the U.S. Army cases showed that all had received yellow fever inoculations at least eight months before they developed jaundice in England. Of the 40 cases 5 had developed jaundice within four months after their yellow fever inoculations and while still in the U.S. If it be assumed that post-vaccinal yellow fever jaundice is Y disease, then this disease did not confer immunity against an attack of X disease. That the second attack was X disease was proved by evidence of contact with cases of X disease 28 to 30 days before the second attack began.

The evidence so far is suggestive that an attack of Y disease does not immunize against X disease. It might be expected that an attack of X disease does not immunize against Y disease. Of the 360 cases of Y disease in both hospitals evidence of an attack of jaundice preceding the syphilitic infection was obtained in only 6 cases. The time interval between the attack of presumably X disease and the attack of Y disease varied between 10 years and 18 months. The evidence to support a diagnosis of X disease was not good, as it was impossible to secure a history of contact with other cases of jaundice, except in two of the six cases.

The recent large-scale epidemics of post-vaccinal yellow fever jaundice and of infective hepatitis ought to provide in the near future more convincing evidence than has been produced here on whether or not there are two diseases each of which provides no immunity against the other.

Summary of Present Evidence

The evidence at present available may be summarized as follows:

X disease, or infective hepatitis, has an incubation period of about 28 to 30 days and is transmitted by contact. It can exist in a form in which icterus never appears but which is apparently sufficient to confer immunity against reinfection.

Y disease, whether late post-arsphenamine jaundice or homologous-serum jaundice, has an incubation period of 80 to 100 days and is transmitted by inoculation of infective material (blood, serum, or plasma).

An attack of Y disease presumably confers no immunity against X disease.

Evidence that an attack of X disease confers no immunity against Y disease is scanty and incomplete, but is suggestive.

Relapses of Y disease have been explained mostly on the grounds of an infection with X disease.

It is not known whether an attack of Y disease due to inoculation with icterogenic serum or plasma confers immunity against the Y disease occurring during arsphenamine treatment for syphilis.

In view of the identity of clinical, biochemical, and pathological pictures of X and Y diseases the final proof of the reality of two infective factors must rest in the end on the production of convincing serological evidence and the accumulation of more data on their aetiology.

[Since the above was written we have had, through the kindness of Dr. A. M. McFarlan, the opportunity of reading an unpublished paper by himself and Dr. G. Chesney on jaundice following the injection of mumps convalescent serum. These observers have put forward views which are in close agreement with ours but are based on other data and were arrived at quite independently. Dr. McFarlan generously offered to allow us to use some of the unpublished data in his article, but as publication of it is imminent we preferred to see both sets of data appear separately.]

We wish to thank the medical officers of the two hospitals where cases have been under observation for their co-operation and careful note-taking. To Lieut.-Col. H. L. Sheehan, R.A.M.C., we owe more than mere thanks, for the hypothesis which we have put forward was developed during a discussion with him. He is therefore in part responsible for some of the basic ideas here presented. We are grateful to Dr. A. M. McFarlan for reading our manuscript and for many most helpful suggestions which we have adopted. We acknowledge our indebtedness to Dr. Wilfred Pickles for checking his case records of infective hepatitis to determine the incidence of reinfection in his series and also for much useful information he has given us concerning this disease.

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WHAT IS PSYCHIATRY?*

BY

ALAN GREGG, M.D.

Director of the Medical Services Division of the Rockefeller Foundation

It has been my experience that, apart from an imaginative minority, most people have either had close contact with what is called mental disease through having seen a relative or friend attacked by some form of it, or, on the other hand, they have virtually no interest in psychiatry, but merely an ignorance enhanced by aversion and evasion. Close contact even for one day with a friend who has become insane is an experience which beggars any argument for the importance of psychiatry, and only those who have never seen mental disease at close range can shrug their normal shoulders with unconcern or with unruffled resignation assume the adequacy of the nearest asylum.

Whereas the usual physical ills concern the inadequate performance of heart, lungs, stomach, or some other organ in the service it renders to the rest of the body, psychiatric diseases must in the main still be defined in terms of inadequate performance of an individual human being *vis-à-vis* other human beings. Psychiatrists study and treat human beings who are inadequate or actually dangerous in their behaviour as members of society; and so it happens that psychiatry as the study of disordered conduct is intimate to an almost suspicious degree with ethics, with cultural anthropology, with sociology, with metaphysics, with religion, with artistic activities—and this despite the fact that the behaviour of a human individual as a whole should be as soundly understood in terms of medicine as the behaviour or function of any of his component organs. To understand mental disease calls for medical art and science, but also for a wide knowledge of the society and culture to which each of us must learn to adjust.

Let me here offer you two ideas of cardinal importance to your understanding what psychiatry is: first, that the psychiatrist studies the function and the influence of mental processes and emotional states in the whole vast range from incurable disease to optimum health; and, second, that the psychiatrist seldom handles conditions which he can describe without reference to the demands of society upon the individual. As the functions of an automobile are not the functions of its carburettor or its gears, so the functions of man as a whole individual not only transcend but differ radically from the functions of any of his component organs or systems. If psychiatry has especially close connexions with the nervous system, it is quite natural; for the function of the nervous system is to co-ordinate, to integrate, to adjust, to harmonize, to administer the services of all the organs of the individual and to perpetuate his identity as a person through a finite but extremely long series of changing environments—dangers and difficulties and defeats, as well as resting periods, comforts, and delightful successes.

The Range of Psychiatry

I should not be satisfied with the definition of psychiatry as "that specialty of medicine which deals with mental disorders." Like a bad newspaper headline, such a definition confines while condensing and misrepresents by oversimplifying. Psychiatry deals also with the emotional and social life of man, not merely his reasoning mental operations. In so far as experience has shown you that emotional thinking is different from logical reasoning, the whole purview and range of psychiatry is evidently extended. Indeed, the province of psychiatry is the conduct of man, his reactions, his behaviour as an indivisible sentient being with other such beings. Until recently attention has been given only to grossly disordered conduct—to persons locked up in asylums; but now the field is far more inclusive because it reaches into the anxieties, the fatigues, the instabilities, the adjustments, the disturbances of normal everyday living, and also because it includes the effects of mental and emotional functions upon the component organs of the body as well as the effects of disorders of these organs upon the functions of the human being as a whole.

Let us make the position of psychiatry a little clearer by distinguishing it from neurology and from psycho-analysis. Neurology is the study of the nervous system; more specifically, the diseases of the nervous system. Obviously there is much overlapping between neurology and psychiatry when defects or diseases of the brain, spinal cord, and peripheral nerves are involved. But neurology points toward the functions of the nervous system as serving the efficiency of the individual machine, while psychiatry directs its attention to conduct—that blend of mental and emotional functions, dependent, it is true, largely upon the nervous system but involving more than the serviceability of that system to the individual as a whole. The location, structure, and function of the nerves and the paths taken by nerve impulses are the basic knowledge of the neurologist. The neurologist learns to detect where injury or infection is located along a large variety of nerve tracts. To the studies of gunshot injuries of the nervous system American neurology owes its beginnings during the Civil War. The location of a brain tumour, or the cause of a paralysis or loss of sensation due to nerve injury, is the task of a neurologist, not a psychiatrist. Most neurologists practise psychiatry because what the layman calls "nervousness" is not an affair of nerve tracts but is due to emotional conflicts or other psychiatric disturbances. Psychiatric disorders are much more numerous than neurological disorders.

Indeed, the psychiatrist's domain is almost bafflingly large, for it includes derangements of conduct or behaviour often

* An address given to the Trustees of the Rockefeller Foundation of New York.