incidences. There being no other theory that will account for all the phenomena of rheumatism and its treatment, I submit that the anaphylactic explanation must be accepted as the only alternative to a position that is no more than a contradictory muddle of mysteries.

A dictum of Kolmer is a fitting conclusion: "Under proper conditions it is reasonable to suppose that anaphylactic shock or shocks may occur during the course of a given disease, and that the effects of these may contribute to the lesions and symptomatology in no small degree."

BIBLIOGRAPHY

- Amoss, H. L., and Bliss, E. A.: *Journ. Exper. Med.*, 1927, xlv, 411.
 Arthus, A.: *C. R. Soc. de Biol.*, 1903, lv, 817.
 Auer: *Journ. Exp. Med.*, 1920, xxxii, 427.
 Bessau, G., and Detering, C.: *Zentralbl. f. Bakt.*, 1928, I Abt. Orig., cvi, 11.
 Davis, J. D.: *Arch. Int. Med.*, 1915, xv, 555.
 Dean, H. R.: *Journ. Path. and Bact.*, 1922, xxv, 305.
 Derrick, C. L., and Swift, H. F.: *Proc. Soc. Exp. Biol. and Med.*, 1927, xxv, 222.
 Fantus, B., Simmonds, W. E., and Moore, J. I.: *Arch. Int. Med.*, 1917, xix, 529.
 Fleisher, M. S., and Wilhelmj, C. M.: *Zeit. f. Immunitäts. u. exp. Therap.*, 1927, li, 115.
 Harding, A. E. B.: *Journ. Path. and Bact.*, 1926, xxix, 189.
 Hitchcock, C. H.: *Journ. Exper. Med.*, 1928, xlvi, 403.
 Irvine-Jones, E. I. M.: *Arch. Int. Med.*, 1928, xlii, 784.
 Isaacs, M. L.: *Arch. Path.*, 1928, v, 1028.
 Kaiser, A. D.: *Journ. Infect. Dis.*, 1928, xlii, 25.
 Klopstock, A., and Selter, G. E.: *Klin. Woch.*, 1927, vi, 1662.
 Kolmer, J. A.: *Infect., Immun., and Biol. Ther.*, Saunders, 1923, p. 658.
 McLachlan, D. G. S., and Mackie, T. J.: *Journ. of Hyg.*, 1928, xxvii, 225.
 Mendenhall, W. L., McClure, C. W., and Cate, M.: *Boston Med. and Surg. Journ.*, 1926, cxv, 76.
 Muir, R., and Ritchie, J.: *Manual of Bacteriology*, Oxford University Press, 1919, seventh edition, p. 600.
 Nordman, M.: *Physiol. Rev.*, 1931, ix, 41.
 Ratner, B., Jackson, H. C., and Gruhl, H. L.: *Journ. Immunol.*, 1927, xiv, 249, 275, 291.
 Raulston, B. O.: *Biochem. Zeit.*, 1927, clxxxiv, 31.
 Smetana, H.: *Proc. Soc. Exp. Biol. and Med.*, 1927, xxiv, 821.
 Swift, H. F., and Boots, R. H.: *Journ. Exper. Med.*, 1923, xxxvii, 553.
 Swift, H. F., Hitchcock, C. H., and Derrick, C. L.: *Proc. Soc. Exp. Biol. and Med.*, 1928, xxv, 312.
 Topley, W. W. C., and Weir, H. B.: *Journ. Path. and Bact.*, 1921, xxiv, 333.
 Wright, H. D.: *Ibid.*, 1926, xxix, 5.
 Zinsser, H.: *Textbook of Bacteriology*, Appleton, New York, 1922, fifth edition, p. 367.

THROMBOSIS, EMBOLISM, AND THEIR TREATMENT

BY

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With the recent progress of surgery, especially in major operations, there has been an increase of thrombosis and embolism, constituting an ever-present threat to the success of such operations. With this fact in mind I conducted experiments directed towards an inquiry into the cause of these pathological conditions. The results have been published by the *Klinische Wochenschrift*, 1932, ii, 502. On account of the interest taken in this work I will set forth the method of treatment which I have found very successful in the practical application of the principles established therein.

THEORIES OF CAUSATION

Before entering into details of the treatment we must discuss briefly what is the origin of thrombosis, so that it may be determined in which patients the disease is likely to occur. On this point scientific opinion is divided into two main groups—one believing it to be due to bacterial infection, and the other to a special constitutional predisposition. The first group—the supporters of bacterial infection—have reported cases of thrombosis and embolism in which they have tried to prove conclusively that certain infectious diseases are the cause of the

thrombosis. In the majority of cases the cause of infection was attributed to such non-virulent bacteria as the organisms found in influenza, bronchitis, etc., and in cases in which it was impossible to demonstrate any definite organism it was explained that the disease was due to some unknown ultra-filterable virus. The principal advocates of the germ theory—Franke, Kuskow, Kinzel, and Pathy—while subscribing to it in a general way, differ slightly in their conclusions. Either they believe thrombosis to be a direct result of bacterial action or they think that the virus or its toxin affects the individual, producing irregularity in the blood circulation, hypotonia, and damage to the blood vessels, thus giving rise to thrombosis.

The second group deny that infection has any connexion with thrombosis. The supporters of this theory—Payr, Morawitz, and especially Rehm, who is its founder—believe that all patients affected by thrombosis belong constitutionally to one type. They call this condition thrombopathy, and emphasize that in all such cases the patients are found to have a special construction of the sympathetic nerve system. Some members of this group qualify their opinion by stating that thrombosis may be due, not necessarily to a permanent, but to a temporary, difference in this system. Others—Koenig, for instance—in cases in which no cause can be found for thrombosis, nevertheless deny it to be the result of an infection. It is not my intention to criticize this theory at length, but I believe from the experiments I have made that it is absolutely wrong to state that there is a special idiosyncrasy which gives rise to thrombosis, because, if this were admitted, it would be impossible to explain why a patient, operated on two or more times, on one occasion suffers from thrombosis and on another does not, and why the conditions for thrombosis are not present at each operation. If we admit that temporary damage to an organ can bring about thrombosis, why must we exclude damage from a germ? To explain further: if damage to the intima of the blood vessels can produce thrombosis, that damage may arise from bacterial action. It cannot therefore be denied that infection may be a direct cause of thrombosis. On the other hand, one cannot agree with the supporters of the germ theory who believe that thrombosis is due only to infection. There are many cases in which it is absolutely impossible to find any organism, and in which, on the contrary, it is easy to find some special organic construction. The presence of a non-visible germ in these cases is not an acceptable theory.

METHOD OF DETECTING LIABILITY TO THROMBOSIS

I now wish to emphasize my opinion that thrombosis can never be regarded as due to only one class of causes: it is a pathological state which can arise from different causes at any time the equilibrium of the patient is destroyed. (It is irrelevant to discuss here the aetiology of this destruction of the equilibrium.) I do not believe that all patients are liable to thrombosis. In order to be affected by this condition it is not enough to have present the causes for producing it. I divide patients into two groups. In the first and, fortunately, the largest are the patients who can never suffer from thrombosis, even if the organs are damaged. In the second group there will always be a risk without any external influences. In making my experiments my intention was to find a means of detecting which patients can be affected by thrombosis and which can not. I began to search for some agent which would produce definite symptoms enabling me to divide the patients into two groups: those predisposed to thrombosis, and those whom I believed to be immune to it. After long and involved experiments it was possible to demonstrate that by the hypodermic injection of thyroxine the reaction of each group was different.

The method I have adopted is as follows. Two days before the operation all patients are injected with 1 c.cm. of thyroxine. After three hours the temperature, pulse, and blood are examined. In the first group, which may be termed "thyroxine-sensitive," as a result of the injection, the pulse quickens and the temperature rises slightly; difference in the blood composition is noticeable by the variation of the quantity of the blood corpuscles—the number of the red corpuscles increases slightly, whilst the number of blood platelets decreases. After two or three injections of thyroxine these become two or three times more abnormal, and the time necessary for blood coagulation is longer. On a patient in this thyroxine-sensitive group any external influence, especially the operation itself, will produce the effects mentioned above, and for this reason I do not believe they can be affected by thrombosis. In patients in the second group, termed "thyroxine-resistant," as a result of the same injection it is impossible to detect any change in temperature, pulse, or blood composition. A slight variation in blood corpuscles is sometimes noticeable, but is of no importance. To these patients every operation will bring the danger of thrombosis.

The scientific explanation of the reaction to thyroxine is explained in different ways. Freund and Boshamer believe that the thyroxine acts on the vegetative nervous system, and at the same time directly affects the cardiovascular system, producing the clinical symptoms in the patient. In the thyroxine-resistant group of patients the effects of the injection are almost unnoticeable, because the systems mentioned above show very little reaction. In this group of patients the parasympathetic nerve system predominates, thus facilitating the formation of thrombosis. During the last three years I have experimented with more than two thousand patients admitted for operation. In every case thyroxine was administered and the patient afterwards examined. In this way patients were divided into two classes: thyroxine-sensitive and thyroxine-resistant.

PREVENTIVE TREATMENT

After the thyroxine effect had been discovered it was necessary to find some treatment to prevent thrombosis. During this experimental period all cases in which thrombosis occurred were examined and were found, without exception, to belong to the thyroxine-resistant group. This tended to confirm my conclusion that only patients of this group were liable to thrombosis. The treatment that was found to be successful consisted in the administration, hypodermically, of a combination of atropine and ephetonin in the proportion of 1/100 grain atropine to 1/4 grain ephetonin. Not less than three injections in all were given every other day, beginning from the fifth day after the operation. I am able to state that not one patient treated in his way suffered from thrombosis or embolism.

The scientific explanation of the influence of atropine and ephetonin is as follows. With the first injection a variation in the blood composition is noticeable. Six hours after the injection the number of blood platelets is less than 100,000 per c.mm., and the number of red cells is slightly increased. The decrease of blood platelets occurs after every injection until the third, when the same level is maintained. The time necessary for coagulation of blood is two or three times longer than usual. This is shown more clearly in the diagrams.

In small hospitals where there is no laboratory equipment and it is thus impossible to divide the patients into thyroxine-sensitive and thyroxine-resistant, I believe it is advisable to treat all patients as belonging to the latter division. The injection of atropine and ephetonin can never be harmful to the patient, and is even useful for

the prevention of post-anaesthetic lung trouble. In this way mistakes will be avoided and deaths from thrombosis will be reduced to the minimum.

CHART I.—Thyroxine-sensitive patients.

CHART 2.—Result of ephetonin-atropine treatment of "thyroxine-resistant" patient.

SUMMARY

The origin of thrombosis is not confined to one group of conditions. It is rather a special pathological state due to external influences such as constitutional predisposition, bacterial infection, or toxæmia. Not all patients are affected by the thrombosis, but by the use of thyroxine it is possible to divide them into two groups: those immune from, and those predisposed to, thrombosis. The symptoms which demonstrate the difference between the two groups are the pulse, temperature, and variations and change in the blood composition. The treatment is the hypodermic administration on alternate days, beginning on the fifth day, of a combination of ephetonin and atropine.

TORSION OF THE NORMAL FALLOPIAN TUBE

BY

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Torsion of abnormal uterine appendages is not an uncommon condition, but torsion of normal appendages is much less frequently encountered. Torsion may involve either the normal tube or the normal ovary, or both; but this brief review is confined to tubal torsion, of which the following is an example.

A girl, aged 14, was admitted to King George Hospital on April 5th, 1931, under the care of Mr. Harold Dodd. Pain in the right lower quadrant had begun four days previously. After twenty-four hours it had become central, and vomiting occurred. The pain persisted and remained central. The bowels had acted on the day of admission, and there were no urinary symptoms. The first menstrual period had begun on January 1st, 1931, and had lasted seven days; the second, on February 4th, lasted four days; and the third, on March 8th, lasted four days. (The fourth began on April 6th, twenty-four hours after the operation, and lasted two days.) Since the age of 2 she had suffered from attacks