we refer the localized accumulation of this superfluous germinal material. Contractions can only occur through antecedent deposit of fibrous or connective tissue material, such as has been shown to result to greater or less degree throughout the lymphatic system in the active stage of syphilis.

We have then in the treatment of all the late lesions due to syphilis, and according to these views, two pathological conditions which legitimately demand consideration: first, the accumulated material which produces damage to the various structures involved, by mechanical pressure; second, the fibrous material, causing the contraction or obliteration of the lymphatic channels. In the first condition we should expect prompt and efficient relief to the accumulated germinal material through the influence of iodine and the iodide of potassium. Iodide of potassium, says Ringer, is the most soluble of all forms of iodine. It has been found also to be a form the most convenient for administration, and the one most usually acceptable in its effects upon the digestive apparatus. It has, therefore, been most generally used in the treatment of the so-called tertiary stage of syphilis.

Of the practical results of its use, simply as expressing the accepted status of this remedy in the late lesions due to syphilis, I will quote from the most recent authorities on therapeutics in this country, and which is but the reflection of the teachings of distinguished syphilographers throughout the world at the present day. "No therapeutical fact is more conspicuous than the cure of syphilis of the nervous system (arising from the presence of so-called gummy material) by the iodides. Mental disorders, epileptiform seizures, paralytic states, etc., *dependent upon gummatous nodes*, etc., are usually removed (by their use) in a manner little short of magical. Syphilitic paraplegia is equally amenable to the same means. The various neuralgias of syphilitic origin (that is to say caused by the presence of gummatous), *occurring in any situation*, are usually very promptly cured by the iodide of potassium. . . . There is no therapeutical fact more conclusively established than the power of iodide of potassa, in large doses, to arrest destructive syphilitic ulceration of the nares, palate, tonsils, and larynx."¹ Through their solvent or fluidifiant properties; through their action in stimulating the function of the mucous, lymphatic, and salivary glands; through its power to hasten tissue metamorphosis, iodine and its compounds constitute just the remedies which, from a purely scientific stand-point, we should select as best calculated to remove the elements of new formation which are demonstrated in the constitution of the so-called "*gummy material*." We have prescribed the iodide of potassium to arrest the progress of disease in the cases hitherto brought before you (pages 44 et seq.), and it is just the remedy which we shall find it judicious to prescribe in every case of late trouble due to syphilis, of whatever appearance, or in whatever locality, unless through some personal idiosyncrasy this form of iodine is not well borne.

The second condition alluded to as calling for attention in the treatment of the late lesions due to syphilis is that of the lymphatic channels, where we have shown that there is reason for believing contractions and obstructions due to deposit and organization more or less complete of fibrous material. Here we find, as might

have been inferred *a priori*, that the iodides are not sufficient; that while competent to cause the removal of the gummy material, in the great majority of cases (as previously suggested at page 50), the condition for its reaccumulation remains, and relapses occur. This is a well-established clinical fact. It is also as well established that through administration of a mercurial, in addition to or in combination with the iodide of potassium cures are more speedy and permanent; and hence it has come to be considered requisite for the successful management of lesions of all kinds due to late syphilis to combine mercury with the iodide of potassium. Fothergill says, "By the combination, then, of mercury to break down structurally the neoplastic growths, and iodine to stimulate the lymphatics, we can remove the products of excessive or perverted nutrition."²

It has, I think, been conclusively shown that the so-called gummy material of syphilis is, in the broadest view which can be scientifically taken in regard to it, nothing more than "the products of excessive or perverted nutrition." As such, then, we treat them with mercury and the iodide of potassium; not with the intention of neutralizing a virus, but upon principles which enable us, in case of inability from idiosyncrasy of the patient or from any other cause, to supply other remedial agents and measures, which, though less efficient, will yet be in the direction of intelligent effort to remove not alone the immediate results of syphilitic action, but to combat and often dissipate the conditions upon which the persistent recurrence of the lesions due to late syphilis is shown to depend.

Original Articles.

THE INFLUENCE OF AORTIC ANEURISM AND AORTIC INSUFFICIENCY, SINGLY AND COMBINED, ON THE RETARDATION OF THE PULSE.

BY A. T. KEYT, M. D., CINCINNATI, OHIO.

"L'anévrisme de la portion initiale de l'aorte coexistant avec une insuffisance aortique large, on n'observe pas l'exagération générale du retard du pouls qui est caractéristique de l'anévrisme siègeant à ce niveau; l'influence retardatrice de l'anévrisme est contrebalancée, par l'influence inverse de l'insuffisance aortique; la résultante de ces deux effets opposés qui se combinent, est la conservation de la valeur normale ou presque normale du retard du pouls."³

In the above paragraph François-Franck states in effect three distinct propositions:—

(1.) Aneurism of the ascending portion of the aorta increases the retardation of the pulse above the normal value.

(2.) Large insufficiency of the aortic valves diminishes the retardation of the pulse below the normal value.

(3.) The coexistence of aortic aneurism with large aortic insufficiency causes the retardation of the pulse to preserve the normal, or nearly normal, value.

The truth of the first proposition has been sufficiently attested by François-Franck's researches and my own.⁴ That aortic aneurism produces abnormal delay of the pulse is no longer a question, but remains as one of the great facts of clinical sphygmography.

In regard to the second proposition, François-Franck first insisted on theoretical grounds that the pulse

² Fothergill on Treatment, page 449.

³ Journal de l'Anat. et de la Physiol., t. xv. (Mars-Avril, 1879).

⁴ New York Medical Record, November 29, 1879.

¹ Treatise on Therapeutics, page 188. By Roberts Bartholow. Appleton & Co., New York, 1879.

would appear earlier than normal in the condition of large aortic insufficiency, and afterwards was able to produce clinical cases in proof of the correctness of the view. I also, before I had known of M. Franck's researches, had arrived by *à priori* reasoning at this same conclusion,¹ and now, in substantiation, I will place on record the following case:—

Cincinnati Hospital. Service of Dr. C. G. Comegys. Quinn, age thirty-two years. Diagnosis, aortic insufficiency, with hypertrophy of left ventricle.

In practical value this fact, I think, stands first among the list of clinical facts brought to light by the graphic method. It stands as the exponent of large aortic insufficiency. This condition and no other will produce abnormal precipitation of the pulse on the heart.

In this connection, however, it is important to observe that the phenomenon under consideration will only be manifest in complete (large) insufficiency of the aortic valves, — the condition in which they are per-

Graphic records taken March 27, 1880. No. 76 shows simultaneous tracings of the heart and right carotid, and No. 77 of the heart and left radial; the corresponding time in each also is shown. By very careful measurement² the delay of the carotid pulse on the heart is placed at one thirty-second of a second. The normal delay with pulse-rate at 103, as in this instance, would be about twice this interval. The cardio-radial interval is shown to measure one eleventh to one twelfth of a second. The normal interval would be not less than one eighth of a second.

This was a plain case of free aortic regurgitation, and the graphic records can only be regarded as demonstrating a greatly abbreviated cardio-arterial interval as the direct effect of the aortic insufficiency.

What is the explanation of this interesting fact? In large aortic insufficiency the base of the arterial column rests against the sides of the ventricle instead of against the aortic valves, and is advanced, causing rise of the pulse with the first movement of ventricular contraction. The result is due to shortening or near extinction of the presphygmic interval. This explanation is so complete and satisfactory to my mind, that I am surprised that François-Franck should, without reference to the presphygmic interval, attempt to explain the fact through the idea of increased rate of transmission of the pulse-wave under the conditions of aortic insufficiency.

The velocity of the pulse-wave in this case was rapid indeed, as shown in No. 78, where the time-difference between the carotid and radial pulses measures about one nineteenth of a second. But the greatest possible reduction of the cardio-carotid transit interval would signally fail to account for the great reduction here instanced of the cardio-carotid interval. Therefore the reduction must have been due to marked abbreviation of the presphygmic interval.

¹ Cincinnati Lancet and Clinic, March 22, 1879.

² My custom is to make and mark all measurements from the original slides, and then trust photographic transfer and skillful engraving for correct reproductions; and so accurate is this work, that usually the measurements from the reproductions correspond exactly with those from the originals.

manently open, and the beginning of ventricular systole finds no barrier between the blood in the ventricle and the blood in the aorta. The condition of partial insufficiency in which the valves permit reflux for a time after the beginning of diastole, yet finally close so that the beginning of systole finds the barrier intact, will not signal the phenomenon.

The third proposition is necessarily deducible from the two preceding. If aortic aneurism causes retardation of the pulse, and aortic insufficiency causes precipitation of the pulse, then the conjoint action of the two conditions would counteract each other, and cause the succession of the pulse on the heart to approximate more or less nearly the normal time.

The following case is given in exemplification of this interesting and important fact:—

Charles Gardner, aged fifty-six years, came under my notice in November, 1878, presenting the following symptoms and physical signs: General weakness, dyspnoea on exertion, inability to lie down, pains in chest, back of head, left side of neck, and left shoulder; pulse to fingers strong, full, infrequent, and regular; heart's impulse increased; area of cardiac dullness increased; pronounced double murmur heard over front of chest and in the back; the systolic accentuated at the right base, and conducted towards right clavicle; the diastolic also accentuated at right base, but propagated downwards over body of left ventricle; murmur but faintly heard at apex; at the site of the right subclavian pulse, above the clavicle, a pulsating tumor the size of a pigeon's egg; also visible, but less prominent, pulsation of left subclavian. Graphic observations with my compound sphygmograph were taken November 28th. Samples of the tracings are here faithfully reproduced. No. 79 shows the heart and right subclavian tumor; No. 80, the heart and right carotid; No. 81, the heart and left radial; and No. 82, the two radials, — each pair taken simultaneously, with accompanying time in fifths of seconds.

In December, 1879, I noted the case again. There had been progressive decline, the pains had become more severe and persistent. The action of the heart

and arteries had greatly diminished, and the pulsations become more frequent. The subclavian pulsations had subsided within normal limits. The evidences of enlarged heart, and the murmurs, with the same characters, continued; and in addition feeble undulations were perceptible in the second right interspace near the sternum, and in the first and second left interspaces. Also, the voice at times showed failure, and the power of deglutition was not always perfect. December 20th, tracings were obtained as shown in Nos. 83 and 84;

again, January 7, 1880, as shown in Nos. 85 and 86, and January 17th, as shown in No. 87.

The patient continued to decline, and died July 18, 1880, right hemiplegia setting in a day or two previous to his death.

At the post mortem were present Drs. Isham, Freeman, Barrows, and myself. The following are the notes: Pericardium contained several ounces of fluid. Left ventricle hypertrophied, walls fully one inch thick. Mitral valves, with the exception of slight thickening

towards the base, healthy; evidently fatty, competent during life. Aortic orifice enlarged; aortic valves thickened, corrugated, and calcified throughout; evidently wholly incompetent during life. Ascending aorta greatly dilated, atheromatous, and studded with calcareous matter. Transverse portion of arch the seat of a large aneurism. The sac projected from the anterior superior portion of the wall, and was attached to the upper portion of the sternum, the left clavicle, and first left rib, and pressed upon the trachea and œsophagus. It was full three inches in interior diameter; its walls were soft and flabby, about the thickness of normal left auricular parietes. The surface of the sternum, against which the tumor pressed, was eroded, and about half its thickness removed; also, there was erosion of the sternal end of the left clavicle and first left rib. A mass of laminated fibrine the size of a walnut occupied the distal portion of the sac and projected into the ascending aorta. The latter was also much dilated. The innominate and first part of the right subclavian were dilated to about twice the normal diameters. The right and left common carotids and left subclavian were about normal size. Both auricles, the right ventricle, and right-side valves were normal.

This case was complete, and a typical one of combined aortic aneurism and aortic insufficiency. The sphygmographic records were also complete. The latter we examine with interest, especially at present, with reference to the succession of the arterial pulses on the systole of the ventricle. Evidently aneurism, as in the present case, of the transverse portion of the arch, involving the origins of the three great arteries, namely, innominate, left common carotid, and left subclavian, entails the same effect on the succession of the pulses as aneurism of the ascending portion only; that is, the pulses are all abnormally delayed, and the symmetrical pulses of either side are equally delayed.

The tracings were taken and the successions measured with the greatest care; so the intervals expressed on the plates are reliable, and must approximate throughout very closely the true ones. The showings are as follows:—

November 28, 1878. No. 79. Pulse-rate 66-60. Cardio-subclavian interval .109 second (average).

No. 80. Pulse-rate 52-51. Cardio-carotid interval .112 second. December 20, 1879. No. 83. Pulse-rate 82-80. Cardio-carotid interval .0833 second.

No. 84. Pulse-rate 82-80. Cardio-radial interval .1666 second. January 7, 1880. No. 85. Pulse-rate 87-80. Cardio-subclavian interval .0833 second.

These measurements are sufficiently near the normal. In the variations of the time-differences, however, I noticed a bearing towards the longer time, and the average of all the observations, of which a portion only are shown, would indicate rather longer time than obtains in strictly normal conditions, but not sufficiently prolonged to exceed the healthy range or permit the counting of such delay as a definite indication. Then in this case the coexisting aortic aneurism and aortic insufficiency so counterbalanced each other that the retardation of the pulses on the heart was within the normal range.

According to the size and yielding nature of the aneurism must be the amount of delay impressed upon the pulse. An aneurism may be such in these respects as to cause a delay that would overbalance the precipitation entailed by the aortic insufficiency. On the other hand, the conditions may be reversed, and a large aortic insufficiency entail a precipitation that would

overbalance the delay caused by the aortic aneurism. However, when the two conditions coexist, the delay will never be so great as to definitely favor the wrong diagnosis of aortic aneurism alone, nor so small as to definitely favor the wrong diagnosis of aortic insufficiency alone. *If the diagnosis of aortic aneurism be certain, and the pulse, notably the carotid or subclavian, shows no abnormal delay, the conclusion is justified that aortic insufficiency coexists. If the diagnosis of large aortic insufficiency be certain, and the pulse, notably the carotid or subclavian, shows no abnormal precipitation, the conclusion is positive that aortic aneurism coexists.*

So in our case the ordinary signs and symptoms, at the first examination, were sufficient to determine the presence of large aortic insufficiency. Accepting this, the want of abnormal precipitation of the carotid and subclavian pulses enabled me to arrive at the diagnosis of coexisting aortic aneurism before there were any other indications of this condition.

As of interest relating to the localization of aortic aneurism: No. 82 shows the two radial pulses simultaneous, which proves that the aneurism was seated in the ascending portion anterior to the origin of the innominate, or that it was seated in the transverse portion so as to include the origins of both the innominate and left subclavian (and of course also the left common carotid). No. 87 shows the left radial as compared with the right femoral slightly delayed. This is the more usual normal relationship of these pulses, and proves that the aneurism was anterior to the origin of the left subclavian artery. Evidently, had the aneurism been below the origin of the left subclavian, the femoral would have shown delay as compared with the radial. No. 86 shows that the pulsation in the second left interspace succeeded the cardiac pulsation about .0714 of a second, and therefore aided the diagnosis of aneurism as located.

No. 80 is given to show a very ample and finely delineated radial pulse, and the rate of transmission of the pulse-wave between the subclavian and radial points under the conditions then existing in the case. The time-difference measures .108 second, which indicates a rather slow pulse-wave transmission. If this be compared with the carotid-radial interval in the case of Quinn, namely, .052 second, as shown in No. 78, a remarkable difference becomes apparent in the pulse-wave velocity of the two cases. The causes of this diversity are to be found in the dissimilar conditions which pertain to the respective cases.

A CASE OF ENCYSTED ASCITES SIMULATING OVARIAN DROPSY; OPERATION; DEATH; AUTOPSY.

BY AUG. F. ERICH, M. D.,

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THE following case, illustrating the difficulties of the differential diagnosis between encysted dropsy of the peritonæum and of the ovary, is published as a contribution to the statistics of those mistakes in diagnosis which to a wise physician are said to be "more instructive than twenty successes."¹

Annie Talbott, colored, aged twenty, unmarried, but

¹ D. Hayes Agnew. Address before the Pennsylvania State Medical Society, 1878.