AURICULAR FIBRILLATION:

SOME OBSERVATIONS AND DEDUCTIONS FROM A SERIES OF 400 CASES.

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OF all conditions treated at this hospital that of auricular fibrillation forms one of the most important, if not the most important group. It is found in no less than 25 per cent. of all patients admitted to the wards,[1] yet approximately 8 cases out of 10 are not fully recognised as such prior to admission, whereas even without polygraphic or electrographic help not more than 1 out of 10 should be missed.

The following observations are based on 400 cases of this condition, 110 of which were in-patients and 290 out-patients. The youngest was 16 years of age and the oldest 76.

In auricular fibrillation there is a lack of coördinate muscle contraction, and in its place individual fibres or groups of fibres contract incoördinately. Some of these individual stimuli are transmitted through the bundle system, and accordingly the ventricles contract unequally and irregularly and usually with increased rate. This is manifested not only in the complete irregularity of the heart, but also in the complete irregularity of the pulse. This irregular irregularity is the most important sign we have, and is almost pathognomonic of auricular fibrillation. The force of the individual beats varies, and it is usually found that some of the ventricle beats do not reach the wrist, hence it is necessary to take the heart-rate at the apex as well as the pulse, the latter being an index as to the number of efficient ventricle contractions. The greater the difference between the apex- and pulse-rates, the greater the number of inefficient beats, tending to ventricular exhaustion.

Differential Diagnosis.

There are two other conditions giving rise to an irregularly irregular pulse and which must be differentiated from auricular fibrillation, viz.: 1. Multiple premature beats, especially when auricular in origin. 2. Sino-auricular heart-block. In most cases of premature beats of ventricular origin there is an underlying regular rhythm, the contractions of which are practically always of equal force, recognised by continued auscultation at the apex, the premature beat usually being followed by a comparatively long pause. Frequently in these cases slight exercise will eliminate the premature beats, or at least sufficiently so for diagnosis, while in auricular fibrillation exercise almost invariably accentuates the irregularity. Rarely is it necessary to resort to polygraphic or electrocardiographic means for differentiation. In auricular premature beats, a rather infrequent condition, the diagnosis is made certain only by an electrocardiogram or polygram. Clinically, however, auricular premature beats may precede the onset of auricular fibrillation. Sino-auricular heart-block, on the other hand, is usually associated with an approximately normal or somewhat lower heart-rate, while in auricular fibrillation the rate is more often very rapid. Also in sino-auricular block the apex- and pulse-rates are the same, and there are long pauses in which the heart is completely silent. Sino-auricular block can be definitely differentiated from a slow fibrillation only by the electrocardiogram or polygram. It is, however, a comparatively rare condition. Another common irregularity, which ought not, however, to be confused with auricular fibrillation, is sinus arrhythmia. This is a regular irregularity in which the heart-rate usually increases during inspiration and decreases in expiration. This condition is very easily differentiated if watched for, and is frequently found in children.

Investigation of Pathological Condition.

Having diagnosed auricular fibrillation, the two essential points to consider are: (1) the pathological condition of the heart which led to auricular fibrillation; (2) the effect that fibrillation of the auricles will have on the circulation as a whole, and particularly on the left ventricle.

Post-mortem examination shows that there is almost always definite myocardial change, usually fibrosis in the wall of the auricles, interfering with the normal activity of those chambers. Furthermore, one has to determine (*a*) whether this myocardial change is confined to the auricles, or (*b*) whether the ventricles also are affected. Naturally, if the ventricles are involved, especially the left ventricle, the onset of fibrillation is a much more serious matter, because it is the left ventricle that is mainly responsible for the circulation.

In mitral stenosis there is very little doubt but that the left auricle is greatly handicapped, being possibly the only part of the heart muscle affected. There is usually dilatation and hypertrophy followed by muscle failure, ending in degeneration. The degenerated muscle becomes hyper-irritable.[2] When the irritability of the various groups of auricular fibres becomes greater than at the normal pace-maker, auricular fibrillation sets in. Occasionally there is a localised myocardial involvement of the auricles secondary to pericarditis, or, as is even more unusual, auricular damage alone may remain following a general infection.

After the onset of auricular fibrillation the ventricle-rate is a matter of great importance, for the more rapid the rate the more will it tend to produce myocardial exhaustion. The ventricular rate may be greatly influenced by emotion. This is shown clinically by the fact that the practical normal heart-rate of a patient fully under the influence of digitalis may be greatly increased by excitement or fright. The ventricular rate is also increased by exertion, and usually out of proportion to the amount of exertion, especially when there is marked muscle involvement of ventricle. If one compares the effect of emotion and exercise on the ventricular rate, one frequently finds that the heart-rate may go up much higher on slight excitement than after exertion—e.g., in one case a patient without continuous fibrillation was easily made to fibrillate by the breaking of an electric bulb or by the loud slamming of a door, but would not fibrillate on walking up five flights of stairs. Another patient, on the other hand, was easily made to fibrillate by either excitement or exertion, but would soon return to the normal rhythm.

Treatment.

The main object to be achieved when treating auricular fibrillation is to get the heart-rate at the level where it produces an adequate circulation without undue ventricular work, which is usually between 65 and 80 a minute. This is best produced by giving digitalis in sufficiently large doses to bring the heart-rate down to approximately 50 beats a minute, when the drug is stopped. The digitalis is then again started, but in a smaller dose, and gradually increased until the desired rate is procured. The resulting dose is continued indefinitely, with changes only as varied circumstances may require. Occasionally it is desirable to keep the rate nearer to 90 a minute, but here the prognosis is worse.

The great therapeutic agent in auricular fibrillation, therefore, is physical, mental, and cardiac rest. Cardiac rest is produced by the digitalis group, resulting in partial paralysis of the bundle of His, less impulses being transmitted to the ventricles. Associated conditions and symptoms, as hyperchlorhydria with wind in stomach, must also be dealt with.

[1] Patients considered suitable for admission include those suffering from acute valvular disease, chronic valvular disease, myocardial disease, advanced arterio-sclerosis, including secondary kidney involvement, advanced Graves's disease with myocardial involvement, acute infective endocarditis, aneurysm, &c.

[2] The normal cardiac contraction starts at the sino-auricular node at the most irritable area.

Analysis of Cases.

Turning to the actual detailed analysis of the 400 cases of auricular fibrillation, we shall consider: (1) Ætiology: (*a*) age- and sex-incidence, (*b*) pre-existing disease; (2) symptoms, relative frequency; (3) associated conditions; (4) X ray appearances, measurements.

1. *Ætiology.*—(*a*) Age and sex incidence. Of the 400 patients with auricular fibrillation examined 220 were males and 180 females, in the proportion, therefore, of 11 males to 9 females. From this it is apparent that one sex cannot be considered very much more susceptible to this condition than the other sex. Going into the ages of these patients, 265 of the 400 cases, or 66 per cent., occurred after 40 years, the largest number 112, or 28 per cent. of the total, occurring between 41 and 50 years. (See Table I.) (*b*) Pre-existing disease. Rheumatic fever is by far the commonest antecedent disease in auricular fibrillation, being found in no less than 123 (8 of which also had chorea), or 35 per cent. of the 348 cases in Table II. If to this we add the 14 cases of chorea alone, the cause of which is strongly affiliated with that of rheumatic fever, we have 39 per cent. of the total. On the other hand, there was a history of no previous illness in 66, or 19 per cent. of the total, a very appreciable factor and a distinctly important point in the matter of causation. Of the 46 cases giving a history of influenza, the actual cause is debatable, the severe forms of influenza being, however, of definite importance in cases with heart strain or where the heart muscle has been previously affected. Scarlet fever is not an uncommon disease in childhood; it is doubtful whether it is a cause of cardiac disease. A prominent observation in this series is that Graves's disease was found in ten cases, all of which were myocardial only.[3] The Wassermann reaction was taken in only definitely suspected cases and not as a routine, there being seven positive, in five of which the myocardium alone was involved. Apart from the subject of auricular fibrillation Table II. shows two outstanding facts which might usefully here be mentioned: (*a*) Rheumatic fever is much more likely to involve valves or valves and myocardium—such being the case in 109 out of 123 patients—than myocardium alone; (*b*) of the 14 cases of chorea (without rheumatic fever) all involved valves. In no case was the myocardium alone affected.

TABLE I.—*Age and Sex.*

—	Sex.	Age. 16–20	21–30	31–40	41–50	51–60	61–70	71–76	Total.
Out-patients	M.	3	30	34	42	41	12	0	162
	F.	4	18	26	37	33	5	0	123
In-patients...	M.	0	14	14	15	9	1	5	58
	F.	1	7	14	18	12	5	0	57
Total		8	69	88	112	95	23	5	400

2. *Symptoms, relative frequency.*—There are no symptoms pathognomonic of auricular fibrillation, nor are there any that are much more suggestive of this than of other cardiac conditions. In the 400 cases used in this series (all in-patients) there were no leading questions asked, and the symptoms are those given by the patients practically unaided. It is very apparent here (Table III.) that the commonest symptom in heart failure is dyspnœa and that this symptom is found as often when fibrillation is absent as in its presence. The next two symptoms commonly met with are pain (under or across the heart) and palpitation. Pain is somewhat more common in the absence of fibrillation, while palpitation is found somewhat more often in fibrillation cases. Among the less usual symptoms, cough is twice as common, and insomnia four times as many fibrillation as in non-fibrillation cases. While nervousness is objectively common, it is but seldom complained of as a symptom. Subjective feeling of irregular beating of the heart has been mentioned as a common symptom, but in this series, shown in Table III., it is comparatively rare.

TABLE II.—*Pre-existing Disease.*

(A) Rheumatic fever. (B) No previous illness. (C) Influenza. (D) Rheumatism. (E) Chorea. (F) Scarlet fever. (G) Graves's disease. (H) Enteric fever. (I) Small-pox. (J) Diphtheria. (K) Syphilis.

—	(A)	(B)	(C)	(D)	(E)	(F)	(G)	(H)	(I)	(J)	(K)	Total
Mitral disease	92	20	20	17	13	23	0	3	0	2	1	193
Myocard. disease	16	38	25	7	0	15	10	6	3	2	5	127
Aortic and mitral ...	11	5	1	4	1	0	0	0	0	0	1	23
Aortic	4	1	0	0	0	0	0	0	0	0	0	5
Total... ...	123	66	46	28	14	38	10	9	3	4	7	348

[3] "The Heart in Graves's Disease," by J. Strickland Goodall, Practitioner, July, 1920.

TABLE III.—*Symptoms.*

(A) Dyspnœa. (B) Pain. (C) Palpitation. (D) Vertigo. (E) Cough. (F) Weakness. (G) Insomnia. (H) Nervousness. (I) Choking feeling. (J) Fainting (unconscious). (K) Irregularity.

—	(A)	(B)	(C	(D)	(E)	(F)	(G)	(H)	(I)	(J)	(K)
(1)	182	72	82	40	26	24	16	16	6	8	6
(2)	183	106	70	48	13	21	4	3	15	2	3

(1) = 200 cases of fibrillation.
(2) = 200 cases without fibrillation.

3. *Associated conditions.*—Of 359 cases: (*a*) 133, or approximately 37 per cent., had definite mitral stenosis; (*b*) 132, or approximately 37 per cent., appeared to be primary myocardial; (*c*) 65, or 18 per cent., had mitral regurgitation; (*d*) 24, or 6·7 per cent., had both mitral and aortic involvement; (*e*) 5, or 1·4 per cent. had aortic involvement only, including 2 cases with double aortic. From this one would infer that myocardial involvement alone is found associated with auricular fibrillation practically as frequently as pure mitral stenosis, although some authorities have stated that mitral stenosis is associated with most cases. If, however, we add the mitral and aortic cases having mitral stenosis, of which there were 10 out of 24, we have 143 mitral stenosis cases as against 132 myocardial, or only 8½ per cent. more in a total of 359 cases. Aortic disease alone accompanied auricular fibrillation in only 5 cases and is therefore comparatively unusual, while both mitral and aortic involvement occurred in 24 cases, or 6·7 per cent., of the total. (Table IV.) Group (*c*) consists of patients having only a distinct blowing systolic murmur at the apex transmitted to the axilla. Since most patients with auricular fibrillation are first seen at the hospital after fibrillation has commenced, there is no certain way of recognising those cases which showed a presystolic murmur before

TABLE IV.—*Associated Conditions in 359 Cases.*

—	Age. 16–20	21–30	31–40	41–50	51–60	61–70	71–76	Total
Myocardial	0	13	21	28	48	18	4	132
Double mitral ...	4	19	20	25	13	1	0	82
Mitral regurg. ...	2	5	19	14	20	4	1	65
Mitral stenosis ...	0	14	15	19	3	0	0	51
Mitral and aortic	1	6	4	7	6	0	0	24
Aortic	0	1	1	3	0	0	0	5
Total	7	58	80	96	90	23	5	359

—	Mitral stenosis and double mitral.	Myocardial.	Mitral regurgitation.	Aortic and mitral.	Aortic.
No. of cases ...	133	132	65	24	5
Per cent. ...	37	37	18	6·7	1·4

the onset of this change of rhythm.[4] If the second pulmonary sound is reduplicated or markedly accentuated and the electrocardiogram shows a right-sided preponderance, mitral stenosis is more likely to be present. A diastolic murmur at the apex not of aortic or exocardial origin is, of course, sufficient to diagnose stenosis. Also, considering the very large size of the heart in auricular fibrillation, especially of the auricles, it is apparent that some of the mitral regurgitation murmurs, in spite of all the signs of what is considered an organic lesion, are due to relative mitral regurgitation, as was the case in a recent post-mortem examination at this hospital. These cases would accordingly come under the heading of those associated with myocardial diseases.

4. *X ray appearances; measurements.*—All patients coming to this hospital are X rayed as part of the routine examination. With the tube 6 ft. away from the object the rays are practically parallel, and the image is then the same size as the object. For convenience in actual practice, however, it is desirable to place the tube very much closer to the patient and to allow for the distortion, and as used at this hospital the image on the screen is $\frac{1}{4}$ in. to $\frac{1}{2}$ in. larger than the actual size of the heart. The size of the normal heart varies with age, sex, mode of life, &c., of the individual. Working on healthy hearts of young men under 30, Strickland Goodall showed that the average of the transverse diameters in a series of cases was $5\frac{1}{4}$ in., and this figure is taken as a standard in Table V.

TABLE V.—*Transverse Diameter of Heart.*

Fibrillation.	Without fibrillation.
One case with diameter to $5\frac{3}{8}$ in.	33%
16% with diameter of $5\frac{1}{2}$ to 6 in.	26%
42% ,, ,, $6\frac{1}{8}$ to 7 in.	26%
24% ,, ,, $7\frac{1}{8}$ to 8 in.	11%
16% ,, ,, $8\frac{1}{8}$ to 9 in.	2%

The sign most commonly found in auricular fibrillation, next to irregular irregular rhythm, is enlarged heart, particularly the auricles being so. In the 117 cases detailed here, where the percussion figures were corroborated by X ray, and in which the actual measurements are given, only one heart showed a diameter of $5\frac{3}{8}$ in. or less. Therefore in practically every case the diameter of the heart was above the normal limit. Over 80 per cent. of the fibrillation cases showed a heart diameter of 6 in. or more, as against 40 per cent. in non-fibrillation cases, while 40 per cent. showed $7\frac{1}{8}$ in. or more, as against 13 per cent. in non-fibrillation cases. When in auricular fibrillation the heart is beating rapidly the apex-rate as a rule differs from the pulse-rate, and it is necessary to count both. (This is, therefore, a third sign to be looked for.) Since the apex gives the actual ventricle-rate, while the radial pulse gives only the rate of those beats that are sufficiently powerful to force the blood through the body, a rapid apex-rate leads to earlier ventricular exhaustion. This sign is of great importance in prognosis. As a general rule the greater the difference between the apex-rate and the pulse-rate, especially after exercise or emotion, the worse the prognosis.

The three signs of auricular fibrillation to be emphasised are thus: (1) Complete irregularity of the heart. (2) Increase of the cardiac diameter. (3) A difference in apex- and pulse-rate.

Prognosis.

Paroxysmal attacks of tachycardia occasionally prove to be attacks of auricular fibrillation, and may last only a few minutes to several days and then return to normal. There may be but one such attack in the patient's life, or there may be several, or, as is most usual, once fibrillation starts it continues the rest of the patient's life. If the ventricle, particularly the myocardium, is in good condition, and the rate has been kept down sufficiently to avoid exhausting it (at or near 70), whether by nature, rest, or digitalis, the auricles will continue to fibrillate until they become completely exhausted. The auricle waves in the electrocardiogram then become very small and fine, and ultimately disappear almost entirely. In other words, the auricles now become passive chambers and take very little active part in the cardiac cycle.[5] The ventricles, however, may continue to receive the impulses from the auricles through the bundle of His. Sometimes it is necessary to use a continuous drum over a comparatively long time before any auricle waves can be found. This condition is differentiated from a complete heart-block in that (*a*) the waves in the auricles will be found, if carefully looked for, usually as fine waves, however, while in a true complete heart-block practically normal P waves are present; (*b*) the rhythm of the heart continues irregularly irregular, while in a complete heart-block the ventricle usually takes on a regular rhythm; (*c*) the rate is usually higher than in the heart-block. It is thus apparent that the impulse starts in the sinus or some part of the auricle, that it is transmitted through the auricle and bundle of His to the ventricles, and that the auricles have almost completely lost their power of contractility. This state of affairs, though found in long-standing cases of auricular fibrillation, from a practical point of view is less inconvenient to the patient than when the auricles are actively fibrillating, and when the ventricle in spite of treatment continues to beat at a very rapid rate, or when the rate is greatly increased by the least excitement or exertion.

Summary and Conclusions.

1. The three most constant and diagnosable signs found in auricular fibrillation are: (1) the irregular irregularity in rhythm, (2) enlarged transverse diameter of the heart, (3) a difference in the apex- and pulse-rate.

2. An irregular irregular heart rhythm is almost pathognomonic of auricular fibrillation. It must be differentiated from that found in (*a*) multiple premature beats, especially those of auricular origin, and (*b*) sino-auricular heart-block.

3. There are no symptoms pathognomonic of auricular fibrillation, nor are there any much more suggestive of this than of other cardiac conditions.

4. A diagnosis of auricular fibrillation having been made it is essential to consider (1) the pathological condition of the heart which led to this condition, and (2) the effect that the fibrillation will have on the circulation as a whole, and especially on the left ventricle.

5. It is desirable to decide whether there is myocardial change in the auricles alone, or whether the ventricles also are affected. If the latter is the case, the prognosis is distinctly worse.

6. The main object to be achieved when treating auricular fibrillation is to adjust the rate of the ventricles so as to produce an adequate circulation without undue ventricular work.

7. The great therapeutic agent is rest—physical, mental, and cardiac.

[4] In these cases the murmur was due to the forcing of the blood by a more or less hypertrophied left auricle through the narrowed mitral opening. In auricular fibrillation this action is absent and hence the associated mitral stenosis both as a pathological condition and as the possible underlying cause of auricular fibrillation is often missed. Clinically, however, some patients with auricular fibrillation still give what appears as a presystolic murmur on auscultation. Leaving out of consideration Flint's murmur, a presystolic murmur rarely heard at the apex in aortic regurgitation, these can be divided into two types: 1. Those in which the presystolic is not a true recognised presystolic, but in which the diastolic murmur runs through the period of diastole into the following systole, the heart rest period being short. It is really, then, a diminuendo murmur instead of a crescendo murmur. When the rest period is long, on the other hand, the diastolic murmur ends as usual and there is a distinct interval previous to the following systole. 2. Among my cases there were two in which there was a distinct and true definite presystolic murmur not a prolonged diastolic, and in which there was also fibrillation. There was also a presystolic thrill palpable. The murmur and thrill were present only occasionally and of varying force. It was quite clear, however, that these did not have any relationship to the period between diastole and the following systole. It may be inferred that there is a definite stage between the normal auricular contraction and a condition of auricular fibrillation. Though the impulses are generated there is not a definite complete contraction preceding and in normal relation to the ventricular contraction, but there are a sufficient number of groups of fibres contracting simultaneously to produce a distinct murmur on forcing the blood through the stenosed mitral valve at or near the time when the ordinary presystolic murmur of mitral stenosis would be heard were the heart not fibrillating. If the presystolic murmur had not varied one might have inferred that it was due to occasional periods of normal cardiac rhythm.

[5] It is interesting to note in this connexion that, according to some authorities, 40 per cent. of the ventricular filling in the normal heart is performed by the auricles.

8. The commonest antecedent disease is rheumatic fever. Rheumatic fever or chorea was found in 39 per cent. of the total number questioned, while 19 per cent. of the total gave a history of no previous illness. Graves's disease very definitely leads to fibrillation.

9. Primary myocardial involvement was found in as many cases as were associated with pure mitral stenosis. The other conditions in the order of their frequency were mitral regurgitation, both mitral and aortic disease, and lastly, aortic disease alone.

10. Fibrillation of the auricles can continue until the auricles are completely exhausted and become practically a passive chamber. The contractions may then come on only occasionally and be very slight, but impulses will still be transmitted to the ventricles.

11. There is no predisposition in either sex towards fibrillation.

12. As the pulse-rate usually does not give the ventricular rate of contraction, it is necessary in all cases of auricular fibrillation to count the apex-rate as well.

13. A murmur presystolic in time can be heard in some cases of auricular fibrillation associated with mitral stenosis, but this is usually due to a diastolic murmur running into the following systole when the rest period is short.

14. 25 per cent. of all patients admitted into the wards of the National Hospital for Diseases of the Heart have auricular fibrillation.

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PERCENTAGE AND FRACTIONAL DILUTIONS.

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IN THE LANCET of Jan. 29th, p. 223, Dr. William Fletcher gives formulæ for preparing percentages of fluids and for fractional dilutions. Professor J. W. W. Stephens and Dr. S. R. Christophers gave 12 years ago[1] a simple formula for obtaining percentage dilutions. It shows the amount of diluting fluid which it is necessary to add to 1 volume of fluid of percentage x in order to reduce that volume to the desired percentage y. The formula is $\frac{x}{y} - 1$; where x represents the original percentage and y the desired percentage. Thus, to dilute a solution from 20 per cent. to 5 per cent. add to each volume of solution $\frac{20}{5} - 1 = 3$ of the diluting fluid.

The following formula can be used where it is necessary to prepare specified volumes of lower percentage or higher dilution from higher percentages and lower dilutions respectively.

Percentage dilution.—To prepare a specified volume z of a y per cent. solution from an x per cent. solution, take of the latter $\frac{z \times y}{x}$ volume and make up to z volume with diluting fluid.

Example.—To obtain 12 c.cm. of a 24 per cent. solution from an 80 per cent. solution, take of the latter $\frac{12 \times 24}{80} = 3{\cdot}6$ c.cm., and make up to 12 c.cm. with diluting fluid.

Fractional dilution.—To prepare a specified volume z of a $\frac{1}{y}$ dilution from a $\frac{1}{x}$ dilution, take of the latter $\frac{z \times x}{y}$ volume and make up to z volume with diluting fluid.

Example.—To obtain 60 minims of a $\frac{1}{100}$ dilution from a $\frac{1}{10}$ dilution, take of the latter $\frac{60 \times 10}{100} = 6$ minims, and make up to 60 minims with diluting fluid.

[1] Stephens and Christophers, 1908: Practical Study of Malaria and Blood Parasites, p. 441.

Clinical Notes:

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

A NOTE ON NON-INFECTIVE THROMBOSIS OF PELVIC AND ABDOMINAL VEINS.

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INTRAVASCULAR clotting (thrombosis) may occur under imperfectly understood conditions; this is particularly true of non-infective thrombosis. The following case is illustrative; the cause is obscure, the termination unusual, and the difficulty of diagnosis from the initial symptoms is apparent.

M. D., aged 59, weight 15 st. 2 lb., height 5 ft. 11 in. Always had good health; no venereal disease; led an active athletic life; wore a double inguinal truss since April, 1918. No serious illness up to August, 1918, when he had an operation for appendicitis. Recovery appeared to be complete and satisfactory. He resumed military duty six weeks after the operation, and remained in excellent health up to April, 1919, when he had a severe attack of influenza affecting throat and chest. After four weeks he made a satisfactory recovery, and was in usual health up to Nov. 5th, 1919.

On the 5th he had much abdominal distension, slight abdominal pain, pain in area of prostate gland, congestion of penis and a very thin urine stream, took considerable time to empty the bladder; the congestion was less and the bladder was emptied with more ease in the recumbent position than when standing up. On the 6th, during a round of golf, he felt an acute pain over the left sacro-iliac joint. The next day the pain was more acute in the back, and he had a feeling of great abdominal distension, and some nausea and vomiting. On examination a fibrositic nodule over the left sacro-iliac joint, some tenderness over the gall-bladder, and slight œdema of right foot were found. Temperature normal, heart and lungs normal, pulse 68. No cause could be found for the distension and œdema, and there was no notable point in the history of the case except that he had been taking an aperient dose of Epsom salts in a glass of hot water before breakfast since the attack of influenza in April, 1919. No definite diagnosis could be made.

On the 8th he was seen by surgeon in consultation. Much abdominal distension. Dull continuous pain in abdomen; pain in back more acute; tenderness on pressure over the external abdominal rings. Examination per rectum revealed nothing. Condition remained the same up to the 11th, when in addition to the abdominal pain and distension acute pain commenced in right thigh, and pain over the femoral vessels in Scarpa's triangle. No tenderness on pressure. Œdema of both feet. On the 12th again seen in consultation. Abdominal pain and distension more marked. Temperature 99° F. Urine had trace of albumin, and on centrifugalisation a few red blood cells were seen. An elastic swelling found in right lumbar region. On the 14th left thigh became painful. Much tenderness in Scarpa's triangle. Temperature rose to 101·2°. On the 16th right thigh and groin more painful, and much œdema of both legs from hips to toes. No œdema of back. Temperature varied from 101·2° to 99·6° during the day.

On the 17th examination per rectum revealed nothing further than some ballooning and emptiness. Urine loaded with urates. Fluid appeared in tunica vag. testis. Acute pain to right of umbilicus. Temperature 99°. Œdema of legs increased, much pain in legs, no tenderness. On the 20th he was seen by surgeon in consultation. He found "a definite 'pappy' or semi-elastic swelling in right lumbar region, felt bi-manually in neighbourhood of right kidney." Discussion ensued on the possibility of the condition being inflammatory (diverticulitis). Temperature varied between normal and 99·8°. Castor oil emulsion by the mouth, and simple enemas were given daily; good result, and the tumour in lumbar region rapidly diminished in size.

By the 22nd condition improved: abdominal pain ceased; pains in both legs became less; œdema remained about the same and confined to legs; no œdema or swelling of back, and not higher than Poupart's ligament. Abdominal distension still continued. On the 23rd pains in legs and much abdominal distension, with pain and tenderness in right half of abdomen. Temperature 99·8°. On the 24th griping pain in abdomen caused restlessness, but less aching in legs, and less pain and tenderness in Scarpa's triangles. Temperature normal or subnormal. Blood examination: hæmoglobin, 92 per cent.; red cells, 5,200,000; white cells, 9100. Urine: minute trace of albumin, centrifugalised deposit showed red blood cells in small numbers.

On the 29th consultation with the two surgeons. Operation was discussed and the decision come to not to operate. Discussion followed on the probability of the tumour being malignant and partially occluding the inferior vena cava. From this date up to Dec. 4th condition remained about the same, œdema tending to subside. Pain and tenderness remained along the femoral vessels in both legs, in Scarpa's triangles, and Hunter's canal. Weight was found to be increased by 2 lb., accounted for by the fluid in the tissues of the legs. On the 12th patient was partially dressed and walked a little in bedroom; much aching in both legs followed and œdema increased. Notwithstanding this, he got up each day for varying periods and steadily improved from this date.

On Jan. 7th, sixty-fourth day of the illness, consultation of physicians and surgeons. Tumour in right lumbar region much smaller and difficult to locate. Change of air and scene recommended. Arrived Hyères, France, Jan. 31st, 1920, and returned May 7th.