is that radium treatment serves a very useful purpose in certain types of nerve trauma and in certain stages in the repair of nerve injuries. The conclusions to be drawn from this work with radium in the treatment of war injuries affecting nerves can be summarized as follows:

1. Radium treatment cannot benefit gross nerve lesions; here operative treatment is indicated, to unite severed nerves or free them from dense scar tissue.
2. After a nerve operation, or after milder degrees of nerve trauma, it would appear to aid and to hasten the return of function in a limb.
3. It improves the nutrition in the area supplied by injured nerves.
4. It may be useful as an aid to diagnosis, and in certain cases will indicate or contraindicate the necessity of operation.
5. It is a valuable adjunct to other forms of treatment.

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ON POISONING BY ARSENOBENZOL COMPOUNDS USED IN THE TREATMENT OF SYPHILIS.

BY

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BEFORE considering the toxic action of arsenobenzol compounds it will be convenient, since the essential pathological processes appear to be closely similar in either case, to refer to a paper² published in 1918 which dealt with the pathology of another group of cases of poisoning in which what is termed toxic jaundice is a characteristic symptom, and in which there are found after death an infiltration of the liver and kidneys with fat, and in some cases an acute atrophy of the former gland. Six poisons, all of which have a special affinity for fats, were referred to as belonging to this group; three of them—chloroform, ether, and tetrachlorethane—are active fat solvents, and the other three—phosphorus, dinitrobenzene (D.N.B.), and trinitrotoluene (T.N.T.)—are readily soluble in fats.

The essential process of poisoning in these cases appears to be as follows: The poison, whether swallowed into the stomach or inhaled into the lungs, or fixed in the fatty constituents of the skin and absorbed slowly therefrom—as may be the case with one or other of the six—becomes fixed in the food fat of the circulating blood. This fat, with the poison fixed in it, is carried to the liver for the purposes of normal metabolism, there to be transformed by a process of desaturation of the fatty acids into the "available organic fat" which is believed to be the principal source of energy for the work of various active tissues. The destructive action of the poison thus concentrated in the liver is such that the functional activity of the gland is impaired, with interruption of the process of desaturation of the food fat and, presumably, disorder of function in other directions. The normal metabolism of food fat being checked, engorgement of the liver with fat and a condition of lipaemia, caused by an accumulation of unchanged food fat in the blood, result. In experimental poisoning of the cat and rabbit with trinitrotoluene it was found that considerable quantities of the surplus fat were excreted by the kidneys, being readily demonstrable in the urine. And so the kidneys become engorged with fat in process of excretion; and, as with the liver, there is disturbance of the functional activity of the gland as a result of the direct action of the concentrated fat-fixed poison. The investigation led to the conclusion, therefore, that the fatty changes which occur in the liver in such cases as those of delayed chloroform poisoning and poisoning by trinitrotoluene represent an infiltration with food fat which the gland is unable to transform, and that the similar condition in the kidney is caused by an infiltration with unchanged and useless fat which is in process of excretion from the body. The changes do not represent, as had been supposed hitherto, a fatty degeneration of the parenchyma cells of the gland. The first effect of the direct action of the concentrated poison on the liver, as seen in the earlier stages of experimental poisoning with trinitrotoluene, is a perivascular round-cell infiltration of the connective tissue along the course of the portal circulation. In the areas occupied by the migrating cells the first microscopic evidence of interrupted metabolism is found in the presence of

minute globules of fat which apparently have passed through from the portal capillaries. At a later stage, in human cases, the gradual accumulation of this fat produces the gross fatty infiltration which is the most obvious feature of the group of cases of poisoning. In the more prolonged poisoning of munition workers with trinitrotoluene the tracts of round-cell infiltration become replaced in places by dense fibrotic tissue, so that the parenchyma cells, already damaged by the direct action of the fat-fixed poison, undergo a further destructive degeneration which is secondary to the cirrhotic change.

The fatty changes in the liver and kidneys in fatal cases of poisoning by arsenobenzol compounds appear to be essentially identical with those just described. There is an infiltration of the liver with unchanged fat, resulting from an interference with fat metabolism caused by the direct action of the poison on the tissue of the gland. With this there is an apparently consequent engorgement of the kidney with surplus unchanged circulating food fat. In poisoning with the arsenical compounds, whilst there is not a ready explanation of the concentration of the poison in the liver which in the other cases is afforded by an obvious special affinity for fats, it appears to be possible that some similar fixation of the poison in the fat does in fact occur.

At a time when an investigation into the pathology of poisoning by T.N.T. amongst munition workers was nearly completed, three deaths amongst patients who had been under treatment with arsenobenzol compounds (novarsenobillon and kharsivan) came under observation towards the end of 1917, and were apparently the first cases of the kind recognized in this country. Many other cases followed, but for various reasons it was inexpedient to publish any details at the time; and in the paper quoted on the action of certain poisons which affect fat metabolism only a very brief allusion was made to the subject. The close resemblance between these cases and cases of delayed poisoning by trinitrotoluene was referred to; and it was stated that in an experiment in which a rabbit had been poisoned with arsenobenzol changes in the liver had been produced which were similar to those found after the experimental administration of trinitrotoluene. It had been found that in the rabbit the first appearance of fat was not in the parenchyma cells of the liver, but in areas of round cell infiltration which appeared along the course of the portal capillaries. Also there was evidence of the elimination of fat by the kidneys.

The following table (I) sets out the facts, so far as they were ascertainable, as to the dosage and time of appearance of toxic symptoms in 7 cases which occurred in the practice of six different hospitals, and in which it was possible to make a microscopic examination of the liver and kidneys after death. The table includes similar details which were obtained in an investigation of the circumstances of 9 cases treated in yet another hospital, in which the appearance of jaundice was followed rapidly by death in patients who were either under treatment with kharsivan or for whom the course had already been completed. Nothing is known as to the actual condition of the organs in these last 9 cases.

For purposes of comparison with these 16 cases two other series of cases recorded respectively by Professor Stuart McDonald⁴ and by Drs. Strathy, Smith, and Hannah⁵ may be referred to. The former series included 5 fatal cases which had been treated with "salvarsan," combined with the usual intramuscular injections of mercury. It is stated that in all of the cases the jaundice, which appeared at first without any serious general symptoms, was followed after an interval of from two to eight days by symptoms of severity, increasing jaundice being accompanied by haematemesis, active delirium, and coma, with death in from one to four days. Dr. Strathy and his colleagues refer to a series of 58 cases treated in a military hospital, in which unfavourable symptoms of one kind or another occurred either during or after a course of treatment with either arsenobillon or kharsivan or galy, combined in each case with intramuscular injections of mercury. Amongst the 58 cases there were 47 in which there was jaundice, and of these 8 died. The number of injections given in the fatal cases varied from four to eleven. The largest amount of the drug given in a fatal case was 6.95 grams and the least amount 2.2 grams. The average interval between the last injection and the onset of symptoms in a fatal case was forty-one days, the longest interval being forty-eight days and the shortest eighteen days. The average interval between the onset of symptoms and death was five days, the longest interval eleven days, and the shortest two days.

Regarded from the clinical point of view the 29 cases, including those recorded by Drs. McDonald and Strathy, which occurred in the practice of nine different hospitals, together with other published cases, appear to fall into

TABLE I.—Sixteen Deaths occurring during the Course of, or after, Treatment with Arsenobenzol Compounds.

Case.	Dose in Grams.	No. of Injections.	Duration of Period of Treatment.	Interval between last injection and first appearance of Toxic Symptoms.	Interval between appearance of Toxic Symptoms and Death.
1	3.1	8	48 days	44 days	4 days
2	2.8	7	42 "	34 "	3 "
3	3.0	6	89 "	21 "	3 "
4	5.4	6	37 "	42 "	15 "
5	Full course	—	Some weeks	Some weeks	5 "
6	Full course	—	" "	" "	5 "
7	0.9	2	15 days	A few days	2 or 3 days
8	—	7	50 "	15 days	4 days
9	—	7	43 "	10 "	4 "
10	—	7	127 "	16 "	12 "
11	—	6	36 "	13 "	1 day
12	—	6	36 "	8 "	3 days
13	—	6	35 "	17 "	4 "
14	—	4	22 "	9 "	5 "
15	—	3	15 "	9 "	8 "
16	—	—	46 "	9 "	1 day

three groups, according to the rapidity with which definite toxic symptoms follow administration of the drug. There are (1) a few acute cases in which the symptoms appear rapidly after only two or three injections have been given; (2) subacute cases in which symptoms appear at a later date, either during the course of treatment or soon after its completion, and in either case within about ten days after the last injection; and (3) cases of delayed poisoning, in which the symptoms do not appear until from two to seven weeks after the last dose of a completed course, the patient having been in apparently good health during the interval. The factors of dosage and time appear, however, to have quantitatively but little determining influence on the final result. Whether the symptoms appear rapidly after a few doses—as in Case 7, in which only two doses of 0.45 gram each were given—or whether they are delayed for six weeks after the completion of a full course of treatment—as in Case 1, in which 3.1 grams of the drug had been given by eight injections—the final result is the same in a large majority of the fatal cases: jaundice, hæmatemesis, delirium and coma, with death within three, four, or five days.

The Process of Poisoning after Administration of Arsenobenzol Compounds.

The pathological problem presented when these cases were first recognized was one of considerable interest. As the results of earlier investigations it was stated in my previous paper² that it appeared to be probable that the toxic effect of the organic arsenic compounds was influenced materially by the benzene constituent—a conclusion which has been strengthened by subsequent observations. Since then, however, others have suggested that the cases represent an arsenical poisoning pure and simple. Dr. McDonald, on the other hand, considers that the changes in the liver are the effects of an infection by a bacillus of the typhoid-colon group, which he isolated from some of his cases after death.

There would appear at least to be a *prima facie* case for regarding this infection as being the new factor which, acting on livers previously damaged by syphilis, and possibly arsenic plus mercury, has completed the damage to the liver cells and allowed autolysis of the tissue, which appears to be the essential change, to occur.⁴

It being difficult, in view of the general course of these cases, to accept a theory which presupposes a bacterial infection as a determining cause of the illness, which is regarded rather as being simply the direct result of a drug poisoning, some experiments have been carried out in order to determine if possible whether the benzene constituent has any influence in aggravating the toxic effect of the

arsenic. The experiments have included the injection of rabbits subcutaneously with (1) arsenic oxide (As_2O_3), (2) with the benzene constituent (ortho-amido-phenol) with which the drugs are compounded, (3) with arsenobillon, and (4) with kharsivan.

Experiments with Arsenic Oxide.—Altogether 9 rabbits have been injected with toxic doses of arsenic oxide, and, except for Rabbits 1 and 9, the liver and kidneys were examined microscopically after death in every case. The arsenic was injected daily, in approximately equal doses, as shown in Table II.

TABLE II.

No.	Weight of Rabbit in Grams.	No. of Doses.	Average Dose Injected Daily, in Milligrams.	Total Amount in Milligrams.	Result.
1	4,000	1	—	16	Died within 18 hours
2	1,420	4	13.1	52.5	Died on fourth day.
3	3,250	3	10	30	Killed under chloroform on third day. Died on fifth day.
4	3,750	5	9.6	48	Died on fifth day.
5	1,190	5	6.6	33	Died on fifth day.
6	1,510	17	5	85	Died on 17th day; weight, 1,360 grams.
7	1,900	17	5	85	Died on 17th day; weight, 1,670 grams.
8	378	21	1.17	24.75	Died on 21st day; weight, 581 grams.
9	378	23	1.23	29.5	Died on 23rd day; weight, 570 grams.

The cases in which a microscopic examination was made after death may be grouped (1) as cases of acute poisoning (Rabbits 2, 3, 4, and 5) with death within three, four, or five days, and (2) as cases of chronic poisoning (Rabbits 6, 7, and 8) in which death occurred on the seventeenth, or twenty-first day. In all there was evidence of acute nephritis, showing in the more severe instances distension and cellular infiltration of the intertubular connective tissue, with injury to the tubal epithelium which sometimes had been shed. In all there were indications of an interstitial hepatitis, with more or less extensive round cell infiltration of the interlobular connective tissue, and usually an indefinite cloudy sort of change in the parenchyma cells, the outlines of which were often indistinct. Fatty changes were well marked in the liver in all four acute cases, and in the kidneys of Rabbits 2 and 3 which died on the third and fourth days. In Rabbits 2 and 3 there was a fairly heavy infiltration of the liver with fat, which was abundant in the parenchyma cells. There was also an infiltration of the cortical zone of the kidney, the fat appearing mostly in the epithelium of the convoluted portion of the tubules. The appearances, so far, were closely similar to those found in cases of human poisoning with arsenobenzol compounds, except that the actual proportion of fat appeared to be less relatively than in those human cases which had been examined. In Rabbits 4 and 5, which died on the fifth day, the livers showed areas of round cell infiltration along the course of the portal circulation; numerous small fat globules present were confined to the interlobular areas of cell infiltration, and had not reached the parenchyma cells of the lobules. In Rabbit 5 small branches of the portal vein could be seen with small globules of fat in what were apparently thrombosed lengths of the vessels. In these two rabbits there were only traces of fat in the kidneys, present in the form of quite minute globules, and mostly in the tubular epithelium. In Rabbits 6, 7, and 8, in which death was longer delayed, the appearances were again those of acute hepatitis and nephritis. In the livers of Rabbits 6 and 7 there was a fair amount of fat in the form of small globules lying in the areas of cell infiltration, but none was found in the liver of the third animal. In each case the kidneys showed an acute nephritis, with marked interstitial and epithelial changes, but without any fat infiltration.

Experiments with Ortho-amido-phenol.—Two rabbits (No. 10, weighing 1,150 grams, and No. 11, weighing 1,250 grams) received each a daily subcutaneous injection of 1 gram of ortho-amido-phenol, given in a normal soda solution; a third rabbit (No. 12, weighing 1,300 grams) was given a single dose of 0.5 gram. The first two rabbits became drowsy after each dose and died on the third and fourth days respectively; the third rabbit became drowsy on the third day after the single injection and was killed under chloroform. After death, an extensive, dark-brownish, gelatinous infiltration was found about the site of the injections; and the organs generally, and especially the spleen and kidneys, were stained a brownish colour. In Rabbits 10 and 11 there was an intense nephritis, with some disorganization of the kidney structure; no unchanged fat could be seen in the gland. The liver of Rabbit 10 showed an interstitial hepatitis, with some infiltration of the lobules with fat; in Rabbit 11 there were hepatitis and nephritis, but not any microscopic evidence of the presence of fat in either liver or kidney. The ortho-amido-phenol, in the large doses given, appeared to have an actively destructive effect both on parenchyma cells of the liver and on the kidney epithelium.

Experiments with Arsenobillon.—Altogether 7 rabbits were given various doses of arsenobillon injected subcutaneously, the total quantity administered ranging between 0.16 gram given in two doses of 0.8 gram each with a twenty-four hours' interval (Rabbit 14), and a total amount of 3.188 grams given by forty-eight injections, spread over a period of sixty-four days (Rabbit 17). In all the cases the rabbit died from the effects of the drug. The changes in the liver and kidneys were similar, in varying degree, to those found in the rabbits poisoned with arsenic. Thus in the liver of Rabbit 17, as in Rabbits 4 and 5, there were patches of round cell infiltration along the course of the portal circulation, without as yet any passage of the fat into the lobular areas. In other cases there was the appearance of destructive change in the parenchyma cells with, or without, the presence of unchanged fat. In all of the rabbits there was a nephritis of varying intensity.

Experiments with Kharsivan.—Three rabbits were injected with kharsivan, and each died within twenty-four hours of the last injection. Rabbit 18 (weight 1,400 grams) received altogether 0.8925 gram subcutaneously by nine injections. Rabbit 19 (2,250 grams) received 1.1 grams by three injections given on consecutive days, and Rabbit 20 (1,900 grams) was given an amount of 1.17 grams by six injections on consecutive days. In all three rabbits there was evidence of nephritis, without any unchanged fat visible in the sections of the kidney. The liver presented the usual indications of an interstitial hepatitis, with in the case of Rabbit 19 small globules of fat amongst the round cell infiltration.

The results of these experiments may be summarized. The action of the arsenic oxide is such that after a comparatively acute poisoning there is an interstitial hepatitis with injury to the parenchyma cells, caused presumably by the direct action of the poison, together with the appearance of considerable quantities of unchanged fat in and amongst the parenchyma cells. In less acute poisoning there is interstitial hepatitis, with fat present in the form of small globules and, as yet, limited to the infiltrated interstitial tissue along the course of the portal circulation. In all of the experimental cases there was a nephritis in addition to the hepatitis. In two of the cases a considerable infiltration of the liver and kidneys with fat had produced an appearance which was comparable exactly with that seen in cases of human poisoning with arsenobenzol drugs. After the injection of ortho-amido-phenol, in the massive experimental doses given, there were acute hepatitis and nephritis, with the presence of unchanged fat in the interstitial tissue of the liver. The action of the poison, in the large amounts, on the parenchyma cells of both liver and kidney, appeared to be severe. The results of poisoning with the two arsenobenzol drugs were comparable histologically with those obtained in the less acute cases of poisoning with arsenic oxide.

In these investigations special attention has been paid to disturbance of fat metabolism as an indication of disorder of function following on damage of the liver tissues caused by the action of various poisons. It is comparatively easy to follow any disturbance of this particular function of the liver, because of the readiness with which unchanged fat can be recognized by histological methods, whilst the biochemical researches of Leathes³ and others after him have given us an understanding of the physiological processes concerned. But of the various functions of the liver that which is concerned with the metabolism of fat is one which may be interfered with, temporarily at any rate, without causing much disturbance of the general health. From the pathological point of view a disorder of other functions which are concerned primarily with the production of urea from nitrogenous waste, and with the elaboration of uric acid from purin bases, are of more importance for the orderly continuance of vital economy. Therefore, whilst the results of the failure of fat metabolism are those which are most clearly obvious on inspection, the concurrent failure of other functions must be looked upon as the more serious and immediate factors in the causation of death. Death with all the symptoms of toxic jaundice, and with fatty infiltration of the liver and kidneys, may follow poisoning with arseniuretted hydrogen gas; so that it appears that the results characteristic of poisoning by arsenobenzol compounds may be produced by the action of arsenic alone. Also the results of my animal experiments show that the fat-metabolizing functions of the liver are directly affected by the action of arsenic oxide in poisonous doses. But in view of the serious damage to the liver and kidney tissues in experimental poisoning with ortho-amido-phenol it is impossible to ignore the probability that the toxic action of the arsenobenzol drugs may be influenced to some extent by the benzene constituent.

Possible Danger in Treatment with Arsenobenzol Drugs.

Three questions of practical importance remain to be considered on the recognition of these cases of poisoning: (1) As to the general risk of poisoning in the treatment of syphilis, malarial infections, and other diseases with arsenobenzol drugs; (2) as to whether the use during the war of other arsenical products in substitution for the "606" of German manufacture, which was in common use before the war, has been in any way responsible for the occurrence of poisoning; and (3) as to whether the so-called "intensive" treatment with arsenobenzol compounds, in combination with intramuscular injections of mercury, which came into vogue during the war, has had any influence in the direction of increasing the risk.

The risk of poisoning inherent in treatment by arsenobenzol drugs cannot be appreciated closely at present. The drugs were used in the treatment of unprecedented numbers of cases during the war, because of the military advantage gained by a shortening of the period during which a man with syphilis has to be kept out of the line; and it is certain that the deaths from poisoning by these drugs which have been recognized and recorded hitherto constitute only a fraction of the number of deaths due to this cause which have occurred during the last six years. This must necessarily be so; because the drug had not been recognized as a possible cause of the toxic symptoms which have been described, and because of the frequency with which a period of apparent good health intervenes between the completion of the treatment and the appearance of those symptoms.

Since these special toxic effects of the arsenobenzol drugs were not recognized during the period when "606" was in common use in this country, it is not possible at present to form any opinion as to whether the arsenical compounds which have been used in succession are more or less toxic than the original drug. As, however, the use of "606" was continued elsewhere during the war, and since the special dangers have come to be recognized, it is possible that some information on this point may be forthcoming later on. But the effects being caused by the essential arsenic, with probably some intensifying action of the benzene constituent, one might, perhaps, infer that when further information has been obtained it will not prove that the precise proportion of the two ingredients has been of material importance in influencing the toxic action of the drug.

With regard to the effect of the "intensive" method of treatment, apart from the conjoined use of mercury, there is not much to be said. It is difficult to trace in recorded cases any correlation between the actual amount of drug administered and either the time of onset of symptoms or the severity of those symptoms. The clinical course of the symptoms appears to be remarkably constant in a large majority of the fatal cases, which are the only ones as to which we have as yet any definite information. In the cases of delayed poisoning the symptoms have occurred usually after the completion of a course of treatment and when a full amount of the drug has been administered; but a precisely similar course of events may intervene during the course of treatment, and after the injection of only two or three comparatively small doses.

The possible effects of the action of mercury on a liver which may be already damaged more or less by the toxic action of the arsenical preparation require special consideration. It is a common belief in therapeutics that mercury has some sort of stimulating effect on the liver cells. The condition of the liver in experimental poisoning with mercury has been described differently by various pathologists. Burmeister and McNally¹ and others describe marked histological changes of a progressive nature in the livers of dogs poisoned with mercuric chloride, but the changes described have not included any of a fatty nature. Practically all observers agree, however, in describing an acute nephritis in experimental mercurial poisoning, with the occurrence of fatty changes in the tubal epithelium.

In a case of human poisoning which I had an opportunity of investigating recently a man, aged 33 years, died on the sixth day after swallowing a tabloid (?1 gram) of mercuric chloride. After death a condition of severe gastro-enteritis was found. Microscopic examination of the liver showed a hepatitis, with a moderate infiltration of the lobules with fat. The examination of the kidneys showed an intense nephritis, without any fat infiltration.

It is evident, therefore, that mercury in large doses has by itself a definite action on the liver tissue, and that its administration in even the moderate doses used with the "intensive" method of treatment may have an injurious effect on a liver already damaged somewhat, with consequent impairment of its functional activity, by the arsenical drug. That such possible unfavourable influence would be of a merely incidental or accessory nature, and not essential, is shown by the fact that mercury had not been administered in at least three out of my series of sixteen cases. Nevertheless it is advisable that the use of mercury in conjunction with arsenic should be discontinued; the more especially so in that it is difficult to understand in what way the additional treatment can have any useful effect under the conditions.

Taking a broad view of the matter in the light of the experience which has accumulated up to the present time, it has to be admitted that there is a certain definite risk in treatment with arsenobenzol compounds. It is impossible to express the risk in any individual case in the terms of a percentage; but the danger is sufficiently real to suggest that a man who is going through a course of treatment should be kept under careful medical regimen throughout the period of administration of the drug and for some weeks afterwards. It is scarcely necessary to say that complete abstinence from alcohol should be compelled; the diet also should be so regulated as to lighten the work of the liver as much as possible. The diet which would appear to be advisable during the course of treatment is the same as that recommended both for the prevention of poisoning by trinitrotoluene amongst munition workers and in the treatment of cases in which symptoms of poisoning have already occurred.

The administration of milk or any other fat food in the course of the treatment of declared poisoning is inadvisable. Whatever may be the exact causation of the disease of the liver two facts are outstanding. In the first place the liver functions become seriously disordered, so that the gland is no longer able to carry out effectively its part in fat metabolism. In the second place, a condition of excessive lipaemia is established. As in preventive treatment sugars should be substituted for fat foods, definite doses of glucose being given together with fruits. The sugar given thus is given not only in substitution for fatty foods, but also because of theoretical considerations which suggest that oxidation of the excessive floating fat will be favoured by its administration. The diet, then, should consist mainly of carbohydrates, with very small quantities of lean meat, or preferably fish—excluding the fatty kinds, such as herring, mackerel, etc.²

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Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

TREATMENT OF RINGWORM.

DURING the past twelve months I have successfully dealt with over sixty cases of tinea tonsurans, the majority of cases being cured within a month.

As a preliminary step the affected area of the scalp must be shaved and cleansed with liquid ethereal soap. The part is then gently and carefully rubbed with a piece of lint which has been dipped in liquor potassae and dried with a piece of cotton-wool. Next the part is sprayed with ethyl chloride for about thirty seconds and allowed to dry; it is then painted with tr. iodi mitis. It is unnecessary to repeat the shaving and cleansing with ethereal soap, but the remainder of the procedure should be carried out morning and evening for the first three days and once daily during the subsequent four or five days.

During this time a mild folliculitis occurs, and as a result the infected hairs fall out. Usually a week of such treatment is sufficient to effect a cure, after which it is only necessary to rub ammoniated mercury ointment into the scalp twice daily, keeping the case under observation for about a fortnight or three weeks.

As a rule the folliculitis quickly subsides and healthy hairs soon make their appearance. Other varieties of ringworm can be quickly cured by like treatment.

Coventry.

W. P. ELFORD, M.A., M.D.

SIMPLE PAROXYSMAL TACHYCARDIA.

As Dr. Thomas Lewis, in his *Clinical Disorders of the Heart Beat*, states that paroxysmal tachycardia is rare in young children, the following case may be of interest, especially as the boy's mother being similarly affected there is a possibility that heredity played a part.

On April 24th, 1920, I was called in to see a boy, aged 13, who complained of "quick beating of the heart." His mother informed me that he had his first attack when 4 years old, and subsequently one at 6, when convalescent from an ordinary attack of scarlet fever, and at 9, after returning home from tobogganing. During the past winter he had two attacks. He stated that the attacks came on suddenly, without warning or known cause, and after lasting one to three hours stopped as suddenly. He had not had rheumatism; since 8 years of age he had won prizes at the school races, and whilst running had never felt any ill effect. Before the present attack the patient felt quite well. When walking in the house about 9 a.m. he suddenly felt his heart beating fast, and this caused him to sit down; he experienced no pain or breathlessness, and no sickly or faint feeling. When I saw him at 12 noon he was still sitting up, the heart was beating forcibly, and the rate was uncountable. The patient had a slightly pale appearance, and his expression was rather anxious. When he lay down no alteration in the rate of the heart's beat could be detected, but he felt more comfortable. The apex beat was just inside the nipple line, and on percussion no enlargement of the heart was found. Auscultation revealed no murmur; the sounds were tic-tac in character. At 1 p.m., although the patient felt hungry, he could not eat much dinner. At 3.30 the beating suddenly stopped, and for a time (he states ten minutes) he felt short of breath; towards the end he had a severe stabbing precordial pain, which disappeared on taking long deep breaths.

I examined him again four days after the attack. No pallor was noticeable and the pulse rate was 74 per minute; in the mitral area a slight presystolic thrill was palpable, and auscultation revealed a short presystolic murmur leading up to a sharp first sound. The second sound in the pulmonary area was reduplicated. The patient stated that since the attack he had not dared to hurry, for fear of bringing on another. I found that his visual acuity was only $\frac{1}{2}$, even with glasses, and he informed me that his sight often became blurred. Dr. Fison kindly estimated his refraction, and found a considerable degree of astigmatism present. He ordered fresh glasses, those in use being unsuitable. As the boy has recently been studying more than usual, the visual defect may account for the attacks occurring so closely together.

His mother is subject to similar attacks, which, in her case, first occurred at 19 years of age, and have since continued at varying intervals. She says that the attack "comes on with a big jump"; she feels the heart beating quickly and the vessels in the neck are seen to beat forcibly. If the attack lasts for any considerable time she feels an aching pain in the upper part of the chest. She is most comfortable lying down, and the attacks end suddenly, after lasting from a few minutes to some hours; they usually come on after over-exertion or too much excitement. On examination of the heart while she was resting no murmur or irregularity was detected. She gave no history of having had Graves's disease, and had no exophthalmos, swelling of thyroid, nor tremor. Her parents are both alive and well, and no history was obtained of cardiac trouble in either.

Harrogate.

A. C. SHARP, M.D. Glas.

OBSTRUCTION DUE TO ADHESIONS OF THE PELVIC COLON.

THE following case is interesting, in that it presented all the general signs and symptoms of increasing obstruction by a malignant growth:

A man of 56 complained of obstinate constipation, which had increased progressively during nine months; for one month he had had diarrhoea every morning, the stools being scanty, slimy, and frothy. He had not noticed blood in them, and was able to pass flatus freely. Two or three similar stools followed the morning stool, and, although he was able to do his work, he almost invariably had one or two "vomiting turns" during the course of the day. He had suffered from rheumatism, and twenty years ago had enteric fever; during the last twelve months he had lost 18 lb. in weight. On abdominal palpation I thought I could detect in the left iliac fossa a small, hard, immovable mass of very ill-defined outline. I was greatly struck with the huge balloon-like cavity shown by rectal examination; the walls of the rectum were felt with difficulty, and were impalpable except in the lowest inch and a half. No tumour was felt. The diagnosis of malignant growth in the lower portion of the pelvic colon was made both by myself, and independently by another surgeon elsewhere.

Assisted by my colleague, Mr. E. A. Guymer, I opened the abdomen by a five-inch incision in the left linea semilunaris. The liver was normal. There were no palpable glands along the course of the large vessels, nor accessory signs of