estimated. Now, if arsenic were the chief cause of so-called alcoholic neuritis, we should expect to find a great diminution in the number of cases occurring after the epidemic, that is, when the beer was free from arsenic. On the contrary, the percentage of cases admitted to the Manchester Royal Infirmary since the epidemic is almost identical with that of cases admitted before the epidemic. Thus, if we take the seven years included by the years 1892–1898, there were 109 cases of peripheral neuritis out of 9,300 medical in-patients, that is, 1.01 per cent.; whilst in the seven years, 1901–1907, there were 116 cases out of 10,630, that is, 1.09 per cent. But in the year of the epidemic, namely, 1900, there were 62 cases out of 1,343, or 4.6 per cent.

Hence it is clear that, before and after the epidemic, arsenic, if present at all in the alcohol consumed, was in very small quantities. Further, it is to be noted that pigmentation, keratosis, and other skin lesions, so prevalent in the epidemic, were not mentioned in the reports of earlier cases by such acute observers as Dr. Ross and Dr. Dreschfeld. On the other hand, we must admit that the majority of Manchester cases occurred in beer drinkers, and that a small amount of arsenic in the beer may have been a contributory factor.

It is therefore desirable to consider the question of neuritis in drinkers of spirits. In this connexion we may note the valuable statistics of Dr. F. Buzzard. In the ten years included by the years 1891–1900, out of 6,458 cases admitted to the National Hospital there were 108 cases of alcoholic neuritis—that is, 1.6 per cent. Of these 108 cases, 25 were due to excess in spirits only, and only 1 case due to beer only. Dr. Buchan, also, in 19 cases of alcoholic neuritis found that whisky was the chief beverage in 18, and the only one in 6.

These results are in accordance with the records of the earliest observers of peripheral neuritis in alcoholic subjects, as witness the graphic account by Dr. Lettsom of the disease as occurring in those who drank brandy and gin in excess, and that by Dr. James Jackson, who attributed the disease to ardent spirits. They are also borne out by the statements of modern authorities on the subject. Thus Dr. T. Buzzard says his experience has been that neuritis is due especially to spirit drinking. The late Dr. Ross, in an analysis of 90 cases, found that the form of alcoholic beverage was as follows: In 22 spirits, in 3 absinthe and vermouth, in 13 beer and spirits, in 5 beer only, whilst in 47 the kind of alcohol was not given. Moreover, of recent years many cases of peripheral neuritis from spirit drinking have been observed in which beer could be definitely excluded. Dr. Williamson has recorded a case from excessive whisky drinking, no beer having been taken, in which the diagnosis was verified by a microscopical examination of the peripheral nerves. In this case three samples of the same whisky gave no reaction for arsenic with Reinsch's test.

Reverting to the question of beer drinking, I may mention that several cases of *well-marked* and *severe* multiple neuritis, which have come under my care since the epidemic, have been carefully investigated, and arsenic as an etiological factor definitely excluded, no trace of this poison being found in the hair or the urine of the affected patients, nor in samples of the beer that had been taken.

It is open to any one to say that it is not the ethylic alcohol but some accidental impurity that is responsible for the neuritis, but until this toxic agent is discovered, which is certainly not arsenic, it is impossible to resist the conviction that alcohol, whether in the form of beer, wine, or spirits, is a common cause of multiple neuritis.

But this statement does not necessarily imply that multiple neuritis is of frequent occurrence in alcoholic subjects, nor that the disease is a direct result of alcohol. Indeed the proportion of alcoholics who suffer from neuritis is a small one, and in such sufferers other factors may play a contributory part. The action of alcohol may be an indirect one in the production not only of neuritis but of hepatic cirrhosis and of changes in the muscle of the heart. It is a reasonable assumption that the lowering of tissue resistance by alcohol permits the successful attack of toxins, derived from micro-organisms or from the changes associated with chronic inflammation, upon the nerves, liver, heart or other structure.

A similar view is held by Dr. F. Parkes Weber, who suggests that the true causes of multiple neuritis of alcoholic type are infectious, alcohol being merely a predisposing agent.

But whether the neuritis is a direct or an indirect result of the poison there is ample evidence to justify the view that well-marked cases of multiple neuritis (although not common) are produced more frequently by alcohol than by other poisons.

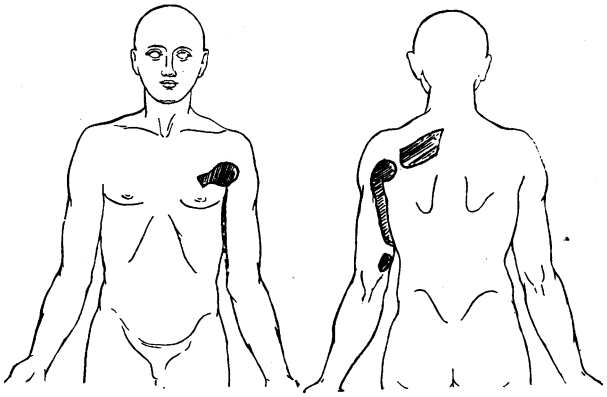
## PAROXYSMAL TACHYCARDIA DISAPPEARING AFTER AN ATTACK OF HERPES ZOSTER.

By ALAN C. TURNER, M.R.C.S., L.R.C.P.,
SHEFFIELD.

The following case presents features of sufficient interest to make it worth recording. The history is briefly as follows:

L. S., aged 15 years, has been under my care for some time suffering from recurrent attacks of tachycardia.

She is rather short, somewhat anaemic, and has never been very strong. She easily takes cold, and has had repeated attacks of influenza. The first attack of tachycardia occurred when she was 5 years old. She awoke suddenly about midnight, feeling "as though she were going to die." She was very pale, and the heart beat very fast. After a short time she vomited a small quantity of liquid; the heart at once became quiet, all symptoms quickly passed away, and she soon fell asleep. This attack was attributed by her friends to a strong cup of tea she had taken the evening before. From that time onwards she had been subject at least every two months to similar attacks. She is obliged to sit still whilst the attack is on, and, however



long it lasts, is unable to take any solid food, but can sip a little liquid. An attack always continues until she vomits, after which all the symptoms quickly disappear. Latterly, she has been given warm salt and water to produce vomiting, and this has cut short the attacks. The vomit consists of one or two tablespoonfuls of sour fluid. The attacks have been attributed at various times to sudden exertion, such as running, to excitement, or to irregularities in diet, but no common etiological factor could be found. In other respects she seems fairly well, the bowels act regularly, and the urine is normal.

The first time I saw her, about five years ago, she was suffering from an attack. She was propped up in bed, very pale, complaining of tightness across the chest and of rapid heart-beat. The pulse was about 160; the impulse was in the nipple line, and no murmurs were audible. Since then I have seen her in several similar attacks; the pulse-rate has varied from 120 to a rate faster than could be counted. They have lasted for various periods, from less than one hour to nearly three days. No drug among the many that have been tried has had any effect in cutting short the attacks, nor has attempted pressure on the vagus been more successful. The attacks have always lasted until she has vomited, when, within fifteen to thirty minutes after the pulse would fall to normal.

On October 10th, 1906, she had severe pain in the left wrist which continued to the next day, when it affected the inner side of the left arm, the front of the left shoulder, and the back. On the morning of October 12th a rash appeared about the left shoulder and arm, the left ear became tender, and she had also some pain in the right wrist. Dr. Arthur Hall saw her at that time in consultation. She had typical herpes zoster affecting the area of the second left dorsal segment—namely, the under and inner part of the left arm, the left breast in the region of the axillary fold, and over the left scapula in the upper part (see diagram).

The eruption ran the usual course, and from the cardiac attack which preceded the eruption by about a month up to the present date—that is, for three years—she has had no recurrence of the tachycardial attacks, her heart is quite normal, and she has never felt in better health than she does now.

There is no family history of any particular tendency to nervous disorders or heart affections.

I have used the term "paroxysmal tachycardia" for this case without thereby meaning to imply any certainty as to the nature of the type of heart beat which characterized it.

Mackenzie (*Diseases of the Heart*, 1908, p. 129) would include under this term only those cases in which a "nodal rhythm" is present. Whether this case fulfilled his requirement or not I cannot say, since no tracing was taken during an attack, and no attacks have occurred since we have been in a position to take such tracings.

The case, however, is typical of the class described by Bouveret (*Revue de Médecine*, 1889) as "La tachycardie essentialle paroxystique," and concerning which Herringham (*Edinburgh Medical Journal*, 1897) has contributed a valuable monograph.

This case in most of its features agrees with one or other of the recorded cases, the variations being chiefly in minor and unessential details. The termination of the original attacks in vomiting and the artificial cutting short of subsequent ones by the administration of emetics is interesting, but does not warrant us in forming any wide inferences therefrom.

The feature which in this case seems to call for publication is the coincidence of an outbreak of herpes zoster of the left second dorsal area with complete cessation of the heart attacks.

Possibly it is no more than a "coincidence," in the popular sense of the word. Cases of paroxysmal tachycardia do cease spontaneously, and it is possible that in this case the cessation may only be a temporary one and that further attacks may occur later. On the other hand, the fact that the second left thoracic nerve is very closely associated with the nerve supply of the heart, and particularly with its accelerator fibres, and the fact that previous to the outbreak of zoster the heart attacks had recurred fairly regularly every two months for ten years, seem to suggest that the two things stand in some direct relation to each other.

## TRAUMATIC RUPTURE OF THE SMALL INTESTINE: SUTURE AND APPENDICOSTOMY: RECOVERY.

By FRANK RADCLIFFE, M.D,
ASSISTANT SURGEON TO THE OLDHAM INFIRMARY.

In the British Medical Journal of May 29th, 1909, Mr. William Sheen of Cardiff records a case of traumatic rupture of the sigmoid colon, in which, after commenting upon the rarity of the lesion, he, by reference to a paper by Berry and Giuseppi, shows that the mortality in the London hospitals in cases of traumatic rupture of the intestine has been over 87 per cent. Under these circumstances it becomes a duty to record such cases, and especially so if, as in the following case, they present features of special interest attached to their treatment.

A. L., aged 33, bricksetter's labourer, was admitted to the Oldham Infirmary on May 10th, 1909, at 6 p.m. He said that he had received a violent blow from the end of a plank, which struck him in the epigastrium; that afterwards he was able to walk a few yards and descend a ladder, but then felt too ill to move. He lay upon the right side with the knees drawn up, and was evidently in great pain. The abdomen was of board-like rigidity, retracted to the scaphoid shape, with the skin thrown into transverse folds, and very sensitive to pressure. The flanks were dull on percussion, but this dullness did not move on changing the position of the patient. The liver dullness was normal in extent. There was no external mark of violence except a few small superficial scratches. Shortly after admission he vomited a little brownish fluid.

It was decided to operate, but the patient would not consent until he had seen his wife, thus delaying the operation until nearly 10 p.m.—that is to say, seven hours after the injury.

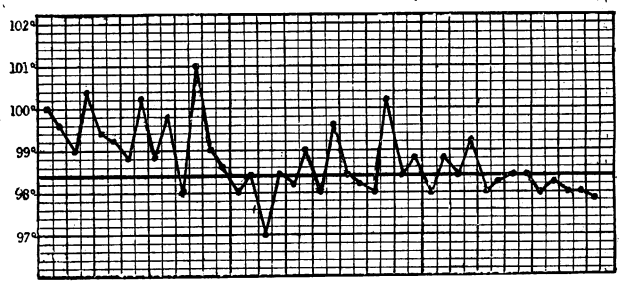
*Operation.*

Chloroform was administered by the junior house-surgeon, Dr. Gray, and I had the assistance of our senior house-surgeon, Dr. Mackenzie.

An incision was made in the middle line above the umbilicus. On opening the peritoneum there was an escape of brownish, clear fluid. On exposing the liver margin more of this fluid welled up, and with it some bubbles of gas. Examination of the stomach revealed no lesion. The transverse colon and omentum were then drawn up, thus allowing more fluid to escape and with it some flakes of lymph. The intestines were deeply injected, and in places covered with large flakes of lymph. Systematic examination of the small intestine revealed a longitudinal rupture about ¾ in. in length, with pouting mucous membrane on the free border of the jejunum, about 2 ft. from its commencement. The rupture, after sponging with weak mercury biniodide lotion, was closed with a continuous Lembert suture, and the examination was continued but no other lesion was found. A suprapubic incision was then made, and on opening the peritoneum more fluid of the same slightly turbid character and containing small food particles escaped; through this opening a Keith's tube was passed down to the bottom of Douglas's pouch. Another incision was then made through the parietes over the appendix, which was brought through this incision, and its mesentery cut after ligaturing it. The appendix was then amputated, leaving about an inch of stump. This was dilated, and a rubber catheter inserted through it into the caecum and fixed by a purse-string suture; the stump of the appendix was anchored to the skin by two fine silk sutures, and this incision closed round the appendix. The epigastric incision was completely closed by through-and-through sutures and dressings applied. The patient was removed to bed and put into the Fowler position. The whole operation took less than an hour from the first incision to the patient being removed from the table.

*After-Treatment.*

A cistern was arranged about 2 ft. above the level of the patient's abdomen, and under it a spirit lamp; the cistern was connected by rubber tubing with the rubber catheter, and the tubing clamped so as to allow one drop of normal saline solution with which the cistern was filled to escape every second.




It was found that to ensure the drops of saline having a temperature of 98° F. we had to keep the cistern contents at 150° F. By this arrangement we were able to cause a continuous flow of saline solution into the caecum for forty-eight hours. The tube then came out, and on reintroduction there was so much pain that it was discontinued. The total quantity of saline solution introduced was two gallons.

The patient continually perspired, passed urine freely, and at the end of the time had some incontinence, passing faeces along with a moderate quantity of saline fluid. Moreover, what was more interesting, there was fluid continuously welling up through the Keith's tube, so that the pad over the tube had to be changed every half-hour.

On the fifth day the Keith's tube was removed, as the flow of fluid through it had almost ceased.

On the seventh day some flatus was passed through the appendix.

On the eighth day the stump of the appendix came away as a slough, and left a faecal fistula, which remained open for a fortnight. There was some suppuration along the track of the sutures in the epigastric wound.

The patient was out of bed at the end of three weeks, and walking in the garden within a month.

I relied entirely upon the infusion of saline solution into the caecum to rid the peritoneal cavity of its irritating contents, by causing the membrane to secrete instead of to absorb, and that this occurred was evident by the copious flow from the Keith's tube, continuing for five days, even after the infusion was discontinued.

The man's condition never caused any anxiety after the first twenty-four hours.

If I should meet with a similar case I would diminish the rate of flow of the saline after the first twenty-four hours, for the patient had considerable inconvenience, from the thirty-sixth to the forty-eighth hour after operation, from the rectal incontinence.

It is stated that a motion is about to be submitted to the Duma making vaccination compulsory in Russia. The Inspector-General of the Medical Department has asked the various administrative authorities and institutions to report on the subject, and the Minister of Public Instruction has sent out a circular to the faculties of medicine.