Clinical and other Notes.

A CASE OF STOKES-ADAMS'S DISEASE OCCURRING IN A SOLDIER.

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The following notes may be of interest, as this disease, rare at most times, is scarcely ever found amongst soldiers. The man affected had led a very active life. The symptoms came on very suddenly, and the case terminated rapidly.

Serjt. S., aged 43, was admitted to British Station Hospital, Secunderabad, complaining of fainting attacks and pain over the heart.

Previous History.—He had a long service in the army, with a very fine record in the War, being in charge of the battalion stretcher-bearers all through the campaign, and gaining the D.C.M., M.M. and bar. He stated that he had always enjoyed good health. He admitted that he had had gonorrhoea, but denied syphilis. His medical history sheet was not obtainable until after his death, when it was discovered that he had numerous entries, first for rheumatism, and later, several times for syphilis. Late in 1918 he was gassed, and he stated that he had never really felt fit since.

History of Present Illness.—He stated that about a month ago he was playing cricket for the battalion, and made a few runs. He felt a bit exhausted, and when he got home he had a slight fainting attack. Since then he had been rather “off colour,” and had not been taking his food well. On being questioned, he stated that he used to get rather breathless when going upstairs. I had seen the man personally a couple of days before admission, just after he had climbed a steep flight of stairs, and noted at the time that he looked rather out of breath and a bit cyanosed. He stated that the night before admission he was sitting in his quarters talking to his wife, and that “he kept on having turns,” in which he said “he lost himself.”

Condition on Admission.—The patient was seen in bed. He was lying down, and did not give one the appearance of being distressed at all. His colour was not very good, but he could not be described as being cyanosed. He gave his history quite clearly. The first that I noticed about him was the extreme slowness of his pulse, which was only twenty-eight to the minute. It was of a curious consistency, being very forcible and slow, somewhat simulating a “water hammer pulse,” but without collapsing again after each wave, the artery remaining full. On examination his chest moved well, and was normal to percussion, with the exception of an increase in the area of cardiac dullness, which extended to an inch external to the left nipple. The lungs were normal.

On auscultation of the heart over the apex beat, a loud blowing systolic bruit was heard, which practically replaced the first sound. This was traceable out to the axilla. In the second right intercostal space, a blowing aortic murmur could be heard. He soon became somewhat collapsed, and strychnine $\frac{1}{100}$ grain was given hypodermically. This was followed almost immediately by two or three convulsions of an epileptiform character, during which the patient began to
breathe rapidly and start twitching his face and limbs. These convulsions lasted from ten to twenty seconds. During the attacks the radial pulse could not be felt. This led me to suspect a cerebral haemorrhage, but the attacks passed off and no paralysis ensued. At the commencement of each convulsion his face became pale, and as the fit passed off a wave of colour spread over his face. It was then noticed that there was a marked pulsation in the neck of the right jugular vein, and the rate of this was greatly in excess of the radial pulse, the ratio being 80 to 28. He continued to have convulsions every two or three minutes, and it was noticed that between the attacks he talked quite reasonably. The radial pulse would then suddenly stop; he would become pale, convulsions would occur, and then pass off. His colour would return, and he would then continue talking. On auscultation of the chest during an attack, loud bubbling râles could be heard all over both lungs, and there was a "death-like" rattle in his throat. Oxygen was administered, but with no beneficial result. Hot water bottles were applied to the left side and to the feet, and champagne was given in small sips. His condition improved, and the convulsions ceased. He was given only fluid diet, which was administered in small spoonfuls at a time. He had a comfortable afternoon, and no return of the convulsions, and slept on and off for the remainder of the day.

Second day: At 2.30 a.m. he had another attack of convulsions, and complained of cardiac pain and faintness. His colour was bad, and he became cyanosed. With oxygen and hot foment to the left side, the condition improved, and he became fairly comfortable, sleeping at intervals. He had another attack at 6.30, and three more attacks during the morning, and it was noticed that the radial pulse was becoming slower, dropping to fourteen beats per minute, whilst the jugular pulse increased to 86. He dozed off at times, and waked with a start, and complained of bad dreams. He spent a comfortable afternoon, taking nourishment well. Patient had a very bad night, with numerous convulsions, which became more frequent, and he became delirious at times.

Third day: During the morning his pulse ratio altered markedly. After two convulsive attacks the radial pulse increased to 50 beats per minute, but dropped again soon after. It was noticed that it always increased in rate just after an attack. He had attacks almost continually all the morning up to 3.15 p.m., the radial pulse dropping gradually to 20, and the jugular remaining at 80. Atropine 3/100 grain and digitalin 1/50 grain given, but with no effect. The remainder of the day was fairly quiet, with occasional attacks.

Fourth day: The next day, at about 11 a.m., the pulse ratio showed a marked improvement. The radial pulse improved both in rate and volume. There was scarcely a couple of beats difference between it and the jugular pulse. The patient felt very much more comfortable, and the bruits were not nearly so marked. This lasted until about 5 p.m., and one was hoping that the ventricles were becoming educated to beat on their own at a decent rate. However, he then had an attack of vomiting, followed by a bad attack of convulsions. He was given digitalin 1/50 grain, which appeared to relieve him, and he slept.

Fifth day: Next day retching and vomiting increased, and he could keep nothing down. He began to get very irritable. A large number of moist, bubbling râles could be heard over both lungs, especially at the bases, but no consolidation was made out.
Sixth day: Condition the next day was much about the same. Irritability was more marked. Vomiting severe. Complaining of pain in the pit of stomach. He coughed up blood-stained rusty sputum. No signs of consolidation of the lungs, but signs of hypostatic congestion. He had two attacks during the night, during which he stopped breathing, and the pulse was imperceptible. Oxygen was administered, and artificial respiration performed. At 12 midnight he had some rest from vomiting, and slept a little. Respiration got very rapid—patient rapidly sinking. He again stopped breathing. Oxygen and artificial respiration again brought him round. Two or three more continuous attacks occurred, and he passed away on the seventh day of admission. A post-mortem was done, and the report is as follows:—

The thorax was opened by removing the sternum and costal cartilages. There was some slight adhesion of the pleura at the base of the right lung.

The oesophagus contained a thick, glairy mucus. On washing this away, the wall was found to be congested.

The trachea was opened and contained frothy blood-stained mucus.

The bronchi were congested, and, on squeezing the lungs, blood-stained mucus poured out. The lungs were engorged with blood-stained mucus, but no area of consolidation could be found, and portions cut from the bases floated easily in water.

The aorta was much dilated, and showed marked atheroma and ulceration of the walls, but no calcareous deposits. There was marked thinning of the wall at one place in the arch of the aorta.

The pericardium was not adherent, and there was no increase in the amount of pericardial fluid.

The heart was greatly enlarged, weighing two pounds. There was great hypertrophy of the left ventricle, and great dilation of the right auricle and right ventricle. An incision was made into the left ventricle by passing scissors down through the aorta. The aortic valves were very much thickened and fibrous, more especially the septal cusp. There was a large organized clot of fibrin completely filling this cusp. The aortic ring was greatly dilated, thus causing incompetence of the valve. The walls of the left ventricle were greatly hypertrophied. The mitral valve showed no appreciable change, and there did not appear to be any dilation of the ring. The left auricle was not dilated. The right auricle was very much dilated. The tricuspid valve showed no thickening of the chordæ tendinae or the cusps, but there was considerable dilation of the ring. The right ventricle was greatly dilated. Just below the septal cusp of the tricuspid valve, there was a firm tense swelling, protruding into the ventricle and corresponding with the lump to be felt behind the septal cusp of the aortic valve. This lump is suggestive of a gumma, and appears to be invading the auriculo-ventricular bundle of His.

The liver was enlarged and very firm. The gall bladder was distended, and there were numerous adhesions to the lower surface of the liver, of the transverse colon and pancreas. On section, the liver showed marked "nutmeg" appearance, and was very engorged. The gall bladder was distended with blood-stained bile. The pancreas was greatly enlarged, and was adherent to the under surface of the liver. There was small hæmorrhage into the gland, and in the centre of the body a large cyst was found. The pancreatic duct was greatly hypertrophied.
The kidneys were congested, and the capsule stripped easily. The spleen was enlarged and congested.

The stomach was dilated and very much engorged, and there were some submucous haemorrhage.

Death was caused by failure of the heart, due to Stokes-Adams's disease.

A histological section taken of the tumour found in the bundle of His showed fibrous tissue only. No muscle fibres could be found at all.

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A CASE OF ACUTE SPINAL ABSCESS.

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The patient, Private K., was admitted to hospital in France from a convalescent depot on April 22, 1918. He was fairly well nourished and his general musculature was good. He gave his age as 28 years, but he appeared to be much older, his hair being quite white. He had been diagnosed as a case of "neurasthenia," apparently owing to his suffering from general tremors, tremor of the tongue and lips, and complaint of insomnia. On examination the above tremors were present, but in addition he complained of severe pain in his "tail" and of constipation. He stated that he had been sent down the line as suffering from debility. He had not been confined to bed at any time during his disability until the last two days. On the evening of his admission he was unable for the first time to pass urine and eventually he had to be catheterized.

April 23. The bladder was found markedly distended in the morning, and seventy ounces of urine were drawn off. He got up in the afternoon but said he felt very weak, so was sent back to bed. General tremors were marked, and he complained of pain over the spines of the fourth and fifth lumbar vertebrae and the sacrum. He also complained of pains shooting down both legs. The region over the lumbar spines was markedly hyperalgesic. The tendon reflexes were absent in the lower limbs, but there was no paralysis, no anesthesia, no Babinski's sign and no ankle clonus.

April 24. The patient's temperature rose to-day to 103°F. Pulse was 120 but strong. He complained of feeling very weak. Urine, which was still being obtained by catheter, was found to contain nothing abnormal. He now said he had difficulty in retaining faeces. His abdomen was becoming distended. Spleen was not enlarged. He was experiencing some difficulty in moving his legs.

April 26. Temperature remained about 103°F, and without remissions. He was catheterized twice daily. There was also now incontinence of faeces. The abdomen had become markedly distended and was tympanitic all over. There was increasing weakness in the legs, pain in the lumbar region, and shooting pains in the legs were constantly complained of.

April 27. A rectal examination was negative. The pains were not so severe to-day, but he was quite unable to move his lower limbs. There was still no anesthesia. He said he felt as if there was some obstruction in his lower bowel, but incontinence of faeces continued.

April 28. He was found to be suffering from epididymitis on the left side.