ideal safety belt should be broad and resilient, attached to the framework of the aeroplane and not to the pilot's seat, should be made to release easily and quickly, not at the centre of the pilot's body but at the side where it is attached to the aeroplane. This release should be effected by means of a small hand lever. It is advisable for all pilots to carry a stout knife in the outside pocket of their flying coat in order to cut the belt should they be held in upside down after a crash.

Safety Helmets.—These are of undoubted value in school work and should be worn by all pupils. They should fit properly and not be easily dislodged whilst flying. The modern ones are much lighter and less high in the crown than the earlier ones used. In a crash they certainly prevent scalp wounds from broken struts and wires and the side flaps protect the ears from injury. Over and over again I have seen pilots thrown out who owe their escape from more or less serious head wounds to their safety helmets.

On the other hand, in a turn over, the added height of the crown may catch the ground and wrench the head either forwards or backwards, causing fracture dislocation of the neck, or severe strain and possible rupture of the muscles of neck and back.

As pointed out by Fleet Surgeon Wells, an ideal safety helmet would take its support from the shoulders.

Goggles.—Most aviators wear goggles, but there are still some who prefer to fly without them. I know of one instructor who, after a year continuously instructing pupils and wearing no goggles, began to suffer from a form of conjunctivitis. I certainly think that goggles should always be worn. There is no doubt that flying without goggles is apt to set up a spasm in the eyes which in the long run is bound to do harm. Triplex or non-splintering material is now almost universally used instead of glass in the manufacture of aviation goggles. Thus rarely in a crash do we get any injury to the eyes. The nose-piece connecting the two lunettes should have no metal in its composition. I have seen some cases where wounds of the nose were caused by the metal connecting part of the lunettes.

Accidents under Dual Control.—Accidents under dual control are not common, as the instructor has usually time to correct in the air any of the pupil's errors in flying. One occurred in the V series, and was unavoidable owing to the character of the ground. In the E series three occurred. All dual control machines should be fitted with a mechanical device for throwing out of action quickly the pupil's control of the machine. I have seen three other crashes under dual control but without injury to either instructor or pupil.

Fatal Accidents.—In two years of school work I have only seen three fatal accidents occur in roughly 200 crashes. Four deaths occurred, of which three were instantaneous and one after four days. All were due to multiple injuries.

ACUTE YELLOW ATROPHY IN SYPHILIS.

(A Preliminary Note.)

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DURING the past two months I have had the opportunity of studying in the *post-mortem* room no fewer than five typical cases of acute yellow atrophy, an experience which must be almost if not quite unique. Though numerous examples of subacute types of the disease have been recorded in recent years, and particularly in munition workers, typical cases of the classical icterus gravis are seen only at long intervals. According to Best¹ (1903) only some 500 cases of acute yellow atrophy were on record at that time.

In a personal experience of over 5,000 *post-mortem* examinations performed since 1898 I have only investigated one other case. The present series of five cases have all occurred in syphilitic subjects in the course of, or subsequent to, treatment by "salvarsan" preparations and mercury; and considering the number of cases of syphilis now being treated throughout the country, a searching

analysis of these fatal cases would appear to be urgently called for to determine, if possible, the relative parts played as etiological factors by the syphilitic toxin, arsenic, and possibly some other exciting agent.

I do not propose in this communication to describe the details of the observed cases, these being reserved for a later and fuller joint report, but would content myself at present with a brief summary of the essential facts.

In practically all the cases a full course of intravenous injections of salvarsan had been given, coupled with the usual intramuscular injections of mercury. Needless to say, in each case the diagnosis of syphilis was not only clear on clinical grounds, but was confirmed by a Wassermann test. These cases were not of undue severity, and indeed showed no special symptom of importance till a sudden onset of jaundice was noticed, without at first any special disturbance, the disease suggesting nothing more than an ordinary catarrhal jaundice.

At a varying period of from two to eight days, however, acute symptoms appeared with dramatic suddenness. They were ushered in by a period of wild excitement and increased icterus, with haematemeses. The patients rapidly passed into a condition of deep coma, and death occurred in the five cases at periods of one to four days from the onset of the acute symptoms.

The urine was markedly bile stained, and in each case showed tyrosin; in some leucin was also demonstrated. In four of the cases diminution in the size of the liver was demonstrated during life.

These cases, then, presented clinically the classical symptoms and signs of icterus gravis of a degree of acuteness which is seldom seen. The morbid anatomy and histology of these cases were equally striking and equally characteristic, the appearances being practically identical, and varying only in degree. To summarize the *post-mortem* results, there was in each case great diminution in the size of the liver, which varied in weight from 27 to 43 oz. Histologically there was the usual intense necrosis rather than a fatty degeneration of the liver parenchyma, the bile duct epithelium largely escaping; there had been, of course, no time for liver-cell regeneration, but a definite inflammatory reaction was present. Though "red" areas were macroscopically apparent in the liver, the condition has been so acute that in most cases the "yellow" areas preponderated. It is to be noted, however, that the naked-eye appearances give no adequate indication of the extensive changes found microscopically. In each case there has been widespread haemorrhages in the serous membranes with a special haemorrhagic infiltration of the lungs. The kidneys, in contrast with the liver, have shown an intense degree of fatty change, with, in some instances, definite signs of an inflammatory reaction. There has been constantly noted also a varying degree of intestinal catarrh.

Bacteriological examination has, in contrast to what has been hitherto found in similar cases, yielded some interesting results which may prove of prime importance in relation to the etiology of the condition. Cultures from the heart blood and lung have given copious growths of organisms apparently of the *coli*-typhoid group, with some definite cultural characteristic features and pathogenic properties (in two at least out of the three strains tested so far). I am unable to say more at present, as their cultural and biological reactions are still being investigated, and as the bacteriology of acute yellow atrophy has hitherto been so indefinite, one must be chary of attaching too much importance to their presence without some experimental evidence of an etiological relationship with the disease. One may be permitted, however, to review briefly the present opinions as to the causation of acute yellow atrophy, or rather acute necrosis as it should be called, and to note how the various hypotheses which have been advanced would apply to the present group of cases. In 1908² I endeavoured to summarize the current views at that time from a study of an acute case, and a series of subacute cases which I had personally observed, and particularly from a series of cases which had been observed in that year in Germany and America.

To quote from that paper, I stated

that there is much to be said in favour of the view that in the causation of acute yellow atrophy we have two factors at least at work. It may well be that the typical condition is

only produced when some special virus acts on a previously damaged liver;

and again,

that the most reasonable view to take would appear to be that the essential cause of acute yellow atrophy is some poison, possibly of microbic nature, produced in the alimentary tract and acting on a liver previously damaged, or whose function at least has been disturbed.

I do not, of course, claim originality for this view; it has been held by others, no doubt, both before and since. I only present it again as a working hypothesis.

The damage to the liver may be produced, as we know, by metabolic poisons—for example, in eclampsia and allied conditions—and there is now abundant evidence that syphilis may in the same way be an essential factor in paving the way for the development of acute yellow atrophy in another group of cases. That syphilis produces definite changes in the liver is common knowledge. The existence of gummata and cirrhosis in both congenital and acquired syphilis is common, but it is apparently not so widely recognized, except by those who are treating large numbers of cases of syphilis, that jaundice is not so very uncommon. According to various authorities, it would appear to occur in from 1 to 2 per cent. in cases treated in the German clinics.

It has also been long recognized that syphilis is not an uncommon precursor of typical acute yellow atrophy, and was noted long before salvarsan was ever used as a curative agent. In 1898 Richter collected 41 cases, and, though later Veszpremi and Kanitz,⁸ in a criticism of Richter's observations, cast doubt on the presence of syphilis in ten of these cases, numerous observations on acute yellow atrophy since leave no doubt that syphilis has been present in a very large number of instances. In another group of cases, poisons not of microbic nature have fallen under suspicion; thus phosphorus, arsenic, mercury, chloroform, and, in quite recent years, trinitrotoluene and tetrachlorethane, have been shown capable of producing changes in the liver which are comparable at least to those seen in icterus gravis. An analysis, however, of most of these cases reveals certain differences from the *post-mortem* findings in typical acute yellow atrophy either in the histological or chemical results of investigation of the liver or in the rapidity with which the condition develops. Thus, in the case of phosphorus and delayed chloroform poisoning, though the symptoms may closely simulate those of acute atrophy, there is a degree of "visible" fatty change which is not apparently seen in the classical acute atrophy, as was pointed out by Sir H. J. Stiles and myself⁴ in a paper on delayed chloroform poisoning in 1904. Tiletson⁶ has recorded a case of acute atrophy, apparently resulting from mercury, where syphilis was not present. I have only found one definite reference to arsenic as a causal agent.

Severin⁶ in 1912 described cases of acute atrophy in syphilitic cases under treatment by salvarsan, though Wells⁷ (1914) refers generally to such cases, and doubtless an examination of recent literature, to which I have not access at present, would reveal other examples.

Fresh interest in the subject has been stimulated since the outbreak of war by the study of toxic jaundice in munition workers, in whom a large number of cases of jaundice have been observed. In 1916, 181 cases occurred in T.N.T. factories in this country alone, with 52 deaths. The subject was discussed at the Royal Society of Medicine in January, 1917,⁸ and the Medical Research Committee has recently issued a monograph on the subject, which I have not yet seen.*

The trinitrotoluene, according to the investigations of Dr. B. Moore, appears to be largely, if not entirely, introduced into the body through the skin, and the *post-mortem* results reveal a condition, as M. Stewart states, somewhere between subacute atrophy of the liver and portal cirrhosis. One case, however, which he describes, though not in detail, in his contribution to the discussion at the Royal Society of Medicine, would appear in its clinical course and morbid anatomy to be indistinguishable from a typical case of acute yellow atrophy.

It may be remarked that these cases occurred particularly in munition workers who had shown definite symptoms of toxic gastritis, and it is possible that even in

them the gastro-intestinal catarrh is in some way associated with a secondary intestinal infection, acting on a previously damaged liver. *Post-mortem* examinations in these, for the most part subacute cases, might not reveal the microbic invasion which has been so marked in the present series. Still less likely would *post-mortem* examination throw much light on the etiology of the by no means inconsiderable number of cases of subacute atrophy which occur for the most part in children without any previous history of "poisoning" or infection.

These cases have not till comparatively recently been widely recognized, in this country at least. In 1908, together with Dr. Lindsay S. Milne,⁹ I published a series of five such cases in which the clinical histories and *post-mortem* appearances left no doubt as to their true nature, but owing to the longer clinical course run, little light could be thrown on the primary cause of the condition.

In still another group of cases, namely, undoubted septicaemia from various organisms, appearances in the liver simulating typical acute yellow atrophy are sometimes observed, but a critical study of such cases reveals some differences from the conditions observed in the so-called idiopathic acute atrophy.

The bacteriology of acute yellow atrophy has so far been very indefinite. This is easy to understand when one remembers that the liver is endowed with special functions for resisting microbic invasion from the alimentary canal. The Kupfer cells lining the sinusoids in the liver have, as we know, a powerful phagocytic action, and it is difficult to imagine that such a mass of highly specialized and actively functioning tissue as the liver presents is not intimately concerned with immunity processes in general. If anything, then, of a toxic nature acts on the liver, as is evidently the case in acute yellow atrophy, secondary microbic invasion is almost to be expected, and it becomes a matter of considerable difficulty to differentiate between a possible exciting agent of prime importance and a number of more or less harmless saprophytes, some of which may have entered the body after death. The isolation of the causal organism under these circumstances, if there be one, must not be expected to be an easy matter.

That infection of some kind apart from syphilis plays some part in the etiology of acute atrophy has long been held. In a large number of recorded cases some previous intestinal catarrh has been noted. The liver being so markedly affected in this disease, it has been justifiably held that the poison, whatever it may be, has been conveyed through the portal circulation.

In some cases *Bacillus coli* has been isolated *post mortem*; most observers, however, have regarded this as a condition without special significance and almost to be expected. Reichmann¹⁰ in 1908 isolated from the blood a sporulating Gram-negative bacillus, but as it has no special pathogenic power he did not regard the observation as important.

Apart from intestinal infection in a number of cases, so-called influenzal attacks are supposed to have played some part in the causation of the disease. Miller¹¹ in 1908 recorded a case in which active pulmonary tuberculosis was present, and was inclined to think that the tuberculous toxins had some causal effect. It may be noted, however, that there was at least a strong suspicion that this patient was syphilitic.

Finally, to apply these various suggestions to our present series of cases:

We have here five typical cases occurring together in a limited period; all have been syphilitic subjects, and all have been treated with salvarsan preparations *plus* mercury.

It is remarkable that though similar cases amounting to thousands have been treated by the same methods, such a complication as we have recently experienced has not been observed until now. This would seem to point to some other factor having been introduced. There is no evidence at present that the salvarsan has materially altered, at least in the last three years, and the syphilitic toxin and mercury factors may be taken as constant. Together with this, we have the observation that in each case there was found at the *post-mortem* examination microbic infection of a special type.

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 the syphilis, and possibly arsenic *plus* mercury, has completed the damage

* Readers interested may refer to the official memorandum published in the BRITISH MEDICAL JOURNAL (December 16th, 1916, p. 842), which contains a section dealing with this subject.

to the liver cells, and allowed autolysis of the tissue, which appears to be the essential liver change, to occur. Some such combination of circumstances might account for the rarity of the disease.

It is evident that a very considerable amount of work from a histological, chemical, and bacteriological standpoint is necessary in the cases already observed. I am indebted for assistance to Mr. P. C. Laws, M.A., in investigating the bacteriology of the cases.

It should be noted that neither clinically nor pathologically do the cases observed bear any close similarity to cases of spirochaetal jaundice.

Professor R. A. Bolam, who has seen some of the cases during life, is engaged with some others on the clinical side of the problem, and we hope in the near future to describe the cases in detail, and to give our conclusions, based on a more thorough study than other pressing work has permitted till now.

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PERICARDIAL KNOCK.

BY

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THERE is a sound heard on auscultation in cases of penetrating wounds of the chest near the cardiac region which is unlike anything met with in civil practice. Its origin is, I believe, not yet established. The occurrence of this sound is now familiar to most of those who are dealing with these cases at the clearing stations. It is of the nature of a click, and varies from a faint sound heard by careful auscultation, to a noise which may be compared to that heard in the ear-piece of a telephone when the lever is moved up and down. It may be heard sometimes when standing at the foot of the patient's bed. Sometimes the patient is conscious of it, and on one occasion has himself called my attention to it. In nearly every case the sound disappears after forty-eight hours, although in one patient who was recovering from a chest wound without any complications the sound persisted for ten days. He was then sent to the base, and I have no further information about him. The sound has been, as a rule, most marked near the apex beat. It is usually double, corresponding to the heart sounds, and its intensity waxes and wanes with respiration, sometimes disappearing completely in full expiration. A distinct fremitus can be felt by the hand laid on the chest.

The first case in which I noted it was shown to me by Captain W. W. Rees, R.A.M.C. I then suggested that it was due to a shell fragment lodged where the pleura and pericardium are in contact. I thought this possible as I had previously heard a metallic rumbling sound in a case in which a shrapnel ball was seen by x-ray screen in the costo-diaphragmatic angle moving up and down with respiration. A skiagram in Captain Rees's case showed the fragment to be in the neighbourhood of the pericardium.

That this explanation was not the correct one was proved by the case of an officer shown to me by Lieut.-Colonel Newlands of the A.A.M.C. The characteristic sound could be heard when standing beside the patient's bed. The entry wound was outside the heart on the left side, and the track passed in the direction of the pericardium. Two days later a right-sided subphrenic abscess developed and was drained. The foreign body was found lying in the right side of the abscess cavity. An infected haemothorax was also drained. The patient died a few hours later, and at the autopsy a "shaggy" purulent pericarditis was present. This case showed clearly that the foreign body was not itself directly responsible for the sound.

In the next case I saw the foreign body was visible on the screen in the cardiac area. It moved continuously

with the heart beat, so that a skiagram showed only a blurred image. This patient died, and at the autopsy the pericardial surface was normal. There was no infection, exudate, or any sign of pericarditis. The cavity contained a few drops of blood-tinged serum due to the passage of the missile, which had traversed the pericardium and lodged in the interventricular septum. This case, as also several others in which the "knock" disappeared after twenty-four or forty-eight hours in patients who never had serious symptoms, shows that pericarditis is not the cause.

I may note in passing that the continuous movement of the foreign body shadow with the heart beat, as distinguished from an oscillatory movement at certain phases of the respiratory and cardiac rhythm, has indicated that the foreign body is actually in the heart wall or cavity.

It has been suggested that the sound is due to emphysema of the connective tissue of the mediastinum, but in one case the missile passed through the chest antero-posteriorly on the left side clear of the upper and outer margin of the heart. Moreover, a case has been reported by Captain Rees in which the sound was heard over the apex beat, which was normal in position. It was heard in systole and was loudest in full inspiration. The foreign body was seen on the x-ray screen moving with respiration, and in full inspiration there was an oscillatory movement communicated to it by the cardiac impulse. There was a clear space of some 2 in. between the heart shadow and that of the foreign body. The entry wound was over the lower end of the left scapula. Localization of the foreign body on anatomical cross-sections showed it to be lying behind the root of the left lung in close proximity to the rami bronchiales, and over 1 in. from the nearest point on the pericardium. The man's condition when he was sent to the base was very good.

The usual disappearance of the sound after a short period, and the absence of serious symptoms in many cases, suggests that it may be due to air in the interstitial connective tissue of the lung which is struck upon by the beats of the heart. Further records will doubtless throw more light on the subject.

A SIMPLE APPARATUS FOR NITROUS OXIDE-OXYGEN ANAESTHESIA.

BY CAPTAIN A. S. WILSON, R.A.M.C.

THE infrequent use of nitrous oxide-oxygen as an anaesthetic for major operations in the British Expeditionary Force would make it appear that its very great advantages have not been fully realized. The rapid induction of anaesthesia and the immediate recovery are most important factors; with any general anaesthetic, the shorter the duration of its influence the better for the patient. Nitrous oxide is shorter than any other.

It is by far the most agreeable anaesthetic to take, and struggling and resistance are uncommon. Post-operative nausea and vomiting are hardly ever encountered; headache, restlessness, and bronchopneumonia are much less frequent sequelae than after ether or chloroform.

With reasonable skill and care on the part of the anaesthetist it is by far the safest general anaesthetic. Cyanosis under nitrous oxide, as under any other anaesthetic, is always a danger signal; when present, it is an indication that an insufficient proportion of oxygen is being supplied; such a mixture will never produce satisfactory relaxation, and usually results in muscular twitching and disturbed respiration. On the other hand, an excess of oxygen causes persistent movement, and prevents the onset of unconsciousness; such excess is frequently indicated by a red flush across the patient's forehead.

Desirable variations in the depth of anaesthesia to suit the requirements of different phases of an operation are much more rapidly and safely attained than with ether or chloroform. It is beyond the scope of this short paper to reiterate Crile's well-known findings on the physiological advantages of nitrous oxide as an anaesthetic.

Even after a prolonged administration, lasting up to two hours or more, the patient always completely recovers from the anaesthetic before leaving the operating table; this is invaluable when the number of post-operative cases is so large that it is impossible to provide a sister or trained orderly to stand by each patient till he is round