the cycle, instead of running to waste. If the flow is adjusted as described, the pressure within the bladder during expiration should be less than within the face-piece, the valve of which will therefore be kept shut, and consequently no oxygen will pass into the mask. (4) During inspiration the bladder acts as a reservoir of supply. If there were no such reservoir the patient would be dependent solely on the oxygen flowing through the tubing at the time. Unless the flow was very extravagant he would quickly empty the tubing and feel suffocated.

Points of Importance

There are three important points in regard to oxygen cylinders which apply to all methods of administration, the one described above included, but which are not yet universally known; they are therefore mentioned. First, effective giving of oxygen by any face apparatus necessitates oxygen flows of the order of 5 to 8 litres per minute, and consequently it is desirable to use large cylinders (that is, 100 cu. ft.). The ordinary small 20-cu. ft. cylinders, so frequently seen, contain only approximately 570 litres and consequently can last for little more than an hour. Secondly, in order to determine how much oxygen there is within a cylinder it is essential that a pressure gauge be attached, otherwise it is impossible to have any idea. Thirdly, oxygen cylinders should always be fitted with automatic pressure regulators, securing delivery at a constant low pressure (for example, 5 pounds); otherwise—as when an adjustment valve alone is fitted—the delivery pressure falls all the time as the cylinder empties, and must necessitate repeated opening up of the valve unless the flow is correspondingly to fall.

Summary

A simple method is described of converting the civilian respirator to an oxygen face-piece apparatus.

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CONTUSION OF THE MYOCARDIUM*

BY

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A blow over the chest or compression of the thorax may give rise to a variety of traumatic heart lesions. Sudden death suggests ventricular fibrillation, which event has been recorded experimentally in dogs (Kissane, 1937). Any form of abnormal rhythm may result. Rupture of a valve may occur. In association with these clinical pictures there have sometimes been transient changes in the electrocardiogram, indicating myocardial injury in addition (Beck, 1935; Kissane, 1937; Warburg, 1938).

In considering traumatic lesions of the myocardium the following clinical problems arise: Symptomless cases with complete recovery; delayed rupture of the heart; clinical features of a disordered heart; haemopericardium; persistent myocardial weakness; heart-block; coronary thrombosis; myocardial injury in association with angina pectoris, auricular fibrillation, or valve rupture.

There is evidence that any part of the heart may be bruised or that any one of the chambers may be ruptured.

* Based on a communication to the Association of Physicians of Great Britain and Ireland on May 10, 1940.

The site of the lesion is not necessarily in relation to the situation of the blow. The anterior aspect of the right ventricle is injured most frequently, but Beck (1935) records the case of a man who fractured his sternum on the steering wheel of his car. When he died on the sixth day myocardial bruising was found on the posterior aspect of the heart. In many cases fractures of the bones of the thorax occur, and distress from this cause may mask the symptoms of heart disability. But there are many records of myocardial injury in the absence of fractures, and indeed without any signs of external bruising. Bilderbeck (1919) in an adult, and Hamilton (1934) in a child, have recorded traumatic rupture of the heart immediately fatal without external injury.

Symptomless Cases with Complete Recovery

During the past two years Dr. G. R. Osborn and I have been studying heart injuries in cases of fatal accident. These have been more frequent than we expected. We have paid particular regard to less serious lesions, compatible with recovery, in those who have died from causes apart from the heart. These have been sufficient in number to suggest that recovery from undiagnosed myocardial contusion is by no means rare. The following case is an example of a well-defined lesion:

A man aged 58 was admitted after being knocked down by a motor-bus. He died the next day from cerebral injuries. The heart had received no special consideration during life. Necropsy showed that the cerebral injuries would account for death. A contusion about an inch in diameter was present over the lower part of the right ventricle in front; it did not involve the endocardium and there was no fluid in the pericardium. Two ribs were fractured on the left side, but the bones were not displaced and the parietal pericardium was uninjured.

From this heart lesion recovery was possible, and perhaps without any symptoms to suggest a contusion. More probably after an interval there would have been some distress from involvement of the pericardium.

In another motor accident case, in which death was due to a fractured skull, bruising of both the anterior and the posterior aspect of the heart occurred, but the endocardium was not injured. The pulmonary artery was ruptured, but the myocardial bruises would not have been fatal.

Delayed Rupture of the Heart

The most convincing evidence that a blow over the chest may injure the myocardium comes from recorded cases which may be described as delayed rupture of the heart.

A recent example (Priest, 1939) is that of a young Sepoy struck over the front of the chest by a cricket ball. After a short interval he continued to play throughout that day, was all right that night, and played cricket throughout the second day. That evening he complained of pain in the chest and died shortly afterwards. At necropsy a rupture of the right ventricle was seen to have occurred, with haemorrhage into the pericardium. In effect this young man played cricket for two days with a symptomless myocardial contusion.

This rare occurrence has been recorded by others. Groom (1897) notes that in a boy of 16 years who died suddenly a month after a chest injury the myocardial rupture appeared to have developed from the endocardium outwards. In this connexion my colleague Dr. Osborn has shown me an interesting specimen from a fatal accident case in which the right ventricle was ruptured but in the left ventricle there was a rupture of the endocardium, with the outer part of the heart muscle and the pericardium intact. Presumably the lesion in the left ventricle alone would have given rise to no symptoms until fatal rupture took place, or perhaps a cardiac aneurysm

might have resulted. O'Neill (1914), Gunewardene (1934), and others have described cases of delayed rupture of the heart following a blow to the chest or compression of the thorax. Rupture has taken place at varying intervals, most commonly between one and two weeks after the injury, but sometimes considerably later.

It is reasonable to suggest that this rare event is probably the result of a blow which ruptures the endocardium.

Clinical Features of a Disordered Heart following an Injury

There is ample evidence that direct trauma to the chest or compression of the thorax may produce myocardial injury. The study of case histories is therefore probably the most important means of making a diagnosis. The physical signs of localized myocardial injury will be negligible unless some transient changes in the T wave of the electrocardiogram can be detected. Beck (1935), Kissane (1937), and Warburg (1938) have recorded such findings; and the first-named two found them comparable to experimental records in dogs. But such signs must be sought for early, and, however favourable the opportunity, they will be absent in many cases. Radiology will confirm signs suggesting a pericardial effusion, but small haemorrhages will escape detection.

In the clinical history of trauma of a valve the symptoms of distress are immediate, but in myocardial contusion it is more probable that there will be an interval between the injury and the development of the more obvious symptoms pointing to a cardiac lesion. This is illustrated by the two following examples:

A lorry-driver aged 40, in excellent health, was lifting blocks of limestone into the lorry when one broke and a large piece struck him on the front of the chest, knocking him backwards. He felt a sharp "stitch" above his left nipple, but there was no surface injury. He ceased loading and rested a little, but felt quite well and drove the lorry for several hours. He went to bed apparently all right, but woke up with precordial pain, dyspnoea, a cold sweat, and his legs numb. His doctor on being called only recognized a rather weak but regular pulse. He remained in bed seven weeks, with some pain and discomfort in the left side of the chest. When I saw him two months after the accident no abnormalities could be detected on physical examination or with radiology or the electrocardiogram. He still complained of a feeling of weight in the chest and inability to exert himself. His exercise tolerance, tested by climbing the stairs, was poor. Six months after the accident he had recovered completely.

A healthy man aged 51 was walking in a garage in the dark. He fell into the inspection pit and struck the front of his chest on the opposite side of the pit. He was taken by car to hospital, where fractures were excluded. There was a feeling of bruising in the chest, but no serious distress. He was strapped up and sent home by car. He walked upstairs to bed quite well. In bed he could not raise his arms properly on account of bruising of the chest muscles. By the third day he was much better, and set out to walk to hospital to report. He was pulled up by a severe attack of dyspnoea, and had to be helped home. The distress passed off with rest. He was in bed several days. When I saw him two months later there were no abnormal signs, and an electrocardiogram and an x-ray examination were normal. His response to exertion was poor. He has since improved, but a year later his exercise tolerance is poor.

In a third case (Barber, 1938), in which a moderate degree of pulmonary oedema and expectoration of clear frothy serum suggested an inefficient left ventricle from contusion, the symptoms were in evidence on the second, third, and fourth days after the blow over the sternum.

If there has been a chest injury, whether severe or apparently slight, any symptoms suggestive of heart dis-

ability must be considered. After an interval, which varies in time, there may be dyspnoea, a feeling of oppression in the chest, perhaps with anginal pain and possibly the expectoration of frothy sputum. Sometimes the symptoms come on immediately. Obvious symptoms followed by complete recovery furnish reasonably clear proof of a contusion. Signs of cardiac inefficiency that persist may suggest disease from natural causes, but a carefully assessed history which points to trauma must receive due regard.

Haemopericardium

Rajasingham (1939) records the case of a young woman admitted to hospital with a large pericardial haemorrhage. The day previously she had received a blow on the front of the chest from a fist. There were no external injuries. She was treated successfully by aspiration.

It is possible that routine radiological examination of chest injuries in the accident wards would reveal some unsuspected haemorrhagic pericardial effusions. Probably the symptoms of heart distress which follow a blow arise when the contusion involves the pericardium, with perhaps a small haemorrhage. A recent case in an accident ward at the Derbyshire Royal Infirmary is suggestive of survival after such a haemorrhage:

A police sergeant aged 50 was involved in a motor accident; five ribs on the left side and also the right scapula were fractured. Some time during the first week in hospital he had a collapse, the description of which is suggestive of compression of the heart from haemopericardium. Six months later radiological examination revealed an appearance of the left border of the heart shadow which probably indicated an abnormality of the pericardium. He has never regained his full response to exertion; and the case serves to illustrate the probability that extensive thoracic injuries may obscure a traumatic heart lesion.

Persistent Myocardial Weakness

I have a note (Barber, 1912) of a man with a permanently inefficient heart following the fracture of several ribs. A few similar cases have since been met with (Barber, 1938), and also a more recent example. Warburg (1938) describes a number of them.

From the recorded cases it is to be noted particularly that if there has been severe chest injury with fractures the immediate history, so important for diagnosis, has been obscured by the distress due to the thoracic lesion. The patient's condition months after the injury has been very similar to that of the senile heart. There have been no abnormal physical signs, and the heart has been normal at rest. It has appeared to be quite incapable of responding to anything like the degree of exertion of which a man of the patient's age and muscular development should have been capable. In contrast a follow-up has shown that a number of men who had sustained severe thoracic injuries, in some cases with pneumothorax, had recovered completely, with good exercise tolerance.

Myocardial weakness is a designation rather ill defined, but it is an accurate description of the apparent condition. These men have been quite free from the nervous element so common in patients with effort syndrome and in other similar conditions. In one or two instances a certain degree of angina of effort has been present.

There is some recorded evidence of post-mortem findings in those who have survived a myocardial injury for a considerable period. Sufficient clinical records of these cases are now available to make a study practicable. Barber and Osborn (1937) recorded a post-mortem finding of deep-seated scar tissue in the heart muscle in a man who survived a traumatic mitral-valve lesion for twenty-two years.

Heart-block and Coronary Thrombosis

Heart-block produces a well-defined clinical picture. Following trauma it may be considered to be proof of a deep-seated contusion. The most convincing cases are those in which it has been temporary. Coffen (1930) has recorded an example in a child and Warburg (1938) has collected others. Kissane (1937) has produced heart-block in dogs experimentally from a blow over the heart. Warburg (1939) writes: "Traumatic auriculo-ventricular block is not excessively rare." Last year I obtained a clinical history very suggestive of this condition in a hospital sister who fractured her sternum. The accident took place abroad, and the opportunity of proving the case was therefore lost. Typical coronary thrombosis proved by the electrocardiogram may come on a short time after a well-defined chest injury (Beck, 1935). Warburg (1939) has collected seven recorded cases of traumatic coronary thrombosis. A myocardial bruise or partial rupture may involve a coronary artery, particularly if it be already diseased. This exceptional accident is within the possibilities of clinical medicine.

Angina Pectoris, Auricular Fibrillation, or Valve Rupture

Clinical pictures typical of these well-defined conditions may follow a blow over the chest. Sometimes there is proof that the myocardium has been injured (Beck, 1935; Kissane, 1937; Warburg, 1938; Barber and Osborn, 1937). A discussion of these conditions would extend the scope of these notes, and the same applies to traumatic pericarditis. Numerous cases are cited in the literature comprised in the references below. Some degree of angina of effort is so common in cases of myocardial contusion that it may be regarded as one of the symptoms. It is possible, however, that a genuine angina syndrome may develop after a chest injury in the absence of definite anatomical changes.

Summary

The clinical and pathological evidence in relation to myocardial contusion is discussed.

The varying clinical pictures are described.

The judicious assessment of the clinical history and the clinical features is the most important guide in diagnosis.

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F. Salmen (*Thèse de Paris*, 1940, No. 55), who records three illustrative cases in children aged from 10 to 13 years, states that whereas it used to be thought that Weil's disease was confined to adults, a certain number in recent years have been recorded in children. Its supposed rarity in children is attributed to difficulty in diagnosis by reason of the atypical or abortive character of the disease at that age. In the abortive form the symptoms consist merely of a meningeal reaction or actual meningitis with or without a very slight degree of jaundice. In another form the disease assumes the appearance of typhoid fever as in the pure febrile leptospirosis of the adult. The diagnosis in such cases can only be made by careful and repeated laboratory examinations such as Martin and Pettit's serum test and inoculation of the cerebrospinal fluid, blood, and urine into a guinea-pig.

ANAPHYLAXIS AFTER INJECTION OF TETANUS TOXOID

BY

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The following report appears to be of interest owing to the recent attention which has been drawn to rare non-fatal occurrences of this nature. Whittingham (1940), Parish and Oakley (1940), and Cooke, Hampton, Sherman, and Stull (1940) have drawn attention to this condition in some detail and have instanced a small number of cases.

Case Report

A healthy member of the nursing staff—J. A.—volunteered for immunization with tetanus toxoid. The only item of interest in her past history was that she had had an injection of diphtheria antitoxin in 1931 that was followed some days later by an intense urticarial rash with joint pains and general malaise. She had had three injections of T.A.B. vaccine between September 16 and October 14, 1939, without untoward effects.

On October 21, 1 c.c.m. of tetanus toxoid (B. W. and Co.) was injected subcutaneously into the upper arm. There were no ill effects until November 11 (that is, three weeks after the original injection), when a sudden rigor after a game of tennis was quickly followed by an intense urticaria preceded and accompanied by marked skin irritation. The lips, eyelids, and tongue began to swell, causing great mental agitation, but all the signs abated when 0.5 c.c.m. of adrenaline diluted 1 in 1,000 was administered. On December 4 the second injection of tetanus toxoid was given, but in view of the previous history 5 minims of adrenaline was injected with 1 c.c.m. of tetanus toxoid into the upper arm. Five minutes later tingling of the ears and some skin irritation were experienced; all went black before her eyes and she collapsed. A further 5 minims of adrenaline was given with the usual treatment for shock, and as she regained consciousness there was a rigor with vomiting and diarrhoea. The patient felt very poorly afterwards for about twenty-four hours.

Dr. H. J. Parish of the Wellcome Physiological Research Laboratories, Beckenham, very kindly came to the Park Hospital on February 27, 1940, and tested the skin reactions of the anaphylactic subject and five other volunteers, all of whom had had tetanus toxoid injections without any ill effect. The solutions used were Witte peptone, beef extract, and tetanus toxoid, and the results are briefly summarized in the accompanying table.

Initials of Subjects tested	Previous Injections					Intradermal Tests (0.1 c.c.m.)				
	Diph. Antitoxin	Sc. Fever Antitoxin	Staph. Toxoid	Diph. Formo' Toxoid	T.A.B. Vaccine	History of Serum Rash	Witte Peptone 1/10,000	Witte Peptone 1/1,000	Beef Extract 1/1,000	Tetanus Toxoid 1/1,000
J. A. . .	1931	—	—	—	1939	1931	+++	Not carried out	+	+
A. A. C.	—	1933	—	—	1939	1933	++	++	—	±
M. H. . .	—	—	—	—	1939	—	++	++	—	—
E. M. . .	1919	—	—	—	1940	—	++	++	—	—
N. K. . .	1931	—	—	—	1939	—	++	++	—	—
N. W. . .	—	—	—	—	1939	—	++	++	—	—

The reactions from the intradermal test in patient J. A. were much greater using Witte peptone 1/10,000 than in the other volunteers when 1/1,000 dilution was used. There was immediate pseudopodial wheal formation, which at the end of half an hour measured 20 by 25 mm. when the surrounding flush measured 45 by 70 mm. A faint punctate erythema also spread up the arm towards the shoulder, but there was no general reaction. Owing to the severity of the local reaction we did not carry out a test with 1/1,000 Witte peptone. The evidence all