Clinicopathological Conference**A CASE OF CRUSH INJURY OF THE CHEST***A Saturday Forum held at the Western Infirmary, Glasgow, on Saturday, October 22, 1960***Chairman: Professor E. J. Wayne, M.Sc., Ph.D., M.D., F.R.C.P., F.R.F.P.S.G.****Editors: H. I. Tankel, M.D., F.R.C.S., and
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FIG. 1.—(a) Shows the central type of crushed chest in which there is a transverse fracture of the sternum. (b) Shows the lateral type in which the sternum is intact.

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MR. D. H. CLARK: Professor Wayne, Ladies and Gentlemen, I wish to present a case of crush injury of the chest. There are two common types of crush injury (Fig. 1), a *central type* in which the sternum is fractured, and a *lateral type* in which there is a large number of fractures of the ribs on one or both sides. The patient I wish to present was of the latter type and had two large lateral flail fragments.

The patient is a labourer, aged 40, with chronic

bronchitis. On September 8th he and a workmate were at the bottom of a 12-ft. trench when the sides suddenly caved in. His workmate was killed and the patient sustained a severe crush injury of the chest. He was admitted to this hospital and, although not *in extremis*, he was seriously ill and so distressed that oxygen was immediately administered. He was sweating, cyanosed and in severe pain. He had marked surgical emphysema of his chest, fore and aft, on his neck and spreading

FIG. 2.—The patient in bed showing the tracheostomy, a water-seal drain in the second right interspace and traction applied to wires around the ribs.

on to his face. Paradoxical respiration was obvious in two large lateral segments. The blood pressure was 150/90 and the pulse rate about 100. An X-ray was taken which showed fore and aft fractures of the 5th to 9th ribs inclusive on both sides and surgical emphysema, but no evidence of pneumothorax.

We watched the patient while an intravenous infusion was being installed and reached the following conclusions: (1) He had not lost a great deal of blood; (2) he had a purely thoracic cage and lung injury and there was no intra-abdominal lesion; and (3) he had no sternal fracture. We decided to proceed with the anaesthetic, and this was begun by Dr. Fleming who was later joined by Dr. Wishart. As in all these cases the patient improved immediately. However, the improvement was short-lived; in about 10 minutes he began to deteriorate, and the anaesthetists reported that the amount of pressure required to inflate his lungs was increasing. A spontaneous tension pneumothorax on the left side was diagnosed and an intercostal water-seal drain was inserted through the second left interspace, whereupon air came out under pressure. The

operation then proceeded and consisted of putting slings of wire sheathed in polythene tubing round the 7th and 8th ribs on both sides in the mid-axillary line. A tracheostomy was then performed. Towards the end of the operation it was again reported by the anaesthetists that there was difficulty in inflating the lungs. Further X-ray of the patient now revealed a tension pneumothorax on the opposite (right) side. An intercostal drain was inserted in the second right interspace and again air came off under pressure. The patient was then transferred to the ward and the slings were attached to weights over the sides of the bed. He was given no oxygen. Further progress will be discussed under separate headings.

The Thoracic Cage

After traction was applied the rib cage gave no further cause for concern. Fig. 2 shows the patient in bed with traction applied. He was allowed up on the 11th day, and Fig. 3 shows him standing with the traction still maintained by means of a spinal brace to which encircling arms have been attached. The emphysema subsided rapidly and he lost a considerable amount of

FIG. 3.—The patient standing. Traction is maintained by means of the encircling arms attached to a standard spinal brace.

weight. On the 15th day after operation one wire cut out. The traction was released on the 18th day.

The Lungs

Both intercostal drains were removed on the 2nd day and from then on his left lung gave no further trouble. By the 3rd day it was noticeable that the right lung was not inflating properly. The respiratory murmur was poor and there appeared to be a collapse of the right lower lobe

and probably also of the middle lobe. Bronchoscopy was performed under local anæsthesia but did little good. He continued to be very ill with a high temperature, rapid respiration and cyanosis. On the 5th day after operation air again began to leak from the right supraclavicular region with emphysema spreading subcutaneously over the chest and into the neck and face. X-ray confirmed the presence of a further pneumothorax and an intercostal drain was again inserted in the second right interspace. Thereafter he gradually

FIG. 4.—Blood pressure chart showing hypertension due to carbon dioxide retention during the first week. The late readings show the patient's normal levels.

improved, despite a hæmoptysis on the 14th day, which was felt to be due to secondary hæmorrhage from lacerated lung or the opening of an area of atelectasis. The most recent X-ray shows that the right lung has cleared considerably though there is probably still a very small pneumothorax at the apex.

I do not think there is any doubt that this man had CO_2 retention for at least ten days. Unfortunately we have no readings of the arterial CO_2 tension. However, we did record his blood pressure faithfully and I have summarized the findings in Fig. 4. His blood pressure on admission was 150/90, and during the first few days it rose, until on the 4th and 5th days it was in the region of 190/120. In the two or three days prior to his discharge from hospital his blood pressure remained in the region of 120 to 130 systolic, and this would appear to be his normal blood pressure. We submit this as evidence that he probably did have a significant degree of CO_2 retention.

The Tracheostomy

He had a tracheostomy for 24 days. At first a cuffed tube was used and we released the cuff for five minutes in every hour. Aspiration of the bronchi through a Tieman's catheter was required every half-hour and sometimes more frequently

as he was a chronic bronchitic. As time passed the amount of aspiration required became less. After about a week the rubber tracheostomy tube was changed for a metal one, and he found this more comfortable. These tubes were changed regularly.

Bacteriology at different times revealed the presence of staphylococci, *B. proteus*, diphtheroids and streptococci. The only antibiotic to which all were constantly sensitive was chloramphenicol.

At this point the patient was presented and the chest expansion demonstrated to be 1 inch. The chest was stable and the wounds healed. There was no cough.

PROFESSOR WAYNE: Thank you very much, Mr. Clark. It seems that many of the problems had to do with the anæsthetic and I would ask Dr. Wishart to talk about these.

DR. H. Y. WISHART: The main interest in this case from our point of view was the fact that the patient came in without a tension pneumothorax and that this became evident during anæsthesia. The patient was curarized in the normal way and breathing was being controlled by hand when it was noticed that increasing pressure was needed to do this. When this happens there may be three causes: (1) The muscle paralysis may be wearing off, (2) secretions may be blocking the tracheo-bronchial tree, or (3) a tension pneumothorax

may be developing. In this case the patient was making small respiratory efforts and so he was reoxygenated but without improvement. He was sucked out and again there was no improvement, so of course tension pneumothorax was suspected, although breath sounds could be heard. When a drain was put into the left side of the chest a large amount of air under pressure came off.

When a patient is admitted with a crushed chest, and surgical emphysema is present, you must suspect laceration of lung tissue or surgical emphysema would not be there. You must therefore expect a pneumothorax to be present, which may become the tension type. There is a view that if there is surgical emphysema present when the patient is admitted one of the first steps after the induction of anaesthesia should be to insert a drain intrapleurally to relieve any tension. As an alternative I would suggest that if you suspect a tension pneumothorax a syringe with a needle attached should be inserted into the pleura, and if there is a tension pneumothorax the plunger will come out. A water-seal drain can then be inserted.

PROFESSOR WAYNE: I wonder if Mr. Fraser has any comments to make as a thoracic surgeon interested in this particular problem.

MR. KENNETH FRASER: Mr. Chairman, first of all I want to emphasize that the most important person at the admission of these patients to hospital is the anaesthetist. By intubating the patient and controlling the respiration he will overcome the paradoxical movement of the chest. This will in turn control the restlessness of the patient; and this is an opportunity to point out that CO₂ accumulation can produce extreme restlessness and irritability. I am sure it is not always appreciated just how important this may be.

Next, it is important that having obtained an initial film with no pneumothorax present you should not be content to think that this situation is necessarily maintained until the end of the operation. One has only to open a chest with broken ribs, and see and feel the sharp edges, to realize that while the control of breathing improves the patient it may also produce a pneumothorax by rubbing the lung against the rough rib ends.

A further point is brought out by another patient I saw recently at the Vale of Leven Hospital. In addition to multiple fractures, intra-abdominal injuries and a similar bilateral crushed chest injury, he also had a traumatic hiatus hernia. The X-ray (Fig. 5) shows the stomach bubble within the chest about halfway up the clavicle. The point I wish to make is this, that before one intubates a patient with an injury like this one must pass a gastric tube to deflate the stomach.

FIG. 5.—X-ray of a patient with a traumatic hiatus hernia on the left side showing the stomach bubble high into the chest, and the mediastinum pushed over to the right. Fractures can be seen on the 4th-7th ribs. (Vale of Leven Hospital.)

If the patient is anaesthetized, curarized and put on to hand-controlled respiration without this precaution, then the right lung will be inflated and push the mediastinum against the grossly distended stomach. If the stomach is sufficiently distended the patient will certainly develop asystole during the course of the induction. Where you have such a hernia it is therefore imperative to introduce an intragastric tube to reduce the tension before proceeding to anaesthesia. If it is impossible to introduce the tube (due to rotation of the stomach) then I believe that you are justified in putting a trocar and cannula through the chest wall into the stomach and deflating the stomach by passing a Malecot catheter before doing anything else. In this second case I used the method of traction that Mr. Clark has already described with equally good results.

PROFESSOR WAYNE: We are fortunate in having with us this morning Mr. Sillar of the Southern General Hospital, who has been specially interested in this type of chest injury.

MR. W. SILLAR: Sir, this has been a most interesting case. There are a great many things one could comment on, but I would like to bring

up only one aspect of the problem at the moment and this is the question of the early anaesthetic. The patient may be admitted *in extremis*, he may be in an agonal state, and his life can be saved only by the immediate induction of anaesthesia; this must never be forgotten. But not all the patients are so nigh unto death and there are some disadvantages in immediate anaesthesia. You do not have an opportunity of examining the abdomen, you do not know what intra-peritoneal damage may have occurred, you do not have an opportunity to examine the central nervous system, and you are no longer in a position to assess accurately the damage to the rib cage since positive pressure anaesthesia stops paradoxical movement. All these are serious disadvantages which will have to be accepted and overcome when the patient is very seriously ill. However, if he is not so ill, a little time will enable us to clear up these points before we are committed to the operative procedure. If, therefore, the patient can be resuscitated and an air way guaranteed before he is anaesthetized, it might make the assessment of his injuries more complete and the planning of the operative procedure much easier. On the other hand, if you must anaesthetize him to save his life then there are some devices which can help. If necessary, a small mid-line laparotomy can be made and the wartime practice of plunging a swab on a holder into the four corners of the abdomen carried out. If the swab comes out clean, the little laparotomy wound can be closed quickly and not much time is wasted. It is important to determine the size and shape of the flail segment, and the anaesthetist may be able to oblige at a later stage, after the patient's condition has been improved, by allowing spontaneous respiration to return and then temporarily obstructing the airway to demonstrate paradoxical movement.

I would like to emphasize again the danger of a pneumothorax developing after anaesthesia. This is a real danger and one must be ready at once to deal with it.

PROFESSOR WAYNE: Thank you, Mr. Sillar, for that valuable contribution. Perhaps Dr. Pinkerton would care to comment further on the anaesthetic problem.

DR. PINKERTON: When these patients come to hospital they suffer from three major disabilities—pain, shock and respiratory embarrassment. There is no question that the respiratory embarrassment is the paramount condition and that it is due to paradoxical respiration and the presence of secretions. To anaesthetize the patient, intubate him and take control of the respiration produces an immediate and, to those who have not seen it an almost unbelievable improvement. The para-

doxical respiration is cured by the control of the respiration, the sucking out of secretions follows the intubation.

When the operation has been completed and such fixation as can be achieved has been done, the decision has to be taken on the matter of a tracheostomy and I would say no more than that if there is any doubt whatever as to whether a tracheostomy should be performed, and usually there is none, it should be performed. I think that there is a great value in biochemical control of these patients, and I think there is little doubt that the elevated blood pressure during the first ten days in this case indicated CO₂ retention. It is possible that more prolonged ventilation by means of a respirator would have improved this.

PROFESSOR WAYNE: I wonder if perhaps Professor Barnes as an orthopaedic surgeon interested in trauma would like to say anything.

PROFESSOR ROLAND BARNES: Like most people here I have very little experience of this type of injury, but I would like to ask some questions. One of the problems that we have not dealt with very effectively to date is the treatment of paradoxical respiration. There are two approaches to this. The first is the application of some form of fixation to the chest, such as the metal plate devised by Mr. Sillar for fixation of the sternum, or, as we have seen this morning, traction wires passed through the rib cage. The alternative, of course, is the control of paradoxical respiration by hyperventilation. I would like to hear a discussion of the merits of these two methods. We also have used wires through the thoracic cage, but it may be that these are liable to cut out, as we saw in today's case, and as Griffiths (1960) has mentioned in his recent communication to the Royal College of Surgeons of Edinburgh. For this reason he was a very firm advocate of hyperventilation. It may be that there is room for both these methods. If we have a sternal fracture is it better to deal with it by fixing it with a plate? If we have multiple rib fractures, would it be better to consider hyperventilation?

MR. SILLAR: I think that the problem of controlling paradoxical motion by a respirator is a difficult one. We have not had good fortune here in using a mechanical respirator. We used the Pneumatron and were not able to synchronize the patient with the machine. This has been the experience of others. In general there are four other methods of overcoming the conflict between the patient and the machine when dealing with a conscious patient who is not paralysed—which is a different problem from that presented by the patient with poliomyelitis: (1) *Muscle relaxants*; the patient's resistance can be overcome by muscle relaxants, though this is not a feasible pro-

cedure apparently in the conscious patient. (2) *Hyperventilation* as used by Professor Mörch in Chicago (Avery, Mörch and Benson, 1956). He seems to have had very good results indeed by deliberately over-ventilating the patient, inducing alkalosis and apnoea, and so keeping the patient alive for a considerable time without spontaneous respiration. (3) Dr. Griffiths in Edinburgh, not wishing to go all out for this method and not wishing to depress the patient with high dosage of morphine, decided to steer a middle course by using some morphine and a little hyperventilation to try to avoid the danger of both. He has three patients at least who have recovered on this management. (4) *Continuous flow*, a method of which we have no experience. A continuous flow of oxygen is provided at low pressure through the tracheostomy without a cuff. The patient's respiration then consists of expiratory efforts only against a continual gentle inflation.

The only method we have tried is, as I have said, the patient-triggered respirator which is supposed to detect instantly and to instantly augment the patient's own inspiratory effort before the flail movement of the damaged portion can occur. We have not been able to make this work, because we found it hard to synchronize the patient with the type of machine available, and so we have turned again to methods of stabilizing the rib cage. It seems to me that if normal respiratory physiology can be restored by repairing the rib cage this must be a healthier arrangement than deliberately blowing air down into a patient's lungs over a considerable number of days at the risk of interfering with the clearing of secretions.

MR. H. WAPSHAW: The treatment in this case has been very successful, but, if I may say so, the success has been partly due to the fact that the patient had no other injuries. The mortality in this condition is really dreadful—well over 60%—and that is everybody's experience. In these very serious chest injuries there are three main types. Mr. Clark has described two. There is a third, and the French have the only word for it, and that is bone 'marmalade'; in these cases, very little can be done for the patient.

MR. A. D. ROY: Why did you maintain the rib fixation for as long as 18 days? I would have thought that the ribs might have consolidated before then. How long does it take for these fractures to stabilize and consolidate if they are not fixed?

MR. CLARK: Rib fixation was maintained for so long only because the patient felt uneasy without it.

MR. FRASER: It is generally thought that 12 to 14 days is adequate for the ribs to find their own fixation. I would like to say a further word about

the Mörch machine of which we have already heard. It seems to me from reading the literature to be the one most likely to be suitable. Mörch does not use an inflated cuffed tube. He claims that if sufficient pressure is used, then the secretions are blown up the trachea and can be sucked out from the back of the throat or from the tracheostomy.

MR. T. B. GARDINER: I think that the point that Mr. Wapshaw made about associated injuries is a very important one. I think it worthwhile pointing out that the case with the traumatic hiatus hernia mentioned by Mr. Fraser had, in addition to lateral flail chest, a ruptured spleen, torn omentum and pancreas, and bilateral fractures of his transverse processes, femurs and ankles. His recovery was helped by an extensive transfusion and the fact that we did not have to do much to the fractures to secure stability.

DR. PINKERTON: I think there is a point we are missing in the discussion. If, in these crushed chest injuries, the flail segments can be securely fixed, clearly the patient may recover without the need of assisted respiration of any kind afterwards. This happened in this morning's case. Spontaneous respiration has clearly got him through, although at some little cost of CO₂ retention, and he has recovered most admirably as we can see. However, we are slipping past the point of the patient in whom adequate fixation is not achieved or is not possible. Such people must have some kind of controlled respiration with a mechanical respirator for some time. Even those who have had a reasonable fixation are usually the better for some form of assisted respiration. I would not attempt to improve on the description which Mr. Sillar has already given of the problems which face us there. There are plenty of such injuries but not many come into the hands of any one group, so that it is difficult to compare one method of treatment with another. The respirators vary in their ability to control and to assist and to go with the patient, and it is agreed that the triggering of respirators has not provided the answer to the problem. The hyperventilation method suggested by Griffiths and by Mörch in Chicago may possibly be an answer. It so happens that Dr. Fleming who was the first to anaesthetize this patient has recently returned from Switzerland and has seen something of the treatment of such cases there.

DR. FREDA W. FLEMING: It is sad to hear people say that patients cannot be maintained adequately on respirators, yet I suppose it shows our good fortune in not having many of these tragic cases to deal with. Both in the University Hospital in Lausanne and the Claude Bernard Hospital in Paris they have been handling respira-

FIG. 6.—The effect of the flail anterior segment in interfering with the descent of the diaphragm is shown.

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tory catastrophes of many kinds, including crushed chests, in special units. They use various kinds of respirators, but mainly the Swedish Engström which seems to overcome many of the difficulties which have been mentioned (Engström, 1953); patients can be adequately triggered, and they can continue using this machine even when more or less fully conscious and co-operative. Gases can be supplied in varying proportions as required and for a considerable time. The chest injuries are fixed mechanically as well, but the use of the respirator with adequate suction permitted the normal blood physiology to be preserved until it was quite obvious that these patients could maintain this function for themselves. It means, of course, an elaborate set-up with individual cubicles, much apparatus and specially trained nurses and physiotherapists.

MR. H. I. TANKEL: Mr. Sillar, in what proportion of these chest injuries is fracture of the sternum the main factor? Could you tell us something of your results to date with fixation of the sternum by means of a plate, and do you feel that internal fixation of the ribs is of any value?

MR. SILLAR: Of 35 patients seen in Glasgow

TABLE I
35 PATIENTS WITH FLAIL SEGMENTS

		Deaths
15 pure lateral segments		8
8 pure anterior segments		7
4 mixed type		3
8 details insufficient		7

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hospitals in four or five years up to 1960 (Table I), 15 had a pure lateral injury, and of these there were seven survivors; but of the eight cases in which the anterior segment bore the brunt of the damage there was only one. I think the reason for this significant difference is as follows. If the sternum is flail and a large part of the rib margin forms part of the flail segment also, then the diaphragm is denied its usual fixed anchorage anteriorly. The xiphisternum falls back during inspiration and so the diaphragm slides ineffectively over the underlying viscera but does not descend (Fig. 6). Thus there is a very serious inroad into the patient's vital capacity. When there is a lateral segment injury I do not think the diaphragm is usually affected to such an extent. We therefore searched for a method of fixing the sternum. Since collecting the 35 cases mentioned earlier, we know of six cases where

the sternum has been fixed by rigid internal fixation (Sillar, 1961) and with no slings; these have shown that this procedure can be effective. Two of them died, neither from causes related to the chest, and in every case paradoxical movement was controlled. We have tried to deal at the same time with the ribs by using little medullary rods of Kirchner wire, and this has sometimes been successful.

I hasten to say that it would be very, very foolish to regard this as a matter of competition between chest fixation and respirators. With these desperately ill patients, each presents a different problem. In the cases of multiple comminuted rib fractures the only possible chance lies in the use of a positive pressure respirator. The methods are complementary and I am sure we must be versatile, we must have a lot of methods at our finger-tips and in our minds if we are going to do the best for these patients.

PROFESSOR WAYNE: Thank you, Mr. Sillar. Could I just ask a question myself as a physician? Do you do electrocardiograms on these patients? Barber (1944), of course, showed that electrocardiographic abnormalities were very frequent in crush injuries.

MR. CLARK: We did not in this case.

PROFESSOR WAYNE: Is it generally done by surgeons or not? We have Barber's opinion that it should be; has this been assimilated by thoracic surgeons? I have only seen this point as a rule referred to in medico-legal circles, where it has been shown that there is a very considerable incidence of what has been called bruising of the heart, which must on occasions be of clinical significance.

MR. SILLAR: It was in fact shown at the Glasgow Royal Infirmary by Dr. Malcolm Fletcher (1960), when routine ECGs were done on a series of chest injuries, that some had tracings which were indistinguishable from those of coronary thrombosis. These tracings returned to normal, usually in two or three weeks.

MR. JACK STEVENS: On the matter of the tension pneumothorax which may occur during the induction of the anaesthetic, I think there is a good deal to be said for needling both sides of the chest as a routine at the time of induction of the anaesthetic for diagnostic purposes. This would also help in the diagnosis of hæmothorax. Griffiths (1960) describes a series of 38 patients with crushed chests, 29 of whom died; 21 of these 29 deaths he thought might have been avoided by better treatment of asphyxia, respiratory insufficiency and hæmorrhage. So there is a good deal of hope that we should be able to do more for these patients than we have been doing in the past.

DR. M. T. HARRISON: The danger of tension pneumothorax arising in this sort of injury has been very much stressed and rightly so. There is one other complication, however, which may be seen, namely, surgical emphysema of the mediastinum. This may produce angina-like pain, if the patient is conscious, and a crunching sound over the heart as the air churns around during the heart-beat (Hamman's sign). Later one finds subcutaneous emphysema in the neck and finally obstruction of the superior vena cava, cardiac tamponade and death. I wonder if we could hear a word about the incidence of this in the thoracic surgeon's experience and the best way to treat it. I wondered also if fat embolism should be considered as another possible cause for hypertension.

DR. W. C. DYKES: In the earliest stage of surgical emphysema, while the air is still confined to the pulmonary interstitial tissue, it can already make for ventilatory difficulty and reduced circulation and add to the difficulty of inflation from the anaesthetist's point of view. When the air reaches the mediastinum, and the mediastinal pressure rises, it produces the further effects Dr. Harrison has mentioned. As pointed out by Macklin (1937), tension pneumothorax may also be produced indirectly in surgical emphysema by splitting of the visceral pleura. As an interesting aside I understand that during the 1918 influenza epidemic a lot of people died in extreme cyanosis, dyspnoea and circulatory collapse and were found at necropsy to have extensive interstitial emphysema of the mediastinum. This complication can occur also with positive pressure respiration, whether intermittent or continuous.

MR. SILLAR: May I say one word about fat embolism? We had a patient in 1957 who appeared to be getting into great difficulties with his respirator before he died. At post mortem he was found to have widespread fat embolism. Indeed, the evidence was there before death in the form of small petechiæ. If fat embolism is a factor there should be signs of it providing the possibility is kept in mind, and the urine and lung fields examined.

DR. BERNARD LENNOX: In cases in which the chest has to be opened in the course of operation it should be simple to diagnose fat embolism very early by lung biopsy. Incidentally, I do not know of any evidence that fat embolism can cause hypertension under any circumstances.

PROFESSOR WAYNE: Would you care to say anything in summary, Mr. Clark?

MR. CLARK: I think that we have learned a number of lessons from this case. Here we had an example of a pure thoracic cage and lung injury in which tracheostomy and traction on the

flail fragments alone were successful. This is not always so. In more severe cases where fixation may be inadequate the use of a mechanical respirator will be necessary for a prolonged period.

The value of tracheostomy is not in doubt, but the aseptic management of it is very important and difficult to achieve.

The early induction of anaesthesia with positive pressure may be life-saving, but attention must be paid to the possible development of tension pneumothorax and hæmothorax. In severe cases it may be advisable to anticipate this by inserting intrapleural water-seal drains immediately after induction.

In the case of multiple extra-thoracic injuries, team-work is essential. It would seem to be ideal to have special facilities for this purpose. It is our opinion that more lives will be saved when such teams gain more experience in their management, are more aware of the problems involved, and become more skilled in the handling of the technological aids that are available.

Acknowledgment

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REFERENCES

- EVERY, E. E., MÖRCH, E. T., and BENSON, D. W. (1956): A New Method of Treatment with Continuous Mechanical Hyperventilation to Produce Alkalotic Apnoea and Internal Pneumatic Stabilization, *J. thorac. Surg.*, **32**, 291.
BARBER, H. (1944): The Effects of Trauma, Direct and Indirect, on the Heart, *Quart. J. Med.*, **13**, 137.
ENGSTRÖM, C. G. (1953): Respirator Designed According to a New Principle, *Svenska Läk.-Tidn.*, **50**, 545.
FLETCHER, M. (1960): Personal communication.
GRIFFITHS, H. W. C. (1960): Crush Injuries of the Chest, *J. roy. Coll. Surg. Edinb.*, **6**, 13.
MACKLIN, C. C. (1937): Pneumothorax with Massive Collapse from Experimental Local Overinflation of the Lung Substance, *Canad. med. Ass. J.*, **36**, 414.
SILLAR, W. (1961): The Crushed Chest—Management of the Flail Anterior Segment, *J. Bone Jt Surg.* In press.