TRAUMATIC DIAPHRAGMATIC HERNIA.
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This is not a rare condition. Chest units see it frequently; it is also a trauma that traumatic surgeons are likely to see. Traumatic surgery is one field of surgery in which surgeons in the Corps have considerable experience. During my service I have had something to do with three patients suffering from this condition.

American literature frequently describes traumatic diaphragmatic hernia but there are fewer references in British journals and one of the infrequent references is found in the JOURNAL OF THE ROYAL ARMY MEDICAL CORPS (Swinney). More recently in this Journal Vere Nichol briefly describes a case. And it is worth while mentioning that this lesion was first described by an Army Surgeon, Ambroise Paré (Morrison).

The purpose of this Paper is to remind surgeons in the Corps of this trauma to recall the thoracic approach (which gives excellent access for the reduction of the hernia and the repair of the defect) and to emphasize that a skilled anaesthetist is necessary.

In the three cases I have seen there were three common factors:—

(1) Desperately ill men on admission.
(2) History of crush accident.
(3) Diagnosis of traumatic diaphragmatic hernia suggested during the recovery from initial shock by hearing abdominal-sounds in the pleural cavity on auscultation of the chest and confirmed by means of a barium follow through.

This is well shown in the case-history of the third patient which is as follows:

One hour before admission to a hospital in B.A.O.R., Pte. R., aged 21, was run over by a wheel of a 3-ton lorry which partially compressed his left leg and abdomen.

When admitted almost moribund, he was sweating profusely, colour ashen, pulse just palpable and very thready. He was immediately given 2 pints of plasma in thirty minutes and morphia intravenously followed by 1 pint of blood more slowly. One hour later his colour and pulse had improved considerably but he was then cyanosed. His B.P. was 134/70.

At this stage examination showed: Dyspnoea with a respiratory rate of 36/min. He complained of severe pain in left upper abdomen. The apex beat was 1 in. inside nipple line, and the right border of the heart 2 in. to right of sternum, while the trachea was to right of the mid-line. Air entry was diminished over the whole of the left side of the chest and absent in lower axilla up to the level of second space. The chest was dull
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to percussion in this area. Compensatory emphysema was present in upper portion of left and whole of right lungs. The coin sounds in left side were suggestive of presence of air in the thorax. The abdomen was completely rigid all over, and hyper-peristalsis was audible. The liver dullness was present.

Three hours later: very much more dyspnœa with considerable bubbling in chest and cyanosis marked. Chest signs were more marked over the same area as noted before. The respiratory rate was now 48 but the pulse was improved. The head of the bed was raised, continuous oxygen through BLB mask started and atrophyine 1/100 gr. given.

Eight hours later: much improved. The colour and general condition were better. He complained of less pain. Respiratory rate was 36 and bubbling had decreased. Fourteen hours later improvement was maintained but he had twice vomited 2 oz. of stomach contents containing blood. I.V. plasma was discontinued.

One day after injury the respiration was 36 but he was less distressed. Chest signs were unchanged. Traumatic asphyxia of face and neck was now present. Abdomen: he complained of spasmylic pains in epigastrium radiating to the left shoulder. The abdomen was soft though still rigid in epigastrium. He vomited small quantities several times during the night, and blood was still present in the vomitus. He passed urine.

Further examination showed a hæmatoma in left sacro-iliac region. The right ankle was very swollen and painful. X-ray of chest, lumbar spine, pelvis and right ankle showed no bony injury. I.V. glucose saline was commenced and discontinued after 3 pints. He did not vomit for twenty-four hours in spite of being given fluids by mouth (sips). That evening the chest was aspirated but nothing was withdrawn.

Two days after injury the respiratory rate was raised. Chest aspiration again attempted but nothing found.

Four days after injury he was much improved but he still had occasional epigastric pain but no vomiting. Respiratory rate was 28 to 30 constantly.

Seven days after injury the chest was aspirated and 2 oz. of blood withdrawn from left pleura.

Nine days after injury auscultations in the left axilla revealed added sounds which were believed to be either bowel sounds or due to pleural friction. A barium meal was therefore given and revealed the stomach and transverse colon lying above the left diaphragm.

On the sixteenth day after injury the patient was evacuated by air to the U.K. and was admitted to this hospital.

At this time his only complaints were of dyspnœa with a feeling of epigastric discomfort after meals. The general condition of the patient was excellent and there was no cyanosis or dyspnœa at rest. He preferred to sleep propped up in bed. There was no tracheal displacement and the apex beat was one inch medial to the nipple line.

There were no abnormal physical signs in the right chest. There was diminished respiratory movement on the left, and the apex was resonant and the lower half of the chest hyper-resonant. Air entry was poor at the apex and absent in the lower two-thirds of the chest. Bowel sounds were heard over nearly the whole of the anterior part of the left chest and in the left axilla.

The heart sounds were normal and the blood-pressure was 110/90. The blood-count showed R.B.C. = 4.5 million, Hb. 87 per cent.

There were no abnormal physical signs in the abdomen. The barium meal was repeated and showed evidence of the stomach and the transverse colon in the left pleural cavity (see fig. 1).

Fig. 1 shows the high level of the diaphragm. Above it lies the air bubble in the fundus of the stomach and above that the air bubble in the colon.

Note how the duodenal spill outlines the first part of the duodenum, which, because it is mobile is dragged over to the left. The non-mobile second part remains on the right side.

Fig. 2 shows the left half of the transverse colon herniated into the chest.
and shows the hepatic flexure dragged to the left. It also shows barium residue in the oesophagus, the stomach, the duodenum and the herniated jejunum following the transverse colon into the chest.

He was allowed to settle down in the ward after his air journey and twenty-seven days after his injury he was submitted to operation.

The patient was grouped and cross matched for intravenous therapy during the operation.

A Ryle's tube was passed, in the ward before operation, through the previously cocainized left nostril.
Premedication was opioidine ½ gr. and scopolamine 1/150 gr. given by H.I. one and a quarter hours before operation.

Anaesthetic Technique.

A small dose of pentothal 0·3 grm. was given intravenously followed by cyclopropane and oxygen in closed circuit. A No. 10 Magill tube, lubricated with 10 per cent nupercaine ointment, was introduced orally under direct vision and connected to the closed circuit by means of a Cobb's connector. The circuit was made airtight by plugging the nostrils with wool and sealing the lips with elastoplast, no pharyngeal pack being used.

Intravenous glucose saline was set up before the operation commenced.

Blood-pressure and pulse readings were taken and charted every fifteen minutes during operation. Continuous stomach suction was maintained throughout.

At the point of closing the diaphragmatic tear the anaesthesia was deepened and controlled respiration was used in order that movement of the diaphragm should interfere as little as possible with the operative repair. After closure of the tear anaesthesia was lightened.

It will be seen from the chart that the systolic pressure maintained an even level throughout operation, but that a marked fall of diastolic pressure, and a moderate rise of pulse-rate, occurred during the replacement of the gut into the abdominal cavity and the repair of the diaphragmatic tear; these gradually returned to normal.

As the pleura was closed the lungs were inflated with oxygen under pressure, in order that a minimal pneumothorax should be left.

The patient's condition during operation remained satisfactory. The total intravenous therapy consisted of one pint of glucose saline during operation followed by one pint of blood at the end of operation and post-operatively.

The patient regained reflexes very shortly after his return to the ward.

The post-operative instructions were that the patient was to be nursed in the sitting position with the left side of the chest uppermost and that he was to

![Graph]

be given inhalations of 5 per cent CO₂ and 95 per cent O₂ for five minutes, four-hourly for forty-eight hours. The Ryle’s tube was left in situ.

**Operative Technique.**

The patient was placed on his right side with a pillow under his right chest and the left arm on an arm rest. The chest was opened by left oblique incision over the 7th rib, which was excised for a distance of five inches, commencing from the neck of the rib. The pleural cavity was then opened and rib retractors inserted. The cavity was found to contain nearly all the stomach, the spleen, the transverse colon, the greater omentum and about two feet of the jejunum, all without peritoneal covering.

Reduction was difficult and although continuous gastric suction was maintained during the operation, the stomach could not be deflated because the Ryle’s tube remained sub-diaphragmatic and did not empty the supra-diaphragmatic portion of the stomach.

However, by continuous pressure, part of the stomach was reduced and the stomach gradually became deflated. Thereafter reduction of all the abdominal contents was relatively simple.

The rent could now be clearly visualized and consisted of, first, an avulsion of the origin of the left diaphragm from the costal cartilages and the sternum. This hole was plugged with very adherent omentum which was freed and replaced in the abdomen. Secondly, extending from this point was a linear tear 5 inches long running obliquely, laterally and backward across the diaphragm. It was found that the exposure was not sufficient to repair the defect and the remainder of the 7th rib was removed by dividing the rib at the costochondral junction. The exposure was now excellent.

The diaphragm was practically immobile (controlled respiration) and so the easily seen phrenic nerve did not need pinching or injecting with local anaesthesia. The tear of the diaphragm was repaired in two layers using No. 3 chromic catgut and the avulsed portion of the diaphragm was reattached to the cartilages by mattress sutures through and around the costal cartilages. This latter was the difficult part of the repair.

The lung appeared to be completely collapsed against the spine with multiple small, red, fleshy, non-bleeding adhesions between lung and lung, and lung and pleura. A considerable number were broken down and the chest closed with pericostal sutures and suture of the intercostal muscles and skin without drainage.

Before closure the diaphragm was seen to be working again and as the pleural cavity was closed the lung was inflated with oxygen under pressure.

At the end of the operation (one and a half hours) his general condition was excellent and there was no rise in his pulse-rate or blood-pressure.

**Progress.**

The day after operation the patient became increasingly dyspnoeic. This was relieved by the aspiration of 300 c.c. of blood-stained fluid; no further aspiration was necessary. On the third post-operative day, air entry was greatly improved
all over the left chest but the patient had an irritating cough. On the fourth
day he was tipped over pillows, given vigorous breathing exercises and suc-
cceeded in coughing up a considerable amount of sticky sputum. After this
the cough rapidly improved and the patient's condition was sufficiently satis-
factory to allow him to get up on the tenth day. An X-ray taken on the fourth
day showed no pneumothorax and almost complete re-expansion of the lung
with a small effusion in the left costophrenic angle.

He was examined prior to demobilization, Category B, three months later.
The lung appeared to have fully expanded and the diaphragmatic movement
appeared normal. These findings were confirmed on X-ray examination. There
was slight respiratory retraction of the chest wall over the region of the resected
rib.

**DISCUSSION.**

Woolsey divides diaphragmatic hernia into three groups:—

1. *Congenital.*—There is incomplete fusion of the primitive components
   of the diaphragm leaving defects which occur on each side posteriorly at the
   foramina of Bockdalek, or anteriorly in the parasternal region at the foramina
   of Morgagni. The defect involves the peritoneum as well, therefore there is
   no hernial sac. Rarely the whole left diaphragm is absent.

2. *Acquired* via a lax œsophageal hiatus. The para-œsophageal hernia is
   covered with a true sac of peritoneum.

3. *Traumatic.*—This is nearly always on the left side because the right side
   is protected by the liver. The cause is either a missile wound or a bursting
   effect from a sudden increase of intra-abdominal pressure. There is no hernial
   sac.

The symptoms of traumatic diaphragmatic hernia are initially those of
severe shock due to the trauma causing the herniation and associated with
dyspnœa and cyanosis and with a high respiratory rate due to embarrassment
of the mediastinum and the lung by the herniation of abdominal viscera into
the pleural cavity.

Hæmatemesis is from pinching of the imprisoned stomach vessels. The
diagnosis is made by hearing bowel sounds in the chest, by a straight X-ray
showing a high diaphragm and a gas bubbling of the stomach or colon above
the diaphragm (see fig. 3) and is established with a barium follow through.

Traumatic hernias may not betray themselves for many years after
the initial injury and give rise to varied symptom complexes, simulating for example
coronary disease, peptic ulcer, gall-bladder dyspepsia or subacute intestinal ob-
struction. High acute intestinal obstruction may occur and the condition may
only be diagnosed at post-mortem. Polson describes a man wounded in 1917
who developed acute and fatal high intestinal obstruction suddenly twelve
and a half years later. The post-mortem showed an acute strangulation in a trau-
matic diaphragmatic hernia. Abdominal viscera are liable to disease whether
they occupy the abdomen or the pleural cavity.

Thus peptic ulcer with all its complications can occur in a stomach herniated
into a pleural cavity.
Frank and Hamilton describe a penetrating ulcer of the herniated stomach and its surgical treatment via the thoracic approach.

The danger of traumatic diaphragmatic hernia is obstruction or strangulation and the treatment is therefore surgical. This is imperative if the bowel is herniated for, if obstruction does occur, the operative mortality is high.

In the literature the surgical approach to diaphragmatic hernia is disputed. Thus Woolsey thinks the parasternal and most of the hiatus herniae are best approached from the abdomen. When many thick adhesions are present the thoracic approach appears to offer the best means of direct attack. At times a combined attack may be necessary.

Boyce states “the thoracic approach is best for the repair of the diaphragm but it is not so good for breaking up adhesions in old cases.” He advises the abdominal route when the bowel is strangulated or perforated. He describes a combined route which eliminates the disadvantages of both without adding to the trauma. He makes an incision in the eighth intercostal space commencing at the mid-axillary line across the costal margin on to the abdomen which is opened for 3½ inches. After the chest is opened he cuts across the costal cartilages and thus opens both cavities with a single incision.

Harrington prefers to work from the abdomen in most cases but Hedblom found fewer recurrences and a lower mortality rate in the thoracic approach.

Vere Nichol and Swinney in their cases of traumatic diaphragmatic hernia used the thoracic approach by an intercostal incision, and divided the ribs above and below as far posteriorly as possible.

Although the approach might be disputed in the literature which is predominately American, Tudor Edwards was emphatic that the transpleural route was best except for herniae via a lax oesophageal hiatus for which he advised the abdominal approach. He stated a combined laparo-thoracotomy...
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approach is never necessary. For a major thoracotomy he used the long intercostal incisions with division of the neighbouring rib necks.

SUMMARY.

(1) A patient suffering from traumatic diaphragmatic hernia is described.
(2) The thoracic approach by resecting a rib was used. Only when most of the rib up to the costochondral junction was removed was exposure excellent.
(3) Skilled anaesthesia is essential.
(4) Briefly diaphragmatic hernia in general is discussed.

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This case is published with kind permission of Colonel A. R. Oram, O.B.E., M.C., Officer Commanding this Hospital.

REFERENCES.