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Incidence and aetiology of a raised hemidiaphragm after cardiopulmonary bypass

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ABSTRACT A raised hemidiaphragm has been reported as an uncommon complication of cardiopulmonary bypass, possibly resulting from cold injury to the phrenic nerve. At Papworth Hospital myocardial protection during cardiac arrest relies in part on irrigation of the pericardial cavity with large volumes of Hartmann's solution at 4°C. Retrospective review of the chest radiographs of 100 consecutive patients undergoing cardiopulmonary bypass showed that 31 had a raised left hemidiaphragm soon after operation. The only significant correlation was with aortic cross clamp time ($p < 0.03$). A prospective study of 36 consecutive patients undergoing cardiopulmonary bypass was then undertaken with diaphragmatic screening and chest radiography. Preoperative screening gave normal results in all patients. In the early postoperative period 16 (44%) had left diaphragmatic weakness or paralysis, two (5.5%) right sided weakness, and two (5.5%) bilateral weakness. Repeat screening of these patients showed resolution in all but four cases (80%) at six months and in all but two (90%) at one year. The greater number of left sided lesions than of right (8:1) is probably due to the fact that the cold jet of irrigating fluid is directed towards the left phrenic nerve. These findings have implications with regard to the optimum temperature of the irrigant fluid for myocardial protection during cardiopulmonary bypass.

A raised hemidiaphragm complicating cardiopulmonary bypass has been previously reported.[1–5] The incidence is variable and the aetiology remains uncertain but has been attributed to cold injury of the left phrenic nerve.[1,2] Gastric distension, left lower lobe atelectasis, pulmonary trauma,[2] stretch trauma,[6] and association with dissection of the left internal mammary artery[5] have also been proposed.

We recently noted that a raised left hemidiaphragm on postoperative chest radiographs of patients undergoing open heart operations was relatively common in our unit. These investigations were undertaken to define the incidence, duration, and possible aetiology of this complication.

Patients and methods

A retrospective review of the previous 100 consecutive patients undergoing operations with cardiopulmonary bypass during 1982 was undertaken. Their mean age was 54 years (range 11–77) and 27 were women. The immediate preoperative, early postoperative (8–10 days), and late postoperative (6 months) posteroanterior chest radiographs were studied. The highest point of the left hemidiaphragm was compared with that of the right. If the point on the left was at the same horizontal level or higher than that on the right it was described as raised. An estimate of the incidence of raised left hemidiaphragm soon after operation and its subsequent resolution by six months was made.

To investigate the aetiology of this complication the early postoperative incidence was compared in different groups of patients with regard to operating surgeon, age and sex, operation performed, and aortic cross clamp time.

Having shown a high incidence of raised left hemidiaphragm we undertook a more comprehensive prospective study. A group of 36 consecutive patients undergoing operations including cardiopulmonary bypass was studied. Their mean age was 51 years (range 27–66) and seven were women. Diaphragmatic screening of both hemidiaphragms was undertaken before operation and eight to 10 days and six months to one year after operation. All

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screening was carried out by one radiologist (LJH) as a single blind procedure. Hemidiaphragmatic excursion during normal breathing and sniffing was recorded.[7] If postoperative hemidiaphragmatic movement was absent or less than at the preoperative screening, this was described as a weak hemidiaphragm. If the excursion was paradoxical the hemidiaphragm was described as paralysed.

Results

The chest radiographs of the 100 patients studied retrospectively showed that four had a raised left hemidiaphragm before operation. Twenty seven more patients developed a raised hemidiaphragm soon after operation (overall 31%). With Student's *t* test no significant association could be found between a raised left hemidiaphragm and age or sex, any of the four surgeons who operated, or the nature of the operation. There was, however, a significant relationship ($p < 0.03$) with aortic cross clamp time, which reflects the duration of myocardial cooling by irrigation of the pericardial wall with cold fluid. Patients without this complication had a mean aortic cross clamp time of 60 (SE 3) minutes, whereas those with a raised left hemidiaphragm had a mean aortic cross clamp time of 74 (6) minutes. Six months after operation in 14 of the 31 patients (45%) with raised left hemidiaphragms there had been a return to a normal position. The four patients with a raised left hemidiaphragm before operation showed no change in early or late postoperative films.

Three of the 36 patients studied prospectively (table 1) showed a raised left hemidiaphragm on preoperative chest radiography. Diaphragmatic screening, however, gave normal results in all patients at this stage. Soon after operation 15 patients (43%) had a raised left hemidiaphragm on their chest radiographs and 12 of these had abnormal results on screening. The three patients with preoperative elevation of the diaphragm and normal screening had normal screening results at this stage. Eight patients (22%) had a normal chest radiograph but abnormal screening. Of these, two had right sided hemidiaphragmatic weakness, two had bilateral weakness and four had left sided weakness.

With Student's *t* test no significant association could be found between the incidence of hemidiaphragmatic weakness or paralysis in this group and aortic cross clamp time. This was probably due to the relatively small number of patients studied. Repeat screening of the affected group of patients showed resolution in 16 (80%) at six months (table 2), and in 18 (90%) at 12 months.

Discussion

The results of these investigations confirmed the suspicion that there was a relatively high incidence of partial or complete transient paralysis of the left hemidiaphragm (49.5%) in patients undergoing

Table 1 *Results of diaphragm screening and chest radiography in 36 patients studied prospectively*

Preoperative		*Postoperative*					
Screening	*Chest radiograph*	*Chest radiograph*	*Screening*				
			Normal	*L side weak*	*R side weak*	*R and L sides weak*	*L side paralysed*
Normal 36	Normal 33 (21)	Normal 21	13	4	2	2	0
	Raised left hemidiaphragm 3* (12)	Raised left hemidiaphragm 15	3*	9	0	0	3
Total	36	36	16	13	2	2	3

*The same three patients.

Table 2 *Results of screening at 6 months in 20 patients with early postoperative screening abnormalities*

Early screening abnormality	*No of patients*	*Screening results at 6 months*
Left hemidiaphragmatic weakness	13	9 normal 3 no change 1 paralysis
Right hemidiaphragmatic weakness	2	2 normal
Bilateral hemidiaphragmatic weakness	2	1 normal 1 R weakness
Left paralysis	3	1 normal 2 L weakness
Total	20	

cardiopulmonary bypass in this hospital. The right hemidiaphragm was shown to be affected much less commonly than the left. Left hemidiaphragm paralysis may be associated with a number of conditions, as recently reported by Morrison and colleagues.[8] Phrenic nerve damage during cardiac procedures has been attributed to direct trauma by Mitchell *et al*.[4] Most of their cases followed thoracotomies in infants, but in four of the 32 there was cardiopulmonary bypass via a median sternotomy. Direct surgical trauma may occur at two points: firstly, where the left phrenic nerve is closely related to the aortic arch and, secondly, where both nerves pass laterally on to the diaphragmatic surface at the left and right pericardiophrenic angles. The extent of pericardial dissection about the aortic arch and at the diaphragmatico pericardial reflection differs between the operating surgeons at our hospital, but there was no significant difference between them in the frequency of raised hemidiaphragms. A possible further cause of injury to the phrenic nerve is internal jugular venous cannulation, as the nerve is closely applied to the scalenus anterior muscle and relatively near the internal jugular vein. Most internal jugular lines are placed on the right hand side, but right sided diaphragmatic paralysis or weakness was not common (11%) in our study. Iverson *et al*[6] suggested trauma to the phrenic nerve by stretching. This is now an accepted explanation of a first thoracic root lesion after median sternotomy.[5] It is unlikely as a cause here for two reasons. Firstly, there were no associated peripheral neurological lesions affecting the phrenic root origin of cervical segments 3, 4, and 5 and, secondly, hitching of the pericardium to the median sternotomy wound edges is practised by only two of the four operating surgeons here. An association between mobilisation of the left internal mammary artery and left hemidiaphragmatic paralysis was reported by Lederman *et al*.[5] None of the patients in our study underwent this procedure. In our study a raised left hemidiaphragm was significantly associated only with aortic cross clamp time. During this period the myocardium is protected from ischaemic damage by cardioplegia in association with profound topical myocardial hypothermia. Irrigation with Hartmann's solution at 4°C and at 300–800 ml/min is used, achieving myocardial septal temperatures of 8–12°C.[9] The flow of cold fluid into the pericardial cavity is directed over the left ventricular mass and so usually falls on the left side of the pericardium first. This irrigation probably causes left phrenic nerve damage by cold injury and is responsible for the fact that left sided hemidiaphragmatic paralysis is more common.

The effect of hemidiaphragmatic paralysis on the patient depends on preoperative lung function and age. Mitchell *et al*[4] found that infants with a paralysed hemidiaphragm after operation spent a significantly longer time being ventilated and required a longer period in the intensive care unit and in hospital. Older children tolerated a paralysed hemidiaphragm better. Arborelius *et al*[10] have shown that hemidiaphragmatic paralysis results in a 25% reduction in minute volume, FEV_1, and vital capacity. This is of little clinical significance in patients with good respiratory reserve, as evidenced by patients of Benjamin *et al*,[2] who had a mean hospital stay little different from those without hemidiaphragmatic weakness. Chandler *et al*[11] presented five patients with bilateral hemidiaphragmatic palsy. All had had ice chip topical myocardial hypothermia during operations with cardiopulmonary bypass. One patient died and a second required 140 days of assisted ventilation.

These observations support Scannel's original proposal[1] that a raised left hemidiaphragm as a complication of cardiopulmonary bypass is due to "a frost bitten phrenic nerve," and also show that the right phrenic nerve may similarly be affected, although less commonly. Topical myocardial hypothermia is a common method of myocardial protection during aortic cross clamping, but should be used with discretion in infants and those patients with compromised respiratory function.

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Book notices

Recommended health-based occupational exposure limits for respiratory irritants. WHO Study Group. (Pp 154; Sw fr 14). World Health Organisation, Geneva. 1984.

This is the fifth in a series of reports from World Health Organisation study groups concerned with occupational hazards and recommended exposure limits. It considers the four most commonly encountered respiratory irritants—chlorine, formaldehyde, oxides of nitrogen, and sulphur dioxide. Earlier reports considered the most commonly encountered heavy metals (1979), solvents (1980), pesticides (1981), and vegetable dusts (1982). This latest report is comparatively brief (154 pages) but commendably concise, and for 14 Swiss francs it is extraordinarily good value for money. For each chemical, properties, uses, and health hazards (in both experimental animals and working human populations) are comprehensively reviewed and referenced, and relationships to exposure levels carefully considered. The study group concludes with its own recommended exposure limits—short term (15 minutes) and eight hour time weighted average—and an assessment of immediate research needs. It is a worthy publication and a must for any medical library.—DJH

Pulmonary embolism and deep venous thrombosis. Samuel J Goldhaber (ed). (Pp 295; £55.) WB Saunders. 1985

This is a worthwhile book. Written by a number of authors, it comprises 16 chapters divided into four sections: "Fundamentals," including radiographic/nuclear medicine diagnosis, the pathology of deep vein thrombosis and pulmonary embolism and haemostatic principles; "Clinical Considerations," including management and prevention; "Special Clinical Topics," covering areas such as massive, non-thrombotic pulmonary embolism, and thromboembolisation in children; and "Future Prospects," including chapters on indium 111 platelet imaging and pulmonary angioscopy. Each chapter is clearly written and concise. To cover a subject of this size and keep within 300 pages requires extensive, extremely up to date referencing and therefore rather less discussion, but the end result is a most effective source book for all physicians and surgeons. Over 1500 references are given, and although the index is slightly lacking at times this is a minor criticism. Every hospital library should be persuaded to hold a copy, as at this price it is rather expensive for individual purchase.—JGA

Colour atlas of mesothelioma. JSP Jones, C Lund, and HT Planteydt. (Pp 201; £49.) MTP Press. 1985.

The main emphasis of this atlas is on primary malignant tumours of the pleura, peritoneum, and pericardium, but benign mesothelioma and mesotheliomas of the tunica vaginalis and atrioventricular node are dealt with briefly. The first 47 pages are comprised of sections devoted to the gross and microscopic pathology and cytology of malignant mesothelioma. Aetiology, epidemiology, and clinical aspects are also covered. Each section is followed by an up to date list of references. The work of mesothelioma panels is outlined, and a list of such panels in various countries is supplied. The rest of the book consists of colour photographs illustrating the gross and microscopic appearances of malignant mesothelioma and other tumours of mesothelial tissue. The pictures are in general of high quality, but there are too many of them. Altogether there are 246 plates, among which are 39 gross photographs and over 100 photomicrographs of typical malignant mesotheliomas. The diagnostic usefulness of so many illustrations of well known tumours is limited. The relatively high cost of this book could be justified only if more attention had been paid to the problems of differential diagnosis, particularly in cytological and biopsy material.—CWE