The anaesthetic management of children with anterior mediastinal masses

H. A. Hack, N. B. Wright and R. F. Wynn

1 Consultant Anaesthetist, 2 Consultant Radiologist, 3 Consultant Haematologist, Royal Manchester Childrens Hospital, Hospital Rd. Pendlebury, Manchester M27 4HA, UK

Summary
Children with anterior mediastinal masses may experience serious complications during general anaesthesia. We retrospectively surveyed the records of children with an anterior mediastinal mass who had been admitted to our hospital over a 7 year period. The presence of pre-operative symptoms or signs, findings of any special investigations performed and the anaesthetic outcome were noted. All radiological investigations were studied and tracheal compression measured. The majority of patients presented with severe clinical signs. There was a poor relationship between clinical signs and size of tumour or tracheal compression on CT scan. Corticosteroids were used prior to diagnosis in 33% of patients, all of whom were considered high risk. A clear diagnosis was made in 95% of these patients. The overall complication rate was 20% and 5% of patients had a serious complication related to anaesthesia. Stridor was the only sign that predicted an anaesthetic complication. Peri-operative respiratory complications were confined to patients with an isolated tracheal cross-sectional area less than 30% normal or less than 70% and associated with bronchial compression.

Correspondence to: Dr H. A. Hack
E-mail: henrik.hack@cmmc.nhs.uk
Accepted: 8 January 2008

Children with anterior mediastinal masses may experience severe problems during general anaesthesia, usually as a consequence of extrinsic compression of the airway, obstruction to the venous return or obstruction to the output of the heart [1]. Guidelines have been published in an attempt to improve the safe management of these patients [1–6]. Such guidelines may be criticised firstly because they depend upon being able to identify those patients at risk and secondly because some of the management options suggested may not be available or are considered inappropriate before a histological diagnosis has been made.

The anterior mediastinal masses that are most likely to cause anaesthetic problems in children are rapidly growing haematological malignancies [7]. Previously published studies suggest that the presence of certain clinical symptoms or results of pre-operative investigations may predict potential anaesthetic difficulty [6, 8–10].

We examined the perioperative management of all such children presenting to our hospital over a 7 year period. Our aims were firstly to estimate the incidence of clinical symptoms and the results of any investigations each patient underwent before an accurate diagnosis was made. The use of diagnostic procedures under local anaesthesia (LA) or general anaesthesia (GA) and any complications that occurred were noted. We then attempted to develop a method of identifying cases with a high risk of complications. Ultimately, we were hoping to compare our practice to published recommendations and to create a set of local guidelines relevant to our patient population and available services.

Methods
After approval from the Local Research Ethics Committee and the Hospital Audit Committee, the case notes, Xrays and CT scans of all children that had presented to our hospital with T Cell lymphoblastic leukaemia (T-ALL), Hodgkins disease (HD) and T Cell lymphoblastic non Hodgkins Lymphoma (NHL) between 1999 and 2006 were inspected. The records of all children with widening of the mediastinum on plain chest Xray (CXR) at presentation were then subjected to detailed examination.
**Pre-operative clinical data**

The signs and symptoms at presentation relevant to possible airway or cardiovascular compromise together with demographic data including age, sex and weight were collected. Particular attention was paid to the presence of cough, dyspnoea (D), orthopnoea (O), stridor (S), wheeze (W) or clinical evidence of superior vena cava obstruction (SVC obs) and loss of consciousness or syncope.

The results of CXR, CT scan, MRI scan and echocardiogram and any other special investigations performed before any general anaesthesia were noted.

A decision to treat patients with either corticosteroids or radiotherapy before a diagnosis had been made and any potential effect it had on clinical course or interpretation of histological specimens was noted.

**Anaesthetic data**

The choice of anaesthetic technique together with any induction, intra-operative or postoperative complications was noted. Complications were classified as Mild (abnormality noted but no significant change in practice required e.g. mild elevation in measured end tidal carbon dioxide concentration, ET CO₂), Moderate (abnormality noted requiring change in practice or additional therapy e.g. moderate airway obstruction responding to change in patient position) or Severe (abnormality requiring rapid intervention to avert potentially dangerous deterioration e.g. airway obstruction requiring intubation or passage of a rigid bronchoscope). Problems were then classified according to their probable site (respiratory system (R) and/or cardiovascular system (C)) and timing (pre/intra/post operative period).

**Radiological data**

Where available, all relevant CXR’s, CT or MRI scans of the thorax were inspected. Hard copies were first converted to a high resolution JPEG file using a digital camera. Capture was achieved using a medium telephoto lens (70–80 mm) with the trachea centred in the photograph to prevent distortion. All files were then processed through Adobe Photoshop to remove personal data whilst retaining scales and window information (see below). Visual inspection and measurements were then performed via computer screen, using Microsoft Windows Picture Viewer, by a Consultant Paediatric Radiologist who was unaware of the patient’s diagnosis and clinical course.

Mediastinal mass ratio (MMR) was measured using the formula MMR = maximum width of the mediastinal mass/maximum thoracic width (cm) [10]. Measurements were made either from plain CXR or the ‘scout’ view from CT scan at time of presentation. King et al. subdivided MMR into three groups: MMR ≤ 0.30, 0.31–0.43 and ≥ 0.44 on their finding that those patients with a high MMR were more likely to have respiratory symptoms at presentation [10]. These limits were applied to our patient group.

Measurement of tracheal diameters was performed in a manner similar to previous studies [8, 10]. The widest (d₁) and narrowest (d₂) diameters were measured at two points: the largest appearing area, usually found at the thoracic inlet with lung apices appearing in the picture, was chosen as the patient’s ‘normal’ trachea and used as its control. A second, smallest area was then selected from available cuts through the trachea above the carina and its diameters measured. Measurements were made from identical cuts from scans in both mediastinal and pulmonary windows (window level: +40 & −600 Housefield Units respectively for thoracic scans at RMCH). The measurements were then interpolated to a window value of −450 HU; this has previously been shown to be the most accurate for measuring tracheal tissue [4]. Cross sectional area (CSA) was then calculated using the formula CSA = π (d₁/2 × d₂/2) and expressed in mm². Thus each patient had two values of CSA: control (CSAc) and the narrowest point (CSAn). The compression caused by any mediastinal mass was then expressed as a percentage of normal using the equation %CSA = CSAn/CSAc × 100. The presence of any carinal or bronchial compression was noted and the antero-posterior diameter of both bronchi measured.

Following assessment by one of the authors (NW) bronchial compression was considered significant if one or both bronchi was occluded or compressed by > 50% at some point along its path or compared to the opposite bronchus. In addition, the presence of any pulmonary (collapse, consolidation or pleural effusion), cardiac (pericardial thickening or effusion) or vascular (superior vena cava or pulmonary artery compression or obstruction) complications was noted. Where there was only an MRI scan available, tracheal measurements were made in a similar way and the degree of compression calculated and expressed in an identical manner. In 25 patients, a computer record of the CT scan was also available to view in addition to the hard copy. In such cases, the tracheal dimensions were measured using the computer software (Advantage Windows 3.1, GE medical systems, Slough, UK). Measurements were made by the same radiologist from identical cuts. These measurements were performed on a separate occasion to those on the hard copies and the radiologist remained blinded to previous data at all times.
Echocardiogram data
The relevant findings from all pre-operative and prediagnostic reports were noted. Particular reference was made to the possible presence of pericardial effusion, right ventricular outflow tract obstruction or ventricular dysfunction.

Statistical analysis
Simple descriptive statistics, unpaired t-test, Fisher’s exact test and Mann–Whitney U-test were applied where appropriate, utilising STATSDIRECT V2.6.2 software. Statistical significance was set at p < 0.05.

Results
A total of 107 patients were diagnosed with either T-ALL, HD or NHL within the study period. Three sets of notes were unobtainable. In the remaining 104 patients, 56 children had evidence of a widened mediastinum on plain CXR and so entered the audit.

Demographic data
Of the 56 patients, 16 had T-ALL: (mean age 9 years; range 4–15 years), 21 had HD: (12; 4–15 years) and 19 had NHL: (9; 2–15 years).

Clinical data
Fourteen patients (25%) had no symptoms attributable to airway or cardiovascular compromise at time of presentation. The incidence of significant respiratory symptoms and signs amongst the remaining 42 patients (75%) is listed in Table 1. Only two patients suffered a loss of consciousness or syncope. The first patient suffered an out of hospital cardiorespiratory arrest (see below). The second patient had suffered a single episode of syncope at home the day before admission. Both of these patients had NHL.

Radiographic data
In 50 cases it was possible to calculate the MMR (No CXR or CT scan was available in the remaining six cases).

Table 1 Patient numbers with clinical signs at presentation by diagnosis.

<table>
<thead>
<tr>
<th>Clinical sign(s)</th>
<th>ALL</th>
<th>NHL</th>
<th>HD</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>5</td>
<td>3</td>
<td>6</td>
<td>14 (25)</td>
</tr>
<tr>
<td>Cough</td>
<td>10</td>
<td>11</td>
<td>12</td>
<td>33 (59)</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>6</td>
<td>12</td>
<td>6</td>
<td>24 (43)</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>5</td>
<td>9</td>
<td>3</td>
<td>17 (30)</td>
</tr>
<tr>
<td>Stridor</td>
<td>3</td>
<td>6</td>
<td>2</td>
<td>11 (19)</td>
</tr>
<tr>
<td>Wheeze</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>6 (11)</td>
</tr>
<tr>
<td>SVC obs</td>
<td>4</td>
<td>4</td>
<td>0</td>
<td>8 (14)</td>
</tr>
</tbody>
</table>

Figure 1 Mediastinal mass ratio (MMR) according to presence of clinical symptoms and signs. Dashed lines indicate MMR ≤ 30% and ≥ 44%.

Data regarding MMR relating to clinical signs were then plotted (Fig. 1).

In 43 cases, CT and/or MRI scans were available for inspection. In four of the remaining 13 patients, no record of the scans was available for assessment. A further seven patients were diagnosed with T-ALL from the peripheral blood film and/or BMA. A CT scan was not performed before commencing treatment. The remaining two children were considered too ill to undergo a CT scan either awake or under sedation. One child had experienced an out of hospital cardiopulmonary arrest due to airway obstruction and so was intubated at the time of their scan. No tracheal measurements were made from this scan. In 25 cases, computer records and hard copies were available for comparison. Two diameters at each level were measured i.e. at the ‘normal’ tracheal level and the ‘smallest, most compressed’ level, giving four paired CT measurements for each patient. The data produced a correlation coefficient of 0.96. Paired data (from computer software and by radiologist) were then plotted according to Bland–Altman [11] producing a mean (SD) of 0.484 (1.12)mm. The calculated %CSA was then plotted according to the presence of clinical symptoms and signs at presentation (Fig. 2).

King’s classification [10] was applied to our group, comparing %CSA of normal with MMR size: MMR < 0.30 (n = 3, mean (SD) = 100% (0)), MMR 0.31–0.43 (13, 74.4% (24.1)), MMR > 0.44 (19, 66.3% (22.2)).

Echocardiography
A total of 13 children had preanaesthetic transthoracic echocardiograms (TTE). A pericardial effusion was noted in six of these (only one case showing evidence of right ventricular compromise). In five cases, there was evidence of tumour encasing or compressing the heart or great
vessels (pulmonary artery (PA) or superior vena cava (SVC)). A further nine had TTE’s between surgery and within 24 h of commencing chemotherapy. This group contained one case of pulmonary artery compression. Overall, there was only one case with impaired left ventricular function (defined as an ejection fraction (EF) < 60%; fractional shortening (FS) < 0.30) with an EF = 28%, FS 0.13.

A total of 29 CT scans showed evidence of some cardiovascular pathology (pericardial thickening, effusion and/or PA or SVC compression): Thirteen cases showed pericardial thickening, associated in eight cases with an effusion. In seven cases, there was evidence of main or branch PA compression and in seven cases there was evidence of SVC compression.

Diagnostic procedures under local anaesthesia (LA)
Eight patients had an initial diagnosis made from their presenting peripheral blood film (all T-ALL). A further three patients underwent bone marrow aspiration (BMA) awake under local anaesthesia. A diagnosis was made in two patients (both T-ALL). The third patient had a diagnosis made (HD) from a lymph node biopsy under general anaesthesia following a non-diagnostic BMA under LA. Although some patients had been offered but refused a BMA under LA, no accurate data could be collected on this.

No patients underwent diagnostic aspiration of pleural fluid under LA. In six cases, pleural fluid obtained under GA was sent for inspection: NHL was diagnosed in two cases; two further cases showed ‘malignant blast cells’; one case showed ‘malignant looking cells’ and one specimen was reported as ‘inadequate’.

Pre-operative use of steroids or radiotherapy
A total of 18 patients (33%) received pre-operative corticosteroids before a clear diagnosis had been made. Choice and dose of drug used was at the discretion of the Consultant Haematologist. All received standard measures to prevent tumour lysis syndrome. In 13 of these 18 cases, the patients had clinically severe signs of airway compromise. Of the remaining five patients, two had evidence of tumour encasing the heart and great vessels or trachea and three had severe, non-cardiorespiratory symptoms.

Comparison of the presence of symptoms in steroid treated and non-treated groups supported this: Cough p = 0.37, dyspnoea p = 0.002, orthopnoea p = 0.001, stridor p = 0.002, wheeze p = 0.009, SVC Obs p = 0.009 (Fisher’s exact test).

In 14 cases, a CT scan was available for study. Data from one of these was excluded from further analysis since the scan was performed 3 days after commencing steroids, by which time the child was almost symptom free. When compared to the others, steroid treated patients were also more likely to have a smaller tracheal \( %\text{CSA} \) at presentation. Steroid treated patients: mean = 54%, SD = 21.8; Non-steroid patients: mean = 80% SD = 21.9 (p = 0.0016, Mann–Whitney \( U \)-test).

A clear diagnosis (T-ALL = 9, NHL = 5, HD = 3) was made in 17 of the 18 patients (95%). These patients had received varying dosages of corticosteroids (usually prednisolone 60 mg.m\(^{-2}\).day\(^{-1}\)) for up to 5 days.

The single remaining child had received 6 days of steroids with good clinical improvement before undergoing a thoracotomy. All biopsies were either normal or non-diagnostic and they were treated for NHL based on the clinical picture.

Anaesthetic data
A total of 55 patients underwent 56 general anaesthetics: one patient had been transferred intubated from another hospital with the diagnosis already made (see above) and another patient underwent two anaesthetics before a diagnosis was made. In 53 cases, the anaesthetic notes were available for inspection. All but six of the patients were anaesthetised by a consultant. A gaseous induction utilising a volatile agent (usually sevoflurane) was chosen in 25 cases. Induction was commenced in the sitting position in two of these cases and in the lateral position in another. An IV induction in the supine position was used in the remaining 28 cases. Anaesthesia was maintained with spontaneous ventilation (SV), utilising either a face mask, laryngeal mask airway (LMA) or endotracheal tube (ETT), in 18 cases. Intermittent positive pressure ventilation (IPPV) was used in the other 35 cases. In 11 of these, the ability to adequately ventilate before the administration of a relaxant was documented.

In 41 cases a pre-operative CT scan was available for analysis for measurement of \( %\text{CSA} \). There was no significant difference in \( %\text{CSA} \) between the groups of patients.
who received gaseous ($n = 19$, mean $\%\text{CSA} = 64$, SD (30)) and intravenous ($n = 22$, mean $\%\text{CSA} = 76$, SD (18.4)) induction techniques ($p = 0.13$). Similarly, there was no significant difference between patients in the spontaneous breathing ($n = 13$, mean $\%\text{CSA} = 63$, SD (29)) and IPPV ($n = 28$, mean $\%\text{CSA} = 75$, SD (22)) maintenance groups ($p = 0.16$).

Anaesthetic complications
Complications were noted in 11 patients (19.6%), one of whom was undergoing a second general anaesthetic (Table 2). There were no perioperative deaths. Only one of the complications could be clearly classified as cardiovascular in origin: a transient fall in blood pressure corrected by a single fluid bolus. The remaining 10 cases were classified as respiratory in origin: eight cases were graded Mild or Moderate, requiring either no or limited intervention. The remaining three cases (5.3%) were classified as Severe (Table 2).

The presence of presenting clinical signs were then compared between patients that did and did not experience anaesthetic complications: Cough $p = 0.530$, dyspnoea $p = 0.053$, orthopnoea $p = 0.147$, stridor $p = 0.006$, wheeze $p = 0.096$ and SVC obstruction $p = 0.34$ (Fisher’s exact test).

In those patients where a CT scan was available for analysis, respiratory complications occurred almost exclusively in six patients (7 GA’s) with $\%\text{CSA} \leq 70\%$. If the patients within this group with significant carinal or bronchial compression are excluded, anaesthetic respiratory complications were confined to three patients (four GA’s) with a $\%\text{CSA} \leq 30\%$.

Discussion
Mediastinal masses (MM) consist of a heterogenous group of benign and malignant tumour types. Tumours in the anterior and superior mediastinal compartments are most likely to cause anaesthetic complications due to their anatomical proximity to the heart and airway. In children, the most common tumour types found in this location are haematological malignancies (HD, NHL and ALL) and teratomas [7]. The former group are the most significant since they are the commonest and most rapidly enlarging. It was for this reason that we chose to study a patient group that consisted entirely of children with anterior mediastinal masses due to haematological malignancies (HD, NHL & T-ALL).

It has been recognised for many years that general anaesthesia can be extremely hazardous in children with anterior mediastinal masses. The numerous anatomical and physiological reasons behind this have been clearly and expertly discussed in previous reviews [1].

Guidelines have been published regarding the management of children with anterior mediastinal masses [1–5]. Significant differences between them have reflected the recognition of important pre-operative risk factors, changes in treatment [12] and the availability of local services such as cardiopulmonary bypass (CPB) and radiotherapy. An accurate histological diagnosis is important so that appropriate and effective treatment can be given. Recommendations have often been based on obtaining a diagnosis using, if possible, alternatives to GA in ‘high risk cases’. Where general anaesthesia is considered unavoidable specific techniques have been recommended [1–5]. However, even relatively asymptomatic patients undergoing general anaesthesia have suffered severe complications [1, 4]. Thus, before we can compare our management with current published guidelines, one of the biggest challenges is being able to reliably identify such potentially high risk cases in advance.

Clinical signs
In our study, the distribution of diagnoses, age range and spectrum of clinical symptoms and signs was similar to previously published results [6, 8, 10] (Table 1). However, our group does appear to contain a greater proportion of children with severe respiratory signs. This may reflect the high risk group of patients studied, although the possibility that ethnic mix or a difference in primary care management, perhaps leading to a delay in presentation, cannot be discounted.

We found no single sign to be reliably associated with either a large mass (MMR) or significant tracheal compression on CT scan. With the exception of stridor, there was no statistically significant association with anaesthetic complications. This maybe due to the use of pre-operative steroids in patients considered at high risk (see below).

Radiological signs
Previous studies have looked at the predictive value of various pre-operative investigations in an attempt to produce a more reliable indicator of compromise or potential anaesthetic complications.

King et al. [10] subdivided MMR into three groups: $\text{MMR} \leq 0.30$, $0.31–0.44$ and $\geq 0.44$ based upon their finding that those patients with a high MMR were more likely to have severe respiratory symptoms at presentation [10]. We found a small trend (Fig. 1) towards a relationship between a larger MMR and the presence of significant pre-operative symptoms and signs. However, a large MMR did not appear to be associated with any particular sign and the relationship is probably not clear enough to be of much use clinically.
Table 2 Details of patients suffering anaesthetic complications.

<table>
<thead>
<tr>
<th>Age years/ Diagnosis/ S = steroids used preop</th>
<th>Procedure</th>
<th>Induction</th>
<th>Maintenance</th>
<th>MMR</th>
<th>%CSA (Carinal/ bronchial compression?)</th>
<th>Symptoms &amp; signs</th>
<th>Problem grade/ type</th>
<th>Timing</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>15 T-NHL S</td>
<td>LP, BMA, Hickman line insertion</td>
<td>Gas SV</td>
<td>ETT / IPPV / no relaxant</td>
<td>0.34</td>
<td>24 (No)</td>
<td>C, D, O, St, W, SVC</td>
<td>Moderate – R</td>
<td>1. Induction 2. Postop</td>
<td>Partial obstruction and desaturation despite guedel airway. Relieved following intubation and turning on side. Transient $O_2$ requirement following extubation.</td>
</tr>
<tr>
<td>15 T-ALL</td>
<td>BMA, LP</td>
<td>IV SV</td>
<td>LMA / SV</td>
<td>0.37</td>
<td>N / A</td>
<td>C</td>
<td>Moderate – R</td>
<td>Post op</td>
<td>Two episodes of stridor and desaturation needing oxygen, 10 h postop.</td>
</tr>
<tr>
<td>15 T-ALL</td>
<td>BMA, LP</td>
<td>IV SV</td>
<td>LMA SV</td>
<td>0.59</td>
<td>93 (Yes)</td>
<td>C</td>
<td>Moderate – R</td>
<td>Induction</td>
<td>Mild obstruction (No desaturation) supine responding to change to lateral position.</td>
</tr>
<tr>
<td>9 T-NHL</td>
<td>LP, BMA, Hickman line insertion, thoracotomy and MM biopsy</td>
<td>IV IPPV</td>
<td>ETT / IPPV/ relaxant</td>
<td>0.88</td>
<td>67 (Yes)</td>
<td>C, D</td>
<td>Severe – R</td>
<td>Post op</td>
<td>Collapse of R lung due to bronchial compression following drainage of effusion Electively ventilated post op. Steroids started post op with improvement.</td>
</tr>
<tr>
<td>3 T-NHL S</td>
<td>BMA, LP, PIC line, biopsy of neck mass</td>
<td>Gas SV</td>
<td>LMA / SV</td>
<td>0.38</td>
<td>N / A</td>
<td>C, D, O, St</td>
<td>Moderate – R</td>
<td>Post op</td>
<td>Mild stridor in recovery responding to single adrenaline nebulizer. Mild obstruction supine with LMA.</td>
</tr>
<tr>
<td>6 T-NHL</td>
<td>BMA, LP &amp; biopsy suprarenal mass</td>
<td>Gas SV</td>
<td>LMA / SV</td>
<td>0.50</td>
<td>20 (No)</td>
<td>C, St, SVC</td>
<td>Mild – R</td>
<td>Induction</td>
<td>Transiently oxygen dependent post extubation. One episode mild positional stridor 2 days post op.</td>
</tr>
<tr>
<td>3 T-NHL</td>
<td>BMA, LP, node biopsy</td>
<td>IV IPPV</td>
<td>ETT / IPPV/ relaxant</td>
<td>0.80</td>
<td>56 (Yes)</td>
<td>D, O</td>
<td>Mild – R</td>
<td>Post op</td>
<td>Fall in BP 70 / 40, responded to 20 ml.kg$^{-1}$ colloid.</td>
</tr>
<tr>
<td>14 T-NHL</td>
<td>BMA, LP &amp; biopsy neck mass</td>
<td>Gas SV (sitting)</td>
<td>ETT SV → IPPV + relaxant</td>
<td>0.46</td>
<td>32 (Yes)</td>
<td>D, O, St, SVC</td>
<td>Moderate – R</td>
<td>Post op</td>
<td>Airway obstructed when supine; not relieved by lateral position. $SpO_2$, HR &amp; BP fell despite atropine –→ abandoned.</td>
</tr>
<tr>
<td>10 T-NHL</td>
<td>LP, BMA, Hickman line insertion &amp; biopsy chest wall mass</td>
<td>Gas SV</td>
<td>ETT / IPPV + relaxant</td>
<td>0.51</td>
<td>100 (Yes)</td>
<td>D</td>
<td>Moderate – C</td>
<td>Induction</td>
<td>Partial obstruction on side. Carinal &amp; LMB compression beyond ETT. RMB intubated for OLV. Elective IPPV post op, transient collapse left lung.</td>
</tr>
<tr>
<td>10 HD S</td>
<td>BMA, LP, node biopsy</td>
<td>Gas SV</td>
<td>FM SV</td>
<td>0.59</td>
<td>26 (No)</td>
<td>C, D, O, St, W</td>
<td>Severe – R, C</td>
<td>Induction</td>
<td>Transient collapse left lung.</td>
</tr>
<tr>
<td>10 HD S</td>
<td>BMA, LP, node biopsy &amp; Hickman line insertion</td>
<td>Gas SV</td>
<td>ETT SV → IPPV + relaxant</td>
<td>0.82</td>
<td>24 (Yes)</td>
<td>C, D, O, St, W</td>
<td>Severe – R</td>
<td>1. Induction 2. Postop</td>
<td>Partial obstruction on side. Carinal &amp; LMB compression beyond ETT. RMB intubated for OLV. Elective IPPV post op, transient collapse left lung.</td>
</tr>
</tbody>
</table>

N / A, not available. Symptoms & signs: C-cough, D-dyspnoea, O-orthopnoea, St-stridor, W-wheeze, SVC-SVC obstruction. Problem type: R-respiratory, C-cardiovascular.
This is not surprising since MMR is no more than an approximate indicator of tumour size relative to the patient’s size. The presence and severity of any clinical signs will depend not only on actual tumour size but its rate of growth, proximity to the airway and cardiac structures, and the possible coexistence of other pathology such as pericardial and pleural effusions.

Tracheal shape, diameter and thus cross sectional area in children have been measured accurately using CT scanning [13–15]. Azizkhan et al. were the first to formally measure tracheal compression on CT scan in children with mediastinal masses [6], using a technique modified from Grisom [13]. Results were then compared to those of a previously published normal population [13]. A large proportion of their patients were either asymptomatic (40%) or had mild symptoms (42%) at presentation. Significant compression was rare in these patients and they underwent general anaesthesia uneventfully. Most of the remaining patients with severe symptoms had CSA < 50% expected. These patients either underwent uneventful diagnostic biopsies under local anaesthesia or encountered significant problems during general anaesthesia. It was suggested that general anaesthesia should be avoided if possible in any patient with severe symptoms or with %CSA < 65%.

Using a similar measurement technique Shamburger et al. reported their experience with a similar group of patients [8]. Most of their patients were either asymptomatic (65%) or had relatively mild symptoms such as cough (24%). Significant symptoms were rare (11%). They found a wide variation in %CSA with little correlation between degree of tracheal compression and severity of symptoms with the possible exception of orthopnoea. They reported no anaesthetic complications, perhaps as a consequence of the use of pre-operative radiotherapy and/or the relatively frequent practice of performing tissue biopsy under local anaesthesia.

King et al.’s group [10] had a higher incidence of symptomatic patients than those previously mentioned. They reported no statistically significant association between degree of compression and clinical symptoms. A third of their patients underwent diagnostic biopsy under local anaesthesia (most with additional intravenous sedation). The remaining patients underwent general anaesthesia and there was one death and a relatively high complication rate. All of these patients had either large masses or severe symptoms.

Analysis of tracheal compression in our study used a method adapted from King et al. Digital capture and enlargement was utilised to improve accuracy of measurement. Comparison of our manual measurements with those performed by computer software showed a reassuring degree of correlation and was consistent with previous studies [8].

Inspection of the presence of clinical signs according to %CSA shows a better graphical trend towards the perhaps more significant signs and a greater reduction in tracheal CSA (Fig. 2). However, for any one sign there was a wide variation in measured %CSA, although such variation was less pronounced for orthopnoea, stridor and SVC obstruction. The previously reported finding by Shamburger of orthopneoa being found only in cases where %CSA < 40% was not replicated in our group. However, orthopnoea, stridor, wheeze and SVC obstruction were found predominantly in patients whose %CSA < 70%. This is similar to Azizkhan’s findings (although they studied a group with a more heterogenous mix of tumours). These differences may represent slightly differing methods of measurement or simply reflect different population groups. An important limitation of measuring reduction in %CSA is that it provides no information regarding possible compression at the level of the carina or bronchi. There has been no previously published data relating to this for various reasons. With the exception of King et al.’s study, previously published work has relied upon comparison of measured tracheal diameters with accepted normal values. Such values for normal bronchi have not been published. Accurate measurement of bronchial diameters and the comparison of right and left sides via CT scan are hampered by technical difficulties, since even in normal children, the bronchi extend from the carina at different angles. Such difficulties will only be magnified if there is associated compression or distortion by an adjacent mass. Despite these difficulties we made an attempt to assess carinal and bronchial compression, albeit via a somewhat arbitrary system. When related to significant clinical signs, it was found that almost all patients with orthopnoea, stridor, wheeze or SVC obstruction exhibited significant bronchial compression in addition to a CSA of < 70% of normal. Most of those symptomatic patients without bronchial compression showed CSA < 40% (Fig. 2).

**Choice of anaesthesia for biopsy**

The overall rate of diagnostic procedures performed without general anaesthesia in our series was low compared to other reports. Only three patients underwent BMA under local anaesthesia (LA) and there were no attempts at pleural fluid aspiration or lymph node biopsy under LA. There are several possible reasons for this low rate. All procedures require a significant degree of cooperation from the patient. In younger children, this may be helped by the use of sedation but at the risk of subsequent complications. Reticence on the part of the operator may also play a part in the low incidence. This
maybe due to the fact that if complications such as bleeding occur during a biopsy under LA it may be essential to convert to a general anaesthetic. NHL is often associated with a pleural effusion, which may provide a possible diagnostic source [16]. Only two out of six associated with a pleural effusion, which may provide a definitive histological diagnosis [18]. Ferrari et al. reported a series of children whom underwent general anaesthesia without the use of pre-operative radiotherapy or chemotherapy [18]. In the symptomatic patients, general anaesthesia was maintained with spontaneous ventilation. There were no perioperative deaths and the authors concluded that general anaesthesia can be safely used in such cases without the use of pre-operative radiotherapy, in anticipation of avoiding diagnostic confusion. However, the overall incidence of symptomatic patients in the group was low and there was a high incidence of perioperative anaesthetic complications (all classified as ‘severe’ according to our methods). Pre-operative radiotherapy was not used in any of our cases to treat symptomatic patients; where indicated corticosteroids were used in preference. There are no radiotherapy facilities on site at our hospital. Treatment would thus involve the out of hospital transfer of an extremely unstable patient. Radiotherapy may also require provision of general anaesthesia in some children, especially the young or unwell, who are unable to lie still. Even if shielded radiotherapy is given, there is no guarantee of preventing subsequent diagnostic confusion.

The use of steroids is often easier, quicker and safer than radiotherapy and is our preferred treatment option when indicated. There is considerable controversy over the prediagnostic use of steroids to improve a patient’s condition since it can potentially interfere with a histological diagnosis [4, 5, 7]. Although some authors have previously advocated their use, there is little published data on the subject [1]. Halpern et al.’s published case series illustrated the potential benefits and problems involved with making a diagnosis following empirical use of steroids. Steroids were used before a diagnosis had been made in 18 patients (33%) in our series. The commonest reason for this was serious signs of airway compromise at presentation. Those patients that received steroids had significantly worse clinical signs and tracheal compression on CT scan compared to the others. Despite the use of steroids, we were able to make a clear diagnosis in all but one patient (95% diagnosis rate). That patient had received steroids for 6 days prior to biopsy.

### Cardiovascular compromise

Only 13 (23%) children had a pre-operative TTE performed. The finding of such a low rate of pre-operative TTE’s is disappointing. Significant abnormalities were found in six (46%) of these suggesting a relatively high incidence of abnormalities in this patient group. This is supported by the discovery that significant cardiovascular abnormalities were seen on 29/43 (67%) CT or MRI scans. With the exception of SVC obstruction, the occurrence of cardiovascular symptoms and signs at presentation was low, suggesting that potentially serious cardiovascular abnormalities remain silent until specifically looked for. However, the incidence of both total and specifically cardiovascular complications during general anaesthesia was low in our study. This may be because we are overestimating the clinical importance of such abnormalities. The classification system that we used may also have affected results: the rapid onset of hypoxia during anaesthetic induction is easily attributed to inappropriate ventilation, especially if mechanical ventilation problems are encountered. What can not be known for certain is the significance of any reduced pulmonary perfusion (as a consequence of inadequate cardiac filling and/or right ventricular outflow) that may have contributed to the deterioration. Another reason for the low rate of cardiovascular complications could be that the early use of pre-operative steroids, used to shrink tumour size in patients with more obvious airway problems, also has a beneficial effect on any existing cardiovascular impairment. The observation of a serial improvement in echocardiography findings in some patients in our series would support this.

The immediate availability of cardiopulmonary bypass (CPB) before induction of anaesthesia has been recommended by some authors and even appears in some published management guidelines [3, 4]. Although CPB is not available at our hospital, it is our belief that the use of CPB in children with MM is an unrealistic option for a number of reasons. If CPB is to be used effectively, it probably requires forward planning rather than being used as a salvage manoeuvre. The necessary equipment and team members must be primed and ready to go as soon as they are needed. Since it is impossible to predict with certainty which patients will have severe complications, this would probably entail having CPB prepared before all cases. Even if such facilities were made available for only high risk cases, its use only when the child has started to
decompensate may be too late. Despite our reservations the ultimately successful use of CPB in children with MM has been recently reported [19, 20]. A total of three cases have been described. It must be noted that in all of the reported cases, treatment with steroids was delayed until some time after presentation. There have been no published reports of the unsuccessful use of CPB in such cases.

When considering the available evidence, it is clear that there is no single clinical sign or test that can accurately predict which patients with a mediastinal mass will suffer complications under general anaesthesia. A CT scan will provide evidence of airway compression at a tracheal and/or bronchial level and the existence of exacerbating factors such as pleural effusion which may limit a patient’s physiological reserve. In our series of patients, respiratory complications related to anaesthesia were confined to those with either an isolated tracheal CSA ≤ 30% of normal or a tracheal CSA ≤ 70% associated with carinal or bronchial compression. In cases where a CT scan is not obtainable, measurement of erect and supine peak expiratory flow rate (PEFR) may provide useful information regarding airway obstruction [9, 10]. As with any retrospective study, there is a danger that complications are under-reported. Although the system that we chose to assess and classify any anaesthetic complications was arbitrary, it was aimed at being as inclusive as possible. The overall complication rate of 19.6% might be considered high when compared to some previous reports. However, the majority of these were classified as mild or moderate, thus requiring no or very limited action. Such minor complications may have gone unreported in previous studies. It did reveal that significant complications can occur at any time in the perioperative period. Severe complications occurred in only two patients (a total of three general anaesthetics). This rate is much less than some previous reports, especially those where pre-operative treatment has been withheld. It is difficult to predict how much more severe any compression seen on scan in an awake or lightly sedated patient will become under general anaesthesia. For this reason, obtaining a peripheral tissue biopsy under local anaesthesia should be considered in all patients but particularly in those considered high risk. If general anaesthesia is considered necessary, it would seem prudent to try and maintain spontaneous ventilation if possible. In cases where IPPV is felt necessary or desirable, ‘a test ventilation’, without the use of long acting relaxants to allow a rapid return to spontaneous ventilation, might be considered.

Overall, our series contained a high proportion of seriously compromised children and yet we discovered a relatively low serious complication rate with general anaesthesia. We would suggest that this may be due, in part, to our early use of steroids. Steroids do cause shrinkage of both normal and malignant lymphoid tissue and thus potentially interfere with making an accurate histological diagnosis. Our results might suggest that provided steroid treatment is limited to a maximum of 5 days before a biopsy is obtained, an acceptable balance can be achieved between the desire to obtain an accurate diagnosis and the very real dangers of anaesthetising untreated children with a mediastinal mass.

In response to our findings and previously published evidence, we have constructed a set of local guidelines outlining the management of children presenting with a newly diagnosed anterior mediastinal mass at RMCH (Fig. 3). Whilst some aspects of it might be considered controversial, we feel it will provide a safe framework upon which to base the management of such cases considering our local population and resources available.

### Assess anaesthetic risk

**Clinical assessment**

- **Clinically high risk if following symptoms or signs present?**
  - Stridor
  - Orthopnoea
  - Wheeze
  - SVC obstruction
  - Collapse/dizziness

**High risk?**

- **Consider biopsy under LA if possible:**
  - BMA + LP
  - Pleural fluid aspirate
  - Lymph node
  - Other tissue?

- **Consider steroids:**
  - Prednisolone 60 mg m⁻² day⁻¹ with IV hydration & rasburicase
  - Monitor daily for improvement
  - Maximum 5 day course

**Not possible/no diagnosis?**

- **If GA required:**
  - Maintain spontaneous ventilation if possible.
  - Fibreoptic bronchoscope available

**Mandatory special investigations**

- CT scan
- Echocardiogram
- PEFR erect and supine

**Low risk?**

**Figure 3** Pre-operative anaesthetic guidelines for a child with a mediastinal mass.
Conflicts of interest

No conflicts of interest declared.

Part of this data was presented in poster format at the AAGBI Annual Conference, Dublin 2007.

Acknowledgements

We wish to thank Andy Vail, (Senior Lecturer, Biostatistics Group, University of Manchester) for his valued help with statistical analysis.

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