Transporting critically ill patients by ambulance: audit by sickness scoring

Patients who are critically ill may be safely transported by specialist teams but no prospective studies to investigate the efficacy of transporting such patients by ordinary ambulances attended by junior doctors have been reported. Such studies are important because most critically ill patients are probably transported in this way. Financial constraints may result in increasing use of ordinary ambulances to transfer such patients to hospital. We performed an audit of non-specialist transport within a district general hospital in which a sickness score was used to control for severity of illness.

Patients, methods, and results

Vascular and general surgery, coronary care, and most medical specialties are sited within a five mile radius of this intensive care unit; patients are therefore commonly transferred to the unit by ambulances, the journey taking a maximum of 30 minutes. The unit accepts all patients except those with uncomplicated head injuries.

We studied 50 consecutive patients transferred to the unit. Severity of illness was assessed with a sickness score, which is a modification of the acute physiology and chronic health evaluation (APACHE II) score. The sickness score was calculated from data collected immediately before and after transport. Arterial blood samples for blood gas analysis were taken before transport, packed in ice, and analysed together with a sample drawn on arrival. Patients were not monitored during the journey. Controlled ventilation was provided with an Ambu bag; patients who had not been intubated received a controlled supply of oxygen from MC facemasks. The partial pressure of inspired oxygen was measured with an oxygen meter. Complications occurring during transfer and the seniority and specialty of the medical attendant were noted. Survival was taken as discharge home.

Of the 50 patients, 31 had operations and 19 had medical conditions (repair of an aortic aneurysm; 13; acute renal failure, 12; sepsis, seven; cardiac arrest, five; and respiratory problems, 13). Seven patients, three of whom had had operations, developed eight serious complications during transfer: obstruction of an endotracheal tube, respiratory arrest on arrival at the hospital, accidental disconnection of arterial and central venous cannulas, withdrawal of crucial inotropic and bronchodilator infusions (two cases), and unrecordable blood pressure on arrival (three). Six of the seven patients were attended by junior staff (registrars or below) who were not anaesthetists ($\chi^2 = 10.79, p < 0.005$).

The table shows the numbers of patients and their mean sickness scores before and after transfer. The difference in scores between the survivors and non-survivors was highly significant ($p < 0.0001$). The mean score for non-survivors showed a small increase after transport, though this was not significant.

### Table: Sickness scores before and after transfer

<table>
<thead>
<tr>
<th>Category</th>
<th>Before transport</th>
<th>After transport</th>
<th>Mean difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Survivors</td>
<td>34</td>
<td>10.02</td>
<td>13.98</td>
</tr>
<tr>
<td>Non-survivors</td>
<td>16</td>
<td>19.83</td>
<td>20.64</td>
</tr>
<tr>
<td>Senior staff</td>
<td>27</td>
<td>12.09</td>
<td>12.53</td>
</tr>
<tr>
<td>Junior staff</td>
<td>23</td>
<td>14.11</td>
<td>15.09</td>
</tr>
<tr>
<td>Anasthetists</td>
<td>34</td>
<td>12.40</td>
<td>13.00</td>
</tr>
<tr>
<td>Other</td>
<td>16</td>
<td>14.10</td>
<td>15.10</td>
</tr>
<tr>
<td>Complications</td>
<td>7</td>
<td>15.00</td>
<td>16.00</td>
</tr>
<tr>
<td>No complications</td>
<td>43</td>
<td>12.60</td>
<td>13.30</td>
</tr>
</tbody>
</table>

### Comment

This study showed that life-threatening complications may occur in critically ill patients when conventional ambulances are used for transport. Complications were more common in patients attended by junior doctors and doctors other than anaesthetists and were not due to more severe illness among these patients. The training in resuscitation received by anaesthetists may be an advantage in caring for patients when monitoring is not available. The complications were not the direct cause of death in any patient, probably because of the short journey; longer transport times might have resulted in a significant increase in sickness scores.

Because severity of illness was controlled for, these results suggest that ineffectiveness in the management of patients who are critically ill is the dominant factor in the development of complications during transfer, confirming earlier work. These results support the need for improved training in resuscitation and suggest that blood pressure should be monitored during transfer.

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Is altered cardiac sensation responsible for chest pain in patients with normal coronary arteries? Clinical observation during cardiac catheterisation

Most patients with chest pain characterised as angina pectoris have obstructive atheromatous disease of the coronary arteries. In a few patients with angina pectoris exercise testing indicates abnormalities but coronary angiograms are normal. Various theories have been proposed to explain these findings, termed syndrome X, and which include abnormalities of coronary reserve or myocardial metabolism and abnormal histological appearances. During routine cardiac catheterisation we observed patients with syndrome X were unusually sensitive to intracardiac instrumentation. We report the findings of a preliminary study.

Patients, methods, and results

We studied seven patients with syndrome X (exertional chest pain associated with ST segment depression of more than 1 mm during exercise and normal coronary arteries); four patients with typical angina but negative results on exercise testing and normal coronary arteries; seven patients with atherosclerotic coronary artery disease; and nine patients with mitral valve disease.
Hyperthyroidism after gonadotrophic ovarian stimulation

We report two cases of hyperthyroidism associated with pharmacological ovarian stimulation in patients with underlying autoimmune thyroidis.

Case reports

Case I—A 29 year old woman was admitted for in vitro fertilisation and embryo transfer because of her husband's infertility. Her history was unremarkable.

Tests of thyroid function yielded normal results: free triiodothyronine concentration was 3.9 pmol/l (normal 2.8-5.6 pmol/l), free thyroxine concentration 9.2 pmol/l (normal 6.6-14.0 pmol/l), and thyroid stimulating hormone concentration 1.4 mU/l (normal 0.1-6.0 mU/l). Antibodies to thyroglobulin and microsomal antibodies were present (1/10 and 1/25600, respectively), and the concentration of immunoglobulins inhibiting binding of thyroid stimulating hormone was 65 U/100 ml (normal <10 U/100 ml). All hormonal determinations were performed during standard available radioimmunoassay kits. From day 3 after the onset of menses follicular stimulation was initiated with clomiphene citrate 100 mg twice daily for five days and enhanced by metronop (Humegon, Organon) follicle stimulating hormone 75 IU and luteinising hormone 75 IU/ml twice daily from day 7. When the follicles were sufficiently mature ovulation was induced by one injection of two phials of human chorionic gonadotrophin (Pregnyl, Organon; 5000 IU). During this procedure the patient developed symptoms of hyperthyroidism: free triiodothyronine concentration was more than 28.7 pmol/l, free thyroxine concentration greater than 48.7 pmol/l, and thyroid stimulating hormone concentration less than 0.1 mU/l. The serum thyroxine concentration was 4767 pmol/l (ovulation occurs at oestradiol concentrations of 1830-11000 pmol/l). She was cured after 18 months of treatment with propylthiouracil 100 mg three times daily.

Case 2—Graves' disease was diagnosed in 1983 in a 34 year old woman. She was successfully treated with methimazole. In 1986 she was admitted for in vivo fertilisation and embryo transfer, the indication being andrological. Tests of thyroid function yielded normal results: free triiodothyronine concentration was 6.6 pmol/l; free thyroxine concentration was 13.5 pmol/l; thyroid stimulating hormone concentration was 4.5 mU/l; microsomal antibodies were present (1/25600); and the concentration of immunoglobulins inhibiting binding of thyroid stimulating hormone was 5 U/100 ml. After a few days of treatment with metronop (Humegon) and before human chorionic gonadotrophin was given she developed symptoms of hyperthyroidism. Free triiodothyronine concentration was 9.7 pmol/l; free thyroxine concentration 29.9 pmol/l; and thyroid stimulating hormone concentration less than 0.1 mU/l. The serum oestradiol concentration was 4584 pmol/l. No antithyroid drugs were given apart from beta blockade; treatment with metonophop was stopped. Three weeks later all signs of hyperthyroidism resolved and the results of thyroid function tests returned to normal.

Comment

Although hyperthyroidism may have developed by chance in these patients with underlying autoimmune thyroiditis, the close temporal relation between ovarian stimulation and the occurrence of hyperthyroidism, and the spontaneous resolution of symptoms in case 2 after follicular stimulation was stopped, suggest that other mechanisms may have been implicated. As thyrototoxicosis that develops in the course of trophoblastic diseases has been postulated to be due to the stimulating effects of human chorionic gonadotrophin on the thyroid and as temporary aggravation of Graves' disease during early pregnancy coincides with maximal serum concentrations of human chorionic gonadotrophin¹⁰ it may be that a similar mechanism is involved in these cases. However, the possibility of a direct causative role for human chorionic gonadotrophin cannot be excluded.

References


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