# Clinical review

*Fortnightly review*

## Diagnosing and managing genitourinary prolapse

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Genitourinary prolapse occurs when faults develop in the mechanisms for vaginal and uterine support (fig 1). An understanding of these mechanisms and systematic repair of these faults will restore normal structure and function. Treatment of prolapse comprises about 20% of gynaecological surgical workload,[1] and with an aging, yet more active, population this contribution will increase. Cystourethrocele is seen most commonly, followed by uterine descent and rectocele. After a hysterectomy the vagina may be susceptible to prolapse owing to loss of support of the vaginal vault.

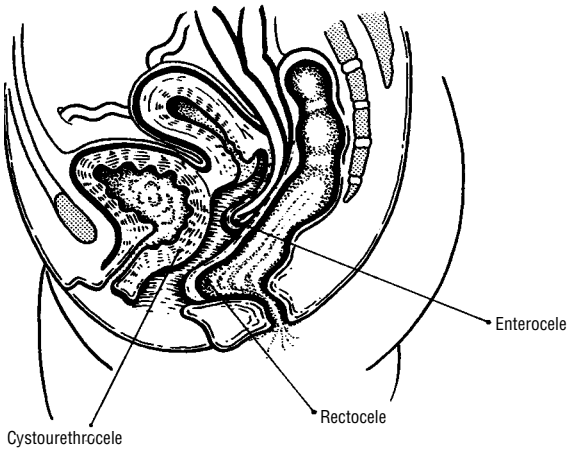


**Fig 1** Coronal section of pelvis showing cystourethrocele, enterocele, and rectocele

### Summary points

- Minor degrees of prolapse should be treated conservatively
- Sexual activity should be borne in mind when considering appropriate surgical procedures
- Long term results of surgery for prolapse are uncertain
- There is little published work comparing alternative procedures and techniques
- Reconsideration of what is normal pelvic anatomy has stimulated interest in restorative, reconstructive surgery for prolapse
- Concurrent urinary incontinence is not always secondary to prolapse and if present should be investigated before surgery

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### Methods

We conducted a Medline search from January 1966 to July 1996 and identified 8802 references to the term prolapse; these were reduced to 544 when we used the additional terms vagina, surgery, genitourinary, conservative, pessary, randomised, and outcome. We identified further references by hand searching relevant textbooks in the library of the Royal College of Obstetricians and Gynaecologists. We did not find any prospective trials comparing the effect of different treatments on outcome measures for prolapse. Observational studies have been published examining the pathophysiology and anatomy of prolapse, and these have stimulated interest in this subject.

### Anatomy

The pelvic viscera are supported by the pelvic floor, with the pubococcygeal portion of the levator ani decussating around the lower vagina and urethra before attaching anteriorly to the pubic bone (fig 2). The

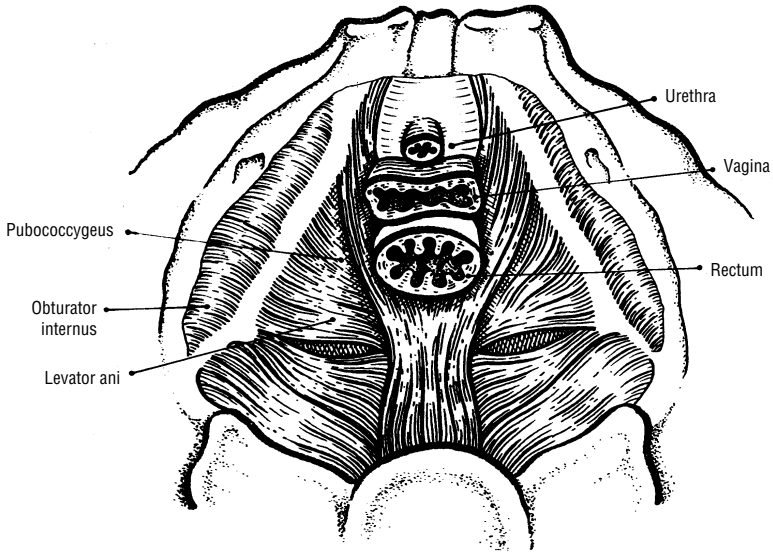


**Fig 2** Anatomy of pelvic floor

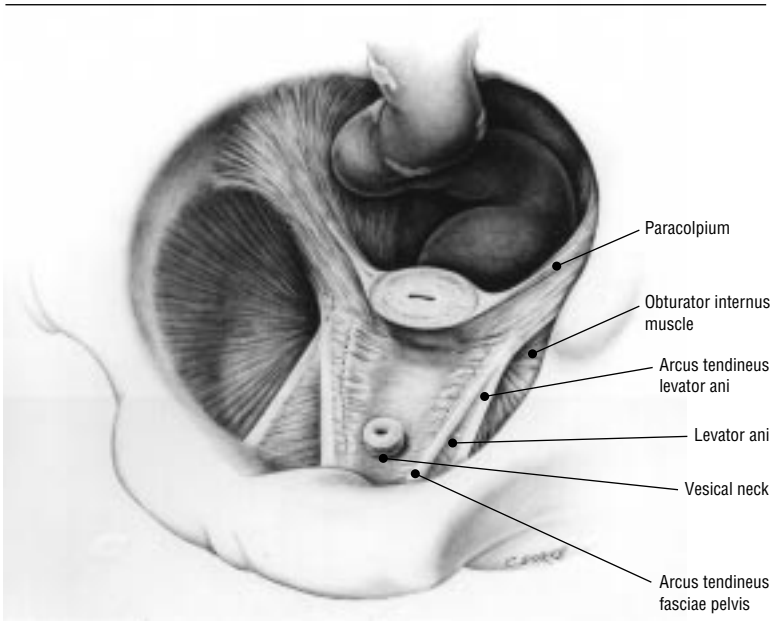


**Fig 3** Anatomy of vaginal support. The bladder has been removed at the vesical neck. (Reproduced with permission of JOL DeLancey)

vaginal wall consists of an inner epithelial lining surrounded by endopelvic fascia, which is composed of smooth muscle, elastin, and collagen and is attached to deeper pelvic supports. The cervix and upper third of the vagina are supported by the uterosacral and cardinal ligaments (part of the paracolpium). The middle third is attached by the pubocervical fascia to the arcus tendineus fasciae pelvis (the so called white line), which runs along the pelvic floor between the pubic symphysis and the ischial spine. The lower third is fused with the urogenital diaphragm, comprising the levator ani fascia, perineal membrane, and perineal body (fig 3).[2]

## Causes

The causes of genitourinary prolapse are summarised in the box.

### Causes of genitourinary prolapse

*Childbirth:*
Large babies
Long labours
Assisted delivery
Poor postnatal exercise regimens
*Congenital:*
Connective tissue disease
*Iatrogenic:*
Hysterectomy
*Increased intra-abdominal pressure:*
Obesity
Chronic respiratory disease
Pelvic masses

*Childbirth:* Vaginal delivery results in pelvic floor dysfunction, which manifests as urinary incontinence.[3] It may also predispose to subsequent prolapse. This may occur secondary to mechanical damage, particularly after forceps deliveries[4] or denervation of the pelvic floor.[5] The risk of denervation is increased by prolonged labour and large babies,[6] and prolapse is associated with such denervation.[7]

*Connective tissue disease:* Some women may have a congenital predisposition to prolapse because of abnormal collagen metabolism. Genitourinary prolapse is associated with joint hypermobility[8] and reduced vaginal collagen content.[9]

*Iatrogenic causes:* Division of the uterosacral and cardinal ligaments without reattachment to the vaginal vault at the time of hysterectomy predisposes to subsequent prolapse of the vaginal vault.[2] There is a further risk of enterocele after vaginal hysterectomy, probably due to inadequate approximation of the uterosacral ligaments at the time of surgery.

## Symptoms

Some of the symptoms of genitourinary prolapse are given in the box. Mild genitourinary prolapse may be an asymptomatic incidental finding noted at the time of vaginal examination. As such, it is best noted, but the patient should not be informed that she has a prolapse unless she mentions symptoms. Symptoms associated with more significant prolapse include feeling a lump within the vagina and observing a bulge if displacement is beyond the introitus. Displacement may result in dragging or aching discomfort, often localised to the back, and if prolapse is beyond the introitus tissue can become excoriated (decubitus ulcer), resulting in blood stained vaginal discharge.

### Symptoms of genitourinary prolapse

*Cystourethrocele:*
Urinary stress incontinence
Urinary retention
Recurrent urinary tract infections
*Uterine prolapse:*
Backache
Difficulty keeping tampons in
Ulceration if procedentia
*Rectocele:*
Dyschezia
Constipation
*Any prolapse:*
Lump coming down
Coital difficulties—dyspareunia, loss of vaginal sensation, vaginal flatus

Symptoms are often worse at the end of the day and after the patient has been standing for a long time. Coital problems, including loss of sensation and orgasm, dyspareunia, and vaginal flatus may be prominent. General discomfort in the vagina postmenopausally is more often associated with vaginal atrophy than prolapse, and a trial of topical vaginal oestrogen treatment daily for four to six weeks should be considered if the prolapse is mild.

Associated genitourinary symptoms may include urinary stress incontinence due to urethral hypermobility, although only 50% of women with genuine stress incontinence have clinically important prolapse of the anterior vaginal wall.[1] If cystourethrocele results in kinking of the urethra the urinary stream may be poor, with recurrent urinary tract infections if voiding is incomplete. In extreme cases chronic urinary reten-

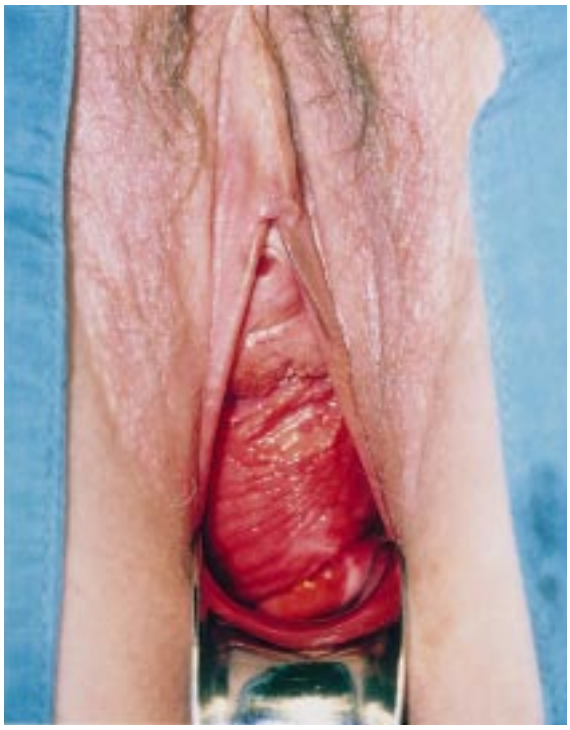

**Fig 4** Cystourethrocele and uterine descent

tion with overflow incontinence may ensue. Rectocele may cause difficulty with defaecation (dyschezia) or a sensation of incomplete defaecation, which is sometimes relieved by digital reduction of the prolapse.

The patient's perception of the trouble her symptoms cause must be considered when management options are being evaluated. Patients' perception should also become a key outcome measure in studies of genitourinary prolapse, but validated symptom questionnaires need to be developed before reproducible comparisons are possible.[10]

## Diagnosis

**Examination**

Genitourinary prolapse is diagnosed clinically (fig 4). Bimanual examination should be performed to exclude the rare possibility of an associated pelvic mass. A prolapse can usually be seen when the patient is lying on her back or side, although it may be necessary to examine her standing up to reproduce the conditions under which prolapse occurs. Descent to or beyond the introitus is observed after asking the patient to bear down.

The vaginal walls, fornices, and cervix are then assessed by inserting a Sims' speculum along the posterior vaginal wall. Gentle retraction of the posterior vaginal wall affords a view of the cervix, lateral and anterior fornices, and anterior vaginal wall. If important cystourethrocele is present the view of the cervix will be obscured and reduction with an examining finger or sponge forceps is necessary. The posterior vaginal wall is then assessed by retracting the anterior vaginal wall.

Enterocele and rectocele are difficult to differentiate clinically, although with the patient standing a cough impulse indicating an enterocele can be appreciated on combined rectal and vaginal examination.

Prolapse can vary in extent from some movement on coughing (this being normal in parous women) to descent to or beyond the introitus. For many years uterine descent has been classed as grades 1-3. Grade 1 is descent within the vagina, grade 2 is descent of the cervix to the introitus, and grade 3, or procidentia, is descent of the uterus outside the introitus. However, this classification is subjective and insensitive and takes no account of cystocele, enterocele, or rectocele. Detailed objective measures of the degree of prolapse are a prerequisite for evidence based studies, and recommended parameters for measuring pelvic organ prolapse have now been agreed by the International Continence Society.[11]

**Investigation**

As the diagnosis of prolapse is clinical, minimal additional investigation is usually required. However, it is important to be aware that other disease may

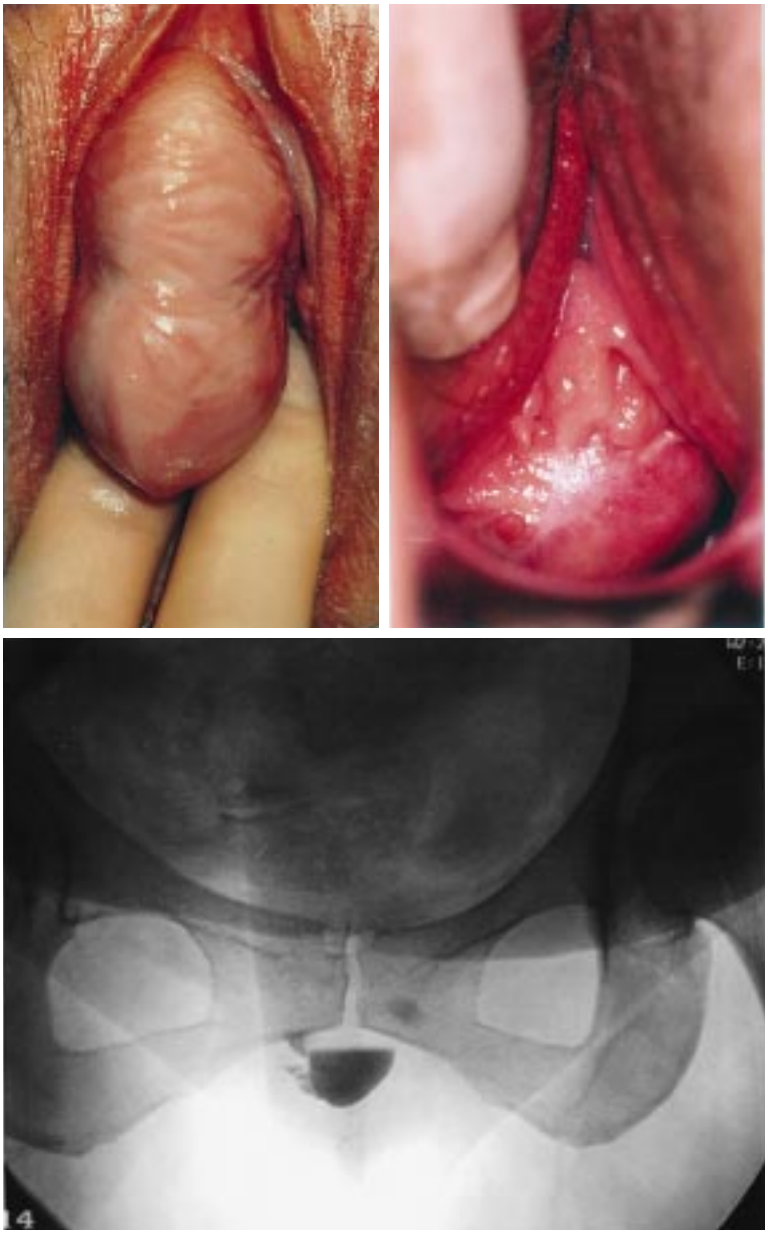

**Fig 5** Left: Paraurethral cysts are rare, but as they occur laterally to the urethra they can usually be differentiated from urethrocele clinically. Right: Urethral diverticula may occur in the midline, mimicking urethrocele. Bottom: Urethrogram shows diverticulum

occasionally be present (fig 5). With a large cystocele the ureterovesical junctions and lower ureters may descend, resulting in potential ureteric obstruction. Therefore, procidentia should be investigated by measuring serum urea and creatinine concentrations and by renal ultrasonography. Concurrent lower urinary tract symptoms such as incontinence should be assessed by cystometry before surgery. Cervical cytology is essential before considering hysterectomy, and any suspected pelvic mass should be investigated. The value of defaecography to evaluate posterior vaginal wall prolapse is undecided.

## Management

### Prevention

*Childbirth*—Appropriate management of labour may have a prophylactic role. The increasing rate of caesarean section in the United Kingdom and better management of labour, with a reduction in instrumental trauma and prolonged labour, may result in a reduction in the incidence of prolapse secondary to obstetric causes.

*Hormone replacement therapy*—Postmenopausal oestrogen supplementation increases skin collagen content[12] and causes trophic alterations in vaginal epithelium. Whether hormone replacement therapy increases the biomechanical strength of tissue or prevents the occurrence of genitourinary prolapse is unclear.

*Pelvic exercises*—Because of the anatomical connections between the pelvic floor, urethra, and vagina, exercising the pelvic floor may, in theory, prevent prolapse occurring secondary to pelvic floor laxity.

### Conservative management

Incidental mild prolapse found at the time of routine pelvic examination, if not associated with symptoms, needs no treatment. Often reassurance and explanation is all that is required. With mild symptoms conservative management should be offered in the first instance. The risks of surgery and anaesthesia should not be taken lightly, especially in elderly women, and the long term morbidity from surgery, including coital difficulties and pain, are often understated.

*General*—Simple treatment of exacerbating factors such as obesity and concurrent chronic coughs is likely to ameliorate the condition.

*Hormone replacement therapy*—Hormone replacement therapy increases postmenopausal vaginal collagen turnover,[13] but whether spontaneous anatomical remodelling and repair of established prolapse can occur is unknown.

*Pelvic floor exercises*—Pelvic floor exercises are an established treatment for urinary stress incontinence,[14] but whether they benefit established prolapse has not been studied.

*Vaginal pessaries*—Genitourinary prolapse can be reduced with vaginal pessaries (box). Pessaries may be appropriate while the patient is awaiting definitive surgery and when surgery is declined or contraindicated because of pregnancy or for medical reasons. Pessaries are commonly rings, and, although they are made of inert plastic, they should be changed every six months to prevent erosion of or embedding in the vaginal wall. The use of oestrogen cream with vaginal pessaries reduces discomfort and erosion. Sizes vary, and the appropriate size is determined at the time of digital examination by estimating the distance from the posterior aspect of the symphysis pubis to the posterior vaginal fornix. Occasionally, although the pessary seems to be the right size, rings will not stay in place. In this case a shelf pessary can be helpful, especially with vault prolapse or enterocele. As well as being used for definitive treatment vaginal pessaries can be used diagnostically: when it is unclear whether a patient's symptoms stem from uterovaginal prolapse the effect of reduction can be assessed by temporary insertion of a pessary. Relief of symptoms would then be an indication for surgery.

### Indications for use of vaginal pessaries

- If patient is medically unfit for surgery
- To gain relief from symptoms while awaiting surgery
- If further pregnancies are planned
- If patient is in the first trimester of pregnancy
- As a diagnostic test to relieve symptoms thought to be due to prolapse
- As a diagnostic test to ensure that correction of a large cystourethrocele would not cause stress incontinence

### Surgery

Ideally, surgery will correct prolapse while maintaining coital function and preserving continence. It is important to ask whether the woman is sexually active before considering vaginal surgery as this may alter the surgical approach, or indeed defer surgery. Care not to reduce the vaginal capacity with overaggressive or repeated vaginal surgery is essential but has been overlooked in the past partly because of a failure to appreciate the underlying anatomical defects. A move towards reconstructive vaginal and abdominal surgery for prolapse and away from excisional, obliterative surgery should reduce this often hidden morbidity.

*Types of repair*

Anterior colporrhapy, otherwise known as anterior repair, has been the favoured operation for cystocele (fig 6). Colpoperineorrhaphy, or posterior repair, is favoured for rectocele. Care must be exercised when removing the redundant vaginal epithelium as vaginal narrowing can result in severe dyspareunia. This is common after posterior repair, particularly when midvaginal levator sutures have been inserted.[15] The levator ani muscles do not normally meet between the rectum and vagina, and suturing them together at the time of posterior repair will lead to coital pain. Prolapse of the anterior vaginal wall may be due to detachment of the lateral vaginal support to the arcus tendineus fascia pelvis. In this case paravaginal repair, either by the transvaginal or abdominal route, is gaining popularity.[16 17]

It has been asserted that surgical cure of some forms of cystourethrocele is associated with subsequent stress incontinence, perhaps secondary to an intrinsic problem with the sphincter that is revealed when the urethra becomes straight again. However, a preoperative and postoperative prospective clinical

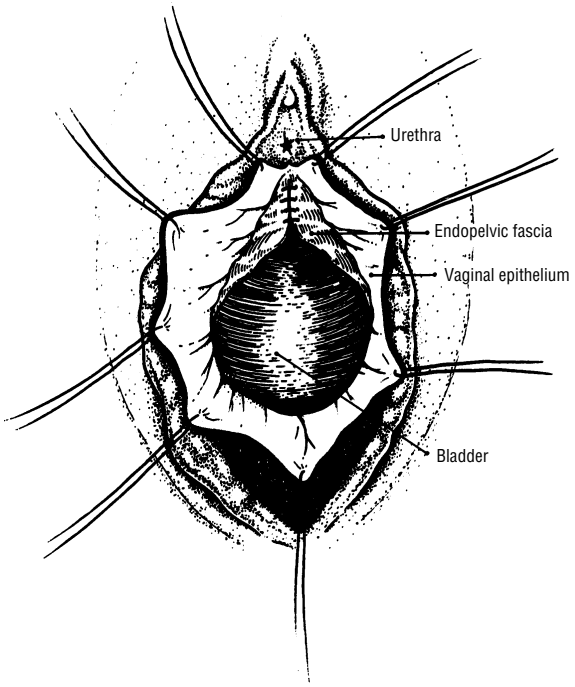


**Fig 6** Anterior colporrhaphy. Redundant vaginal epithelium is excised and endopelvic fascia is opposed before closing vaginal incision

and urodynamic study has shown no evidence that bladder or urethral function is compromised by colporrhaphy or vaginal hysterectomy,[18] although excessive and unnecessary dissection of the bladder neck should be avoided in women who are continent.

If there is concurrent urinary stress incontinence a Burch colposuspension will correct cystocele, as well as giving excellent long term urinary continence.[19] When uterine prolapse is present vaginal hysterectomy is the procedure of choice. This can be combined with anterior or posterior repair when, as is commonly the case, concurrent cystocele and rectocele are present. Although uncommonly performed today, cervical amputation with a Manchester or Fothergill repair can be performed for mild uterine descent, especially when the cervix has become enlarged and conservation of the uterus is desired. A retrospective comparison of Manchester repair with vaginal hysterectomy for uterine prolapse found that both procedures had a similar outcome.[20]

*Recurrence of problems*

The incidence of recurrent prolapse is reported to be 16%.[21] This may be due to a failure to correct the precise initial anatomical defect or it may arise as a complication of the original surgery. Such examples include enterocele after Burch colposuspension, cystocele after sacrospinous fixation, and rectocele or enterocele after sacrocolpopexy. Vaginal vault prolapse will occur if the vault is not secured to the uterosacral ligaments at hysterectomy. Repair can be effected vaginally or suprapubically, and the patient's medical condition and wishes about sexual activity need to be considered when planning surgery.

The simplest procedure is colpocleisis, or occlusion of the vaginal lumen, which can be performed under local anaesthesia. This is appropriate only for sexually inactive women, but it has low morbidity and a low rate of recurrence[22] and is a useful technique in frail elderly women. Sacrospinous fixation, with stitching of the vaginal cuff to the sacrospinous ligament, does not alter vaginal capacity, and recovery time is quick as it is a vaginal repair. Although infrequent, complications are serious as damage can occur to the pudendal artery, pudendal nerve, or sciatic nerve. Injuries may be minimised by avoiding the lateral third of the sacrospinous ligament and placing the stitch superficially.[23] One year cure rates of 90% have been reported with this technique.[24]

Sacrocolpopexy uses the abdominal approach, the vaginal vault being attached by non-absorbable mesh to the sacral promontory.[25] Vaginal anatomy is not distorted, but this procedure also carries the risk of haemorrhage from the sacral venous plexus. Cure rates of 88%-97% have been reported between one and 10 years later.[26 27] Alternatively, the Zacharin procedure corrects the anatomical defect by closing the levator hiatus and suturing the vagina to the levator plate.[28] It entails more extensive dissection than colposacropexy, and a retrospective comparison of the two procedures, both performed by the same surgeon, has shown colposacropexy to have superior results.[29] Laparoscopic sacrocolpopexy has also been described,[30] but no long term follow up data are available.

*Outcome measures*

We did not find any studies that had examined different surgical techniques in a prospective controlled manner. In addition to the procedure performed, outcome may depend on the skill of the surgeon and the quality of the tissues. Improving tissue quality preoperatively with oestrogen has been assessed in one randomised placebo controlled trial.[31] Vaginal wall thickness was increased and the incidence of postoperative cystitis decreased. Long term outcome was not assessed.

## Conclusions

The treatment of genitourinary prolapse is under review. The lack of controversy and research based evidence may, in part, reflect misplaced satisfaction with established practice. However, early studies of recurrence rates and coital satisfaction after vaginal surgery suggest that a hidden morbidity needs to be considered. Until recently, no scientific methodology has been available to assess symptoms or objective degree of genitourinary prolapse; without such methodology the design of clinical trials has been problematic. A better understanding of the underlying pathoanatomy of prolapse has generated increasing interest within this subject, and further research is required into both the underlying pathophysiology and clinical outcome of conservative and surgical treatment.

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*Lesson of the week*

# Acute non-cardiogenic lung oedema after platelet transfusion

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**Consider transfusion related acute lung injury if lung oedema occurs after blood transfusion**

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The commonest cause of lung oedema after transfusion of blood products is acute hypervolaemia leading to heart failure, especially in elderly anaemic patients. However, non-cardiogenic causes of lung oedema after transfusion, though less common, may result in considerable illness and in death, especially if they are not recognised and treated appropriately. One such cause is transfusion related acute lung injury, which is the second commonest immediate cause of death related to transfusion after acute haemolysis due to ABO incompatibility.[1]

We describe a case of transfusion related acute lung injury caused by incompatibility between two donors contributing to a pool of platelets given to our patient.

## Case report

A 67 year old man was referred to the haematology department for investigation of anaemia. He had felt tired for six weeks, and a full blood count had shown pancytopenia (haemoglobin concentration 64 g/l, white cell count $2.7 \times 10^9$/l, neutrophil count $1.8 \times 10^9$/l, platelet count $32 \times 10^9$/l). He had previously been well and had no history of ischaemic heart disease. He had taken diclofenac for backache for many years and did not have a history of drug allergy. Physical examination was unremarkable except that he had marked pallor. A chest *x* ray film was normal (fig 1 (top)).

He was admitted to hospital and given four units of ABO compatible crossmatched blood, followed by a further four units of ABO compatible crossmatched blood two days later, both without any complications. Results of bone marrow aspiration and trephine biopsy led to a diagnosis of acute megakaryoblastic leukaemia. His platelet count fell to $16 \times 10^9$/l, and he was given a pool of platelet concentrates, again without any complications. A pool of platelets consists of platelet concentrates from four separate donors and plasma from one of these donors.

Three days later he received a further pool of platelet concentrates, which was infused over 30 minutes. Fifteen minutes later he became acutely breathless and cyanosed and developed rigors. He had not yet received any chemotherapy, and he was only taking fluconazole and using antiseptic mouthwashes. He was not feverish. His blood pressure was 140/80 mm Hg and pulse 110 beats per minute. Auscultation of the heart showed normal heart sounds without additional sounds or murmurs. Jugular venous pressure was not raised. Widespread inspiratory crepitations were evident throughout both lung fields. Arterial blood pH was 7.48, arterial oxygen tension 8.0 kPa, and arterial carbon dioxide tension 4.1 kPa while he was breathing

room air. A chest *x* ray film showed diffuse bilateral shadowing of the lungs and a normal sized heart (fig 1 (middle)). An electrocardiogram showed a sinus tachycardia but no other abnormalities.

Cardiogenic lung oedema was diagnosed, and he was treated with 60% oxygen and intravenous frusemide, diamorphine, and nitrates. On review the following day the lung oedema was shown to be non-cardiogenic: the clinical response was poor and the patient had persistent hypoxia, a poor diuretic response to frusemide (a total of 160 mg was given), an unchanged chest *x* ray film, no evidence of acute ischaemia or arrhythmia on an electrocardiogram, normal cardiac enzyme concentrations, and normal left ventricular function on an echocardiogram. Transfusion related acute lung injury was diagnosed in view of the temporal relation of the respiratory distress to the platelet transfusion, and treatment with intravenous dexamethasone 8 mg twice daily was started. There was clinical improvement over the next 48 hours, and radiological changes improved over 72 hours (fig 1 (bottom)).

As leucocyte antibodies are central to the pathogenesis of transfusion related acute lung injury, serum from the patient and the donor of the plasma for the pool of platelets was screened for granulocyte specific and HLA antibodies. The patient's serum was negative for both. The plasma donor was a multiparous woman and her serum, although negative for granulocyte specific antibodies, was strongly positive for HLA antibodies against A2 and A28. These are closely related antigens, with considerable crossreactivity. Antibody to HLA-A2 is a potent leucoagglutinin and has been implicated in cases of transfusion related acute lung injury.[2 3] The patient's tissue type was negative for HLA-A2 and HLA-A28 antigens and his leucocytes did not react with the plasma used in the platelet pool. Therefore, the donors of the three other platelet concentrates were also recalled, and one of them was found to be positive for HLA-A28 antigen. Leucocytes from this donor reacted by lymphocytotoxicity and leucoagglutination with the serum of the donor of the plasma used in the platelet pool. Hence, the pathogenesis of this case of transfusion related acute lung injury seems to have been incompatibility between two donors used in the pool of platelets.

## Discussion

Transfusion related acute lung injury, first described over 50 years ago,[4] is a serious complication of blood transfusion that may be overlooked. The incidence in the United States is about 1 in 5000 transfusions.[5] About 70% of patients require mechanical ventilation, and, although up to 80% of cases resolve within 48 hours, the mortality approaches 6%.[5] Transfusion related acute lung injury is characterised by acute lung oedema with marked hypoxia, often associated with fever, rigors, and hypotension, that occurs during or within a few hours of transfusion but without evidence of hypervolaemia or myocardial insufficiency (box).[5] As in our case, patients present with sudden respiratory distress, and chest radiography shows marked bilateral lung oedema without cardiomegaly. The pulmonary oedema is not cardiogenic in origin, and this is confirmed by normal results on electrocardiography

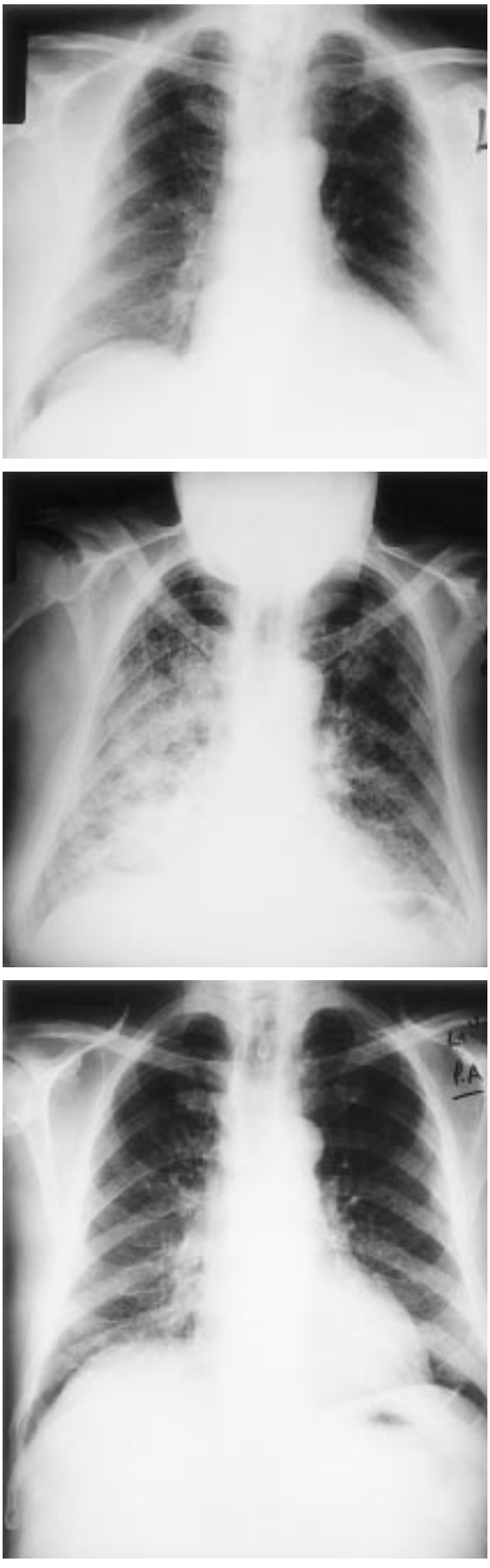

**Fig 1** Chest *x* ray film one day before platelet transfusion (top), shortly after platelet transfusion (middle), and three days after intravenous dexamethasone (bottom)

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## Characteristics of transfusion related acute lung injury

- Recent transfusion, usually within six hours
- Sudden onset of dyspnoea
- Hypoxia
- Rigors
- Fever
- Non-cardiogenic pulmonary oedema as suggested by:
  - Unlikely hypervolaemia
  - No history or symptoms of heart disease
  - Poor response to conventional treatment of lung oedema
  - Normal cardiac enzyme concentrations
  - Normal electrocardiogram and echocardiogram

and echocardiography. The differential diagnosis includes cardiac overload due to hypervolaemia; acute haemolytic transfusion reaction; bacterial sepsis with shock; and acute anaphylaxis in patients with IgA deficiency caused by IgA antibodies in the recipient reacting with IgA in the blood product infused.

Most cases of transfusion related acute lung injury are caused by the transfusion of plasma containing leucocyte agglutinating antibodies,[6] although non-agglutinating antibodies have been implicated.[3] Such antibodies are often seen in multiparous women donors as a result of sensitisation with fetal white cells during pregnancy. They may be granulocyte specific antibodies,[3 7 8] HLA antibodies,[2 3] or 5b antibodies.[9] Occasionally, the recipient may be sensitised as the result of pregnancy or previous transfusions and develop antibodies that react with transfused leucocytes.[3] Rarely, as in our case, transfusion related acute lung injury may be caused by antibodies in the plasma of one donor reacting with leucocytes in the blood product from another donor rather than with recipient leucocytes.[2] The implicated antibodies activate leucocytes and complement in the lung vasculature, liberating anaphylatoxins, which in turn promote cytokine production and neutrophil chemotaxis and aggregation in the lungs. This leads to capillary endothelial damage, fluid leakage into the alveoli, and lung oedema. Chemotaxis of recipient neutrophils mediated by C3a and C5a may also contribute to the pathogenesis. This may have been particularly relevant in our case. Why the lung is the main site for this leucocyte activation and subsequent tissue damage is unclear. The first microcirculation to be hit by any antibodies or immune complexes formed as the result of transfusion is the lung's. In addition, although this is relevant only to HLA antibodies, the lung endothelium is lined with macrophages rich in HLA class I antigens.

An important part of the diagnosis of transfusion related acute lung injury entails testing donor and recipient serum samples for granulocyte and HLA antibodies. Leucocyte crossmatching by leucoagglutination or lymphocytotoxicity will confirm leucocyte incompatibility. Leucoagglutination tests look for agglutination of leucocytes, or more usually granulocytes, in response to the presence of granulocyte specific or HLA antibodies. Lymphocytoxicity tests detect HLA antibodies by their cytotoxic effect, mediated by complement, in a panel of HLA typed lymphocytes. A suspected case of transfusion related acute lung injury must be thoroughly investigated as implicated donors should be prevented from giving further donations. Also, if the donor should require a transfusion, leucodepleted blood products need to be given. The donor implicated in our case was removed from the panel and any unused donations, such as fresh frozen plasma, were recalled.

Management of this condition is supportive, and high doses of steroids may be beneficial. If fluid overload is not a factor diuretics are of no benefit and may be detrimental.[10] Transfusion related acute lung injury is probably underdiagnosed because a high proportion of patients who receive transfusions are under general anaesthesia or because the respiratory distress is attributed to other causes, mainly the adult respiratory distress syndrome. Physicians should be alert to the possibility of transfusion related acute lung injury when acute lung oedema develops after transfusion of blood products containing plasma, particularly when cardiogenic causes are unlikely. Its recognition is important in view of the associated illness and death, for instituting correct management, and for eliminating dangerous donors from donor panels. Screening all multiparous women donors for leucocyte antibodies would help eliminate transfusion related acute lung injury as a complication of transfusion. However, these measures require money, time, and effort. An alternative would be to exclude multiparous women's plasma from clinical use. The use of platelets collected by apheresis from a single donor, which has been advocated to reduce donor exposure, would prevent similar cases to ours. The main disadvantage of using platelets produced only by apheresis would be wastage of platelets in routine blood donations.


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