penile urethra. In female pseudohermaphroditism the external genitalia of a female fetus are masculinised by exposure to high concentrations of circulating androgens—for example, in congenital adrenal hyperplasia. Giving androgens to the mother during pregnancy may have the same effect. The condition is compatible with normal female development provided it is treated early, the external genitalia being refashioned appropriately.

Fortunately all these disorders are rare; together they account for fewer than 1 in 1000 births. They are, however, reason enough to advise against over-confident prediction of sex based on antenatal analysis of the karyotype.


---

**Rheumatoid constrictive pericarditis**

Often silent and symptomless and discovered only at necropsy, pericarditis is nevertheless common in rheumatoid arthritis. In his original account of 19 cases of "a form of chronic joint disease in children" Dr George Still found physical signs "suggestive of adherent pericardium" in two and at necropsy adherent pericarditis was found in three others. In adults Hollingsworth² quotes pericarditis as being found at necropsy in 30-50%, of cases of rheumatoid arthritis and comments that when it is clinically apparent pericarditis is usually a complication of the later stages of the disease—though in both children and adults it may occur early and may even herald the onset of the disease. Like so many of the extra-articular manifestations of the disease, pericarditis is relatively more common in men than in women. Tamponade from effusion is rare but may occur,³ and pericardectomy may be required.⁴ In a recent report the co-existence of cardiac tamponade and constrictive pericarditis was described in a 54-year-old man.⁵

Before 1946 the commonest cause of constrictive pericarditis was tuberculosis. This has now changed, and today pyogenic arthritis and rheumatoid arthritis are more frequent causes. Moreover, the course of the complication in the latter may be quick: one patient whose disease started in January 1962 had to have a pericardectomy for constrictive pericarditis in July the next year.⁶ At operation the whole heart was found to be constricted with a fibrous covering 1-5 mm thick. Dissection of the pericardium from the constricting sclerotic covering of the heart was not difficult, but separating the epicardial surface was much more laborious.

Similar cases have been reported from time to time,⁸ ⁹ and recently John et al¹⁰ have described six patients with rheumatoïd constrictive pericarditis, five seen within two and a half years. All were men, all had rheumatoid factor in the blood (usually in high titre), and all had active nodular rheumatoid arthritis. Diagnosis was suspected from physical examination and confirmed in five patients by cardiac catheterisation, and all had a successful pericardectomy; the sixth case was diagnosed only at necropsy. Before the onset of symptoms of pericarditis the disease had been present for an average of 12 years, but in one case articular and pericardial symptoms had come on simultaneously. Four patients had had a chronic insidious illness, symptoms lasting 4-5-44 months (average 12 months) before diagnosis, and they presented with congestive cardiac failure with peripheral oedema. Two consistent features were a raised jugular venous pressure (which increased with inspiration (Kussmaul's sign)) in three of the five, a paradoxical pulse being present in four and enlargement of the liver in five; four had interstitial pulmonary fibrosis.

John and his colleagues make the good point that, as the only common features in these patients were rheumatoid arthritis and right heart failure, this combination should always alert the clinician to the possibility that rheumatoid constrictive pericarditis or pericardial tamponade, or both, may be present. Indeed, all patients with rheumatoid disease in whom right heart failure develops should be considered to have pericardial disease until this has been excluded. Fortunately pericardectomy is an effective method of treatment.¹¹

---

**Surgical diathermy is still not foolproof**

In surgical operations the use of diathermy current for coagulating blood vessels and cutting through vascular tissue permits a high standard of haemostatic control not readily achieved by any other means. Equally valuable is the use of diathermy cutting and coagulation through endoscopes, techniques which have been exploited particularly by urologists.

The tissues are heated by using a high frequency oscillating current, most modern diathermy machines generating frequencies of 0-5-5 MHz. The current oscillating between two electrodes in contact with the body heats all the tissues between them; hence the name diathermy (Greek dia: through, and therme: heat). The heating effect is inversely proportional to the area of contact with the electrodes, and so the indifferent electrode is made as large as possible. If the electrode is small, the heating effect is concentrated, and so the active electrode—and thus the volume of tissue in contact with it—is kept as small as possible.

The use of diathermy currents in operating theatres carries certain dangers. When a high-intensity current is used, sparking may occur. The danger of explosions from anaesthetic gases or bowel gases is well recognised and can be avoided. The heat from diathermy or electric cauteries may be sufficient to cause explosion or ignition of skin preparations containing as little as 20%, alcohol. Iodine may explode in the presence of nitrous oxide, and nobecutane may explode in the presence of nitrous oxide or high oxygen concentrations.¹

Nevertheless, the commonest cause of accidents associated with diathermy is defective contact of the indifferent electrode with the patient.² Any diminution in the area of contact increases the heating effect and a burn develops. If there is
no contact, or if the indifferent lead is broken, the circuit may still be completed by leakage of the current to earth, particularly if the machine is producing frequencies above 1 MHz. The earth lead of the power supply cable forms the final circuit link to the diathermy machine. If any part of the patient's body touches an earthed metal fitting such as a drip stand or arm rest, leakage occurs preferentially along this path and a burn develops at the point of contact.

New types of diathermy machines are now available, some with transistored circuits which may incorporate additional safety features. In some new machines the diathermy circuit is isolated from earth. If the indifferent electrode is not applied to the patient, or if its lead breaks, the circuit cannot be completed by current leakage to earth, and hence the machine should fail to function. The newly available machines have been tested in Bristol, including one machine that is claimed to incorporate an earth-free diathermy circuit. Provided the intensity of the current was increased, this worked satisfactorily, even when the indifferent electrode was suspended in mid-air, out of contact with any object. This, and other machines that functioned in this manner, operated at frequencies above 1-5 MHz. It was thought that at high frequencies stray leakage of current might be as much as 25\% of some machines operated satisfactorily when the indifferent electrode was placed on the top of the diathermy cabinet, or on the pedestal of the operating table, or connected to an earthed object such as the anaesthetist's trolley or theatre radiator pipes.

The surgeon, as well as the patient, may be at risk from burns from conventional diathermy machines, particularly when using endoscopes. Earth-free diathermy circuits are claimed to protect the surgeon from pericellular burns during endoscopic diathermy procedures, but this was found not to be so when using machines operating above 1-5 MHz. Manufacturers should be encouraged to produce machines with frequencies below 1 MHz and to provide insulated covers for the diathermy cabinets. Even more important, surgeons and theatre staff should remain cautious when using any diathermy machine, and should not consider it foolproof.

Benign recurrent vertigo

Patients often complain of dizziness and disorders of equilibrium, but their symptoms need careful and precise analysis to determine whether they do indeed suffer from vertigo or from light-headedness or some other complaint. Vertigo—best defined as an illusion of altered position or motion that may have objective manifestations—may be the presenting symptom of many different diseases, including temporal-lobe epilepsy, acute ophthalmoplegia, conditions associated with raised intracranial pressure, and some cerebellar disorders; vascular, post-traumatic, congenital, neoplastic, infectious or demyelinating processes affecting the brain stem; cerebello-pontine-angle lesions and lesions of the upper cervical roots; abnormal reactions to a number of drugs; and systemic conditions such as anaemia or hepatic dysfunction. Nevertheless, despite the large number of illnesses in which vertigo may play a part, the most common cause is disordered function of the labyrinth, and the condition most often responsible is Menière's disease.

Diagnosis requires, then, a careful clinical history with physical and neurological examination, particular attention being paid to eye movements. Simple tests of positional nystagmus and other bedside tests of eighth nerve function should be included. Caloric testing, audiometry, radiographs of the skull and internal auditory meatuses, electroneystagmography, and an electroencephalogram make up the routine investigations.

At the end of all these tests certain cases of doubtful vertigo without cochlear signs remain a diagnostic problem. Some of the patients may be suffering from benign recurrent vertigo, a term used by Slater to describe a syndrome of repeated attacks of acute vertigo, which last from minutes to hours and are followed by a period in which positional nystagmus may be found if looked for. The attacks vary in frequency from once a day to once or twice a year. All findings are entirely normal except for the results of electroneystagmography, which show some labyrinthine abnormality. Among the clinical characteristics are a strong family history and precipitation of the vertiginous attacks by lack of sleep, emotional stress, or alcohol. Some patients have headaches strongly suggestive of migraine.

The aetiology of the syndrome is unknown. It bears some resemblance to benign positional vertigo \(^2\) or vestibular neurotis,\(^3\) but there are distinct differences. In children a similar disorder has been called benign paroxysmal vertigo of childhood \(^4\) or paroxysmal dysequilibrium of childhood.\(^5\) The disease known as familial paroxysmal nystagmus with vertigo and ataxia\(^6\) also resembles the syndrome delineated by Slater. Again there tends to be a strong family history and precipitation of attacks by alcohol and lack of sleep.

Slater considers that benign recurrent vertigo may be a variant of migraine; certainly there is good evidence that migraine may affect the vestibular apparatus,\(^7\) and there are several reports of vertigo in migraine.\(^8\) In such circumstances vertigo has been labelled\(^9\) as "migraine equivalent"; it may also be seen as a "disorder of an episodic type occurring in migraine patients."\(^10\) Whatever the diagnostic label, the syndrome has a good prognosis. Slater did not use drugs in the treatment of his patients with benign recurrent vertigo, but prophylactic propranolol and pizotifen have given excellent results. Recognition of the clinical syndrome is therefore important because of its benign nature and response to treatment.

---


---