have been some striking reversals of securely held concepts, and he would be a bold man who dares to say that parity has fallen forever. The conundrum of defining right and left may yet return to haunt us.

Lacking as we do the amenity of wearing a miniaturised physical laboratory on the wrist, and in general not experiencing undue difficulty in differentiating left from right, one is forced to the conclusion that this innate capacity is ultimately dependent on the asymmetry of our bodies. It is therefore appropriate to turn our attention to the phenomenon that, where asymmetry exists in the structure of the organisms of a specie, the asymmetry is constant—that is to say, it is in the same direction in all members (with rare exceptions) of that specie. I refer here to the asymmetries that are observable, for example, in the visceral anatomy of vertebrates, the claws of some crustacea, the spiral shells of some molluscs, and the helical turn of tendrils and some stems. It is not enough to ask why these structures are asymmetrical. We must also ask why the asymmetry is constant in direction. In the course of embryological development there must be a force that turns the enlarging structures in the same direction in all members of the same species. Were this not the case, roughly half the population would have situs inversus, just as, in the absence of a biasing force, a coin will fall heads or tails with equal probability. The work of Afzelius on the immotile cilia syndrome has thrown some light on the area from which this force may emanate, since about half of the subjects of this syndrome have situs inversus, often presenting as Kartagener's syndrome. Some subcellular elements are helically structured and the direction of spiralling is constant. It can only be a matter of time before

a causal chain of events is discovered linking the laevo-amino-acids, which are universal in the essential chemistry of all living things, with the constant laterality of subcellular elements—perhaps something akin to the “coiled coil” of electric light bulb filaments. We are thus driven to the conclusion (or shortly will be) that constant anatomical asymmetry on which our sense of right and left is based, derives from our amino-acids being of laevo form. Retropolating (to coin a phrase) to the origin of life on earth, it was probably by chance that our primordial amino-acid progenitor (PA would seem a suitably paternal abbreviation) was laevo, though a bias has been suggested. It is a curious thought that had PA been the enantiomorph (mirror image molecular structure) our hearts would all be right-sided and a reversal of existing asymmetries would hold throughout the animal and plant kingdoms. What we would in that case have called left and right is best left to professional philosophers.

This is not the place to discuss the cultural sequelae of handedness whence are derived the associations of right with what is correct, just, proper, and straight, and left with what is sinister and inappropriate. These associations, however, have spilled over into the terminology of direction. Some years ago when motoring in France, I inquired the way and was told, “Tout droit, tout droit.” I was about to turn right but was saved by my informant adding “Au fond de la rue.” We likewise may confuse the foreigner by saying, “Carry right on,” unless we add, “. . . to the end of the road.” Dutchmen please note; “Recht door. Tot het eind van de weg.” No confusion here with “Gerade-aus.” Finally, the story must be told of the schoolboy who translated the royal motto “Dieu et mon droit” as “My God, you're right!”

Lesson of the Week

Pulmonary oedema after transfusion with fresh frozen plasma

P C O'CONNOR, J G ERSKINE, T H PRINGLE

Less than 1% of patients have allergic reactions after transfusion with fresh frozen plasma.¹ Severe allergic pulmonary oedema occurring after transfusion with fresh frozen plasma, however, can be fatal.² Standard treatment regimens consist of giving varying doses of antihistamines, corticosteroids, and diuretics.^{1 2}

Allergic pulmonary oedema after transfusion with fresh frozen plasma may be associated with hypovolaemia and need treatment with intravenous fluids

Case report

A 58-year-old man who had five episodes of haemorrhage after dental extraction was referred for investigation and

correction of any haemostatic defect before further urgent dental surgery. Investigations showed a consistently prolonged kaolin cephalin clotting time of 54 s (control 34 s) but normal bleeding time, one stage prothrombin time, thrombin time, platelet count, and factor assays of I, II, V, VIII coagulant activity, VIII related antigen, IX, X, XI, XII and XIII. The reason for his susceptibility to bleed is not known.

Treating him with four units of fresh frozen plasma on the day before dental surgery temporarily corrected the coagulation defect. On the next day before transfusion with fresh frozen plasma he was clinically well. Systemic blood pressure was

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120/75 mm Hg and pulse 84/min (sinus rhythm). Shortly after beginning the fourth unit of plasma the patient complained of feeling warm and lightheaded. He was afebrile (37.2°C) and clammy with warm hands and feet. He had a sinus tachycardia of 130/min, with a palpable systolic blood pressure of 60 mm Hg. The fresh frozen plasma was discontinued and intravenous chlorpheniramine maleate (10 mg) and hydrocortisone sodium succinate 100 mg were given. After 30 minutes his blood pressure had risen to 80/60 mm Hg, but four hours later there was no improvement and chlorpheniramine maleate 10 mg and hydrocortisone sodium succinate 100 mg were again given.

Six hours after the initial reaction the patient became increasingly dyspnoeic and crepitations were audible throughout both lung fields. A chest x-ray film showed widespread pulmonary oedema. An electrocardiogram (ECG) showed no evidence of myocardial infarction. A presumptive diagnosis of left ventricular failure was made, and despite giving 40 mg of frusemide intravenously and a high concentration oxygen his condition was unchanged one hour later. Therefore he was given a further 80 mg of frusemide intravenously and 40 minutes later he passed 400 ml of urine. His clinical condition, however, appeared to deteriorate with a fall in blood pressure to 60/40 mm Hg. Arterial blood gas analysis showed pH 7.38, P_{O_2} 5.32 kPa (40 mm Hg), P_{CO_2} 4.26 kPa (32 mm Hg).

Because his condition was worsening a Swan-Ganz catheter was inserted through a right antecubital vein and floated into the pulmonary artery where the pressure was 15-17/0-7 mm Hg (normal 15-28/5-16 mm Hg),³ using the patient's mid-chest as reference point. The catheter was advanced and pulmonary capillary wedge pressure was 1 mm Hg (normal 5-12 mm Hg).³ Cardiac output measured by a thermodilution technique was 8.7 l/min. Calculated systemic vascular resistance was 432 dyn sec cm^{-5} (normal 600-900 dyn sec cm^{-5}),³ which was characteristic of systemic vasodilation.

A fluid challenge with 500 ml of 0.9% saline given in 30 minutes raised the blood pressure to 110/50 mm Hg and the pulmonary wedge pressure to 5 mm Hg. After this successful response he required another 1000 ml of 0.9% saline over the next six hours to maintain satisfactory blood pressure and pulmonary wedge pressure. His recovery was uneventful. Subsequent chest x-ray examination showed that the pulmonary oedema had cleared, and sequential ECGs and cardiac enzymes failed to show evidence of myocardial infarction. We could not find the basis of the severe allergic reaction. In particular no anti-IgA or anti-Gm antibodies were detected, and no vasoactive substances were shown in the remaining fresh frozen plasma.

Comment

Pulmonary oedema usually occurs after pulmonary capillary hydrostatic pressure rises as, for example, in left ventricular decompensation or mitral stenosis. Other mechanisms that damage the integrity of alveolar capillary membranes can also lead to accumulation of fluid in the alveoli.⁴ In this patient the pulmonary oedema was probably due to fluid leaking across the pulmonary alveolar capillary membrane which was damaged by an immunological reaction.

Measuring the pulmonary capillary wedge pressure showed that the oedema was not due to left ventricular decompensation, but in retrospect this might have been suspected from the chest x-ray film which showed pulmonary oedema without venous congestion of the upper lobes. His warm hands and feet also made a diagnosis of cardiogenic shock unlikely. Although frusemide is known to be beneficial in pulmonary oedema secondary to raised left atrial pressure, it did not lead to any improvement in this patient. Indeed, as a consequence of its venodilation⁵ and diuretic effects it may have contributed to the hypotension. The hypotension associated with allergic pulmonary oedema is secondary to systemic vasodilation with relative hypovolemia, which should be treated with fluid replacement. Dextran, a useful plasma expander, has occasionally been associated with anaphylaxis and impaired coagulations,⁶ which made it unsuitable for our patient.

When allergic pulmonary oedema occurs after transfusion with fresh frozen plasma, antihistamines and corticosteroids used to treat the allergic response may need to be combined with 0.9% saline to correct hypovolemia if it occurs.

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Are the plastic mouthguards used by sportsmen a hazard in a physical contact game such as rugby football?

There are two types of plastic mouthguards: one can be moulded to the roof of the mouth with the use of boiling water and the other is a specially fitted type made by a dentist. Most international rugby players started wearing mouthguards after the British Lions tour of New Zealand in 1971. This resulted from a paper in a New Zealand medical journal recording the reduced incidence of concussion and the reduced severity of faciomaxillary injuries in players wearing mouthguards. The only hazard to the player is that they are difficult to get used to wearing. If they are properly fitted there is no danger of their being dislodged, and if they are of sufficient size no risk of inhalation or swallowing.

What treatment is advised for an incontinent diabetic woman who two years ago had a colporrhaphy for long-standing procidentia?

Incontinence eventually recurs after colporrhaphy in 20-50% of patients.¹ A carefully taken history² is essential to distinguish recurrent stress incontinence from other types, such as urge incontinence.

Examination may show recurrent cystocele, though anatomically the anterior vaginal wall may be quite normal. Cystometry should be carried out if available, but it is not essential.¹ This patient's diabetes should be kept under good control in case polyuria is contributing to her problem. Non-operative treatment includes oestrogen cream, pelvic floor exercises, bladder training, and antispasmodics, such as emepromium. Pessaries are unlikely to help. Various electrical devices stimulate the muscles of the pelvic floor, but their benefit is short-lasting.³ Unless the patient is a very poor operative risk a second operation should be considered—but only if she clearly has stress rather than urge incontinence. A second attempt at vaginal repair may be indicated if she has pronounced cystocele, though the chances of success are less on the second attempt. A suprapubic approach is preferable—either a sling (which is technically difficult) or, better, urethropexy, such as the Marshall-Marchetti-Krantz procedure. The fact that many different operations are described indicates that none is perfect, but in properly selected cases the success rate of operation should be over 70%.

¹ Zacharin RF. Abdominoperineal urethral suspension—a ten-year experience in the management of recurrent stress incontinence of urine. *Obstet Gynecol* 1977;**50**:1-8.

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³ Edwards L, Malvern J. Electronic control of incontinence: a critical review of the present situation. *Br J Urol* 1972;**44**:467-72.