in a clinical state characterised by inaccessibility, little coherent speech, rejection of food and drink, and apparent disorientation, but without overt affective features. Sometimes these are confusional states, when befuddlement is a prominent feature; such a presentation should strictly be described as confusional or delirious rather than as pseudodementia. But clouding of consciousness is rarely prominent in these cases and may therefore be difficult both to identify and to measure. The patient may then be incorrectly diagnosed as suffering from dementia. Why depression should present in this way in the aged is a question to which we do not yet have an answer, but it seems likely that study of the neurochemistry of the aging brain may provide the answer. The term pseudodementia is best confined to these dementia-like presentations of depressive illness. Diagnosis may be especially difficult in the aged when the remediable depressive component is obscured by a degree of actual dementia, but such patients may benefit from skilled care.

What guidelines exist which may enable the clinician to diagnose depression when it masquerades as dementia? Firstly, a short history should raise suspicions, and the duration of the illness and its mode of onset must be established. This will include finding out whether or not there have been "life events," most commonly losses, which might be relevant. Secondly, a family history of depressive illness must be sought (this may be suppressed by relatives).[15] Thirdly, Post has suggested that patients with pseudodementia are apt to complain of their memory defects—indeed "they may almost parade them"—in contrast with the common "anosognosia"—where there is a loss of the ability to recognise one's illness—of true dementia.[16] In pseudodementia patients tend to give clear accounts of their illness and their past life in response to open ended questions, and reply "I don't know" only to direct questions. They often become distressed and make little effort to perform even simple tasks, while patients with dementia do their best, and it is only when they overstretch themselves that they may exhibit a catastrophic reaction.

In his clinical guidelines, Post suggests that the term pseudodementia should be reserved for patients who are capable of cooperating with detailed cognitive assessment. But even elderly, disorganised patients whose affective state may be difficult to assess may have a depressive illness. Hence it is important to seek for evidence of depression, which may be provided by relatives or nursing staff who look after the elderly patient around the clock. They may witness a transient stage when the patient emerges from incoherence or inaccessibility to utter suspiciously depressive statements, for example, "I don't deserve this food." Sensitive observation may detect a sad facial expression or demeanour, which may be transient but points to the diagnosis.[15] If such observations are linked with either a previous history or a clear family history of depression, or other corroborative evidence such as a potentially relevant "life event," a trial of antidepressive treatment may be justified. It is important to ensure that the patient's fluid and electrolyte balance is maintained, for old people who are mentally ill may be incapable of maintaining an adequate fluid intake. Such patients often need the joint care of psychiatrists and physicians.[17]

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Unequal legs

A difference in the length of a child's legs is a source of considerable anxiety to parents. The first important task for the doctor is to establish whether the discrepancy is due to a true difference in the length of the limbs or is an apparent difference—or a combination of the two.[1] Apparent inequality may be due to a hip deformity such as congenital dislocation, a spinal deformity such as scoliosis with tilting of the pelvis, a fixed flexion deformity of the knee, or an equinus deformity of the ankle. In such cases treatment must be directed to the underlying abnormality.

True inequality alters with growth in several different ways. By no means all discrepancies in length increase at a constant rate with time. Various patterns have been identified: some slow down as growth proceeds, some may reach a plateau before the cessation of growth, while others may at first increase, then stabilise, and then decrease—without surgery.[2] This variation reflects the disparate causes. Poliomyelitis may produce considerable shortening, particularly if the paralysis affects the limb early in life. Hemiplegic cerebral palsy usually produces some shortening, but rarely more than 4-5 cm. Soft tissue abnormalities that produce hyperaemia, such as neurofibromatous and haemangiomatous malformations, may increase the length of the leg, as may infections and fractures. Underdevelopment of the femur and leg bones with or without absence of the whole or part of these bones may be due to congenital defects.

Overgrowth is usual after a fracture of the leg.[3] In a series of 74 children under 13 years with fractures of the femoral shaft the average femoral overgrowth was 0·92 cm. This overgrowth occurred regardless of the age of the child at the time of the fracture, its level, or the position of the fracture at

the time of healing. Ipsilateral overgrowth of the tibia averaging 0·29 cm occurred in four fifths of the patients. Most overgrowth occurred within 18 months of injury, but in seven of the 74 patients overgrowth continued throughout the remaining growth period, though at a slower rate. The clear implication is that when children have fractures of the long bones some overlap of the displaced fractured bone ends is preferable to perfect reduction. If a fracture damages an epiphysial plate, causing its partial closure, the resulting shortening and angular deformity may be corrected by excision of the bony bridge produced by premature fusion of the epiphysis. The excised bone is replaced by a free transplant of fat, which allows space for regeneration of the undamaged growth plate[4] and continuation of normal growth.

In the first year of life accurate measurement is impossible and generally unnecessary. A useful method is to mark the tip of each malleolus with a ball point pen and while the ink is still wet apply traction to each foot and press the two ankles together. If the legs are equal in length the two marks will coincide; and if not a gap will show the difference between the two sides.[5] As the child grows clinical measurement needs to be supplemented by accurate radiographic assessment of bone length with a scanogram—a motorised tube head that passes along the length of the limbs and produces an image on an extra long cassette. Accurate forecasting of the ultimate differences in limb length requires radiographs of the wrist to estimate skeletal age. The measurements may then be interpreted in relation to growth charts of femoral and tibial length for boys and girls produced by Anderson and Green.[6]

Surgical correction should be considered when the disparity in limb length at skeletal maturity lies between 3 cm and 15 cm.[7] Lesser differences may readily be masked by minor alterations to footwear—the addition of a small raise to one heel and the removal of a small amount from the other. Most patients are comfortable with a raise slightly less than the exact difference in limb lengths. The simplest method of equalising the length of the legs at the end of growth is by shortening the normal limb by resection of a length from the femur or the tibia and fibula. The main disadvantage is that the patient's height is reduced. This is not a major consideration where the patient is of average height or taller, but the technique is unsuitable for those who are relatively short. Shortening of more than 6·5 cm in the femur and 3 cm in the tibia is not advisable.[7] An alternative procedure is to arrest the growth of the normal limb at the distal femoral or proximal tibial epiphysis by epiphysiodesis, an operation introduced by Phemister in 1933.[8] Growth is retarded by excision and grafting of the epiphysial plate at an appropriate age to allow the abnormal limb to "catch up." This approach requires careful timing.

Surgical lengthening of the leg has a long history and a poor reputation because of the high rate of complications. These resulted from overstretching, inadequate fixation of the bony fragments, and interference with the blood supply with delayed and non-union of the femur or tibia.[9] In recent years, Wagner[1] has improved the technique, successfully overcoming these hazards by secure fixation to the bone of a powerful but neat distraction device attached to one side of the limb only, which enables the patient to walk with crutches two or three days after operation. The technique is still time consuming and requires at least two surgical operations and a period ranging from six weeks to six months in hospital with 12 to 18 months or longer on crutches. In over 150 limbs Wagner has been able to achieve an increase in length of 16 cm in the femur and 8 cm in the tibia.

Shortening of the leg is the easiest procedure and the least time consuming of the methods available, but it has the disadvantage that the operation is on the normal limb. Lengthening is more complex but it can help to equalise much bigger differences in length than can shortening; it requires experience which is not gained in routine orthopaedic practice and is best performed in specialist units.

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Gall stone pancreatitis

In many parts of Europe alcohol is implicated much more often than biliary tract disease in the aetiology of acute pancreatitis,[1 2] but gall stones remain the most common cause in Britain, being held responsible in about half of all cases.[3 4] As small stones may remain undetected in some patients thought to have idiopathic pancreatitis and in some where pancreatitis is attributed to alcohol this figure is probably an underestimate. Patients with gall stone pancreatitis tend to be older than those with alcohol associated disease, and while the overall mortality of acute pancreatitis has fallen to around 10% gall stone pancreatitis remains the more dangerous condition.[1-3]

The fact that gall stones can be recovered from the faeces in some 90% of patients in the first few days after an attack of gall stone pancreatitis[5 6] has supported the concept that pancreatitis in these patients is due to impaction of stones at the lower end of the common bile duct. Impaction proves transient in most cases, and migration of the offending stone into the intestine allows inflammation to resolve. The stones recovered from the faeces are often small, so it is not surprising that they are often difficult to detect radiologically while still in the biliary system. Oral cholecystography and intravenous cholangiography are known to be unreliable in the acute phase of pancreatitis,[7 8] and failure of the gall bladder to opacify cannot be accepted as firm evidence that it is diseased. Similarly, radionuclide scanning is of debatable value, and a strong association reported by Glazer *et al*[9] between obstruction of the cystic duct and gall stone pancreatitis has not been confirmed by others.[8] Ultrasonography, which offers non-invasive investigation without reliance on hepatic function, identified stones in 31 out of 33 patients submitted to early biliary surgery after admission with