right lung, there is usually no sharp line of demarcation between it and the denser medial structures; consequently the border cannot be seen. It is for the same reason that the more anterior of the medial borders of the lung are not shown.

A glance at the sketch will reveal at once why this line has great diagnostic significance, for it shows the close relation of the medial border of the lung to the lateral surface of the vertebral body. If the latter is crushed its transverse

Transverse section of body at 9th dorsal level. The thickened lines are of the postero-medial border of the pleura.

diameter will be increased and the medial border of the lung will be displaced to that extent laterally. If the fracture is associated with a paravertebral haematoma this will show as an added expansion and further lateral displacement of the medial border of the lung. The antero-posterior radiographs show this as a fusiform expansion of the medial opacity, for the postero-medial border of the right lung will be similarly displaced. In a case which came to court the existence of a crush fracture of the vertebral body was disputed until the presence of a paravertebral haematoma was demonstrated to the judge on the radiograph taken immediately after the accident and its absence from radiographs taken a fortnight later. But the commonest demonstration of lateral displacement of the postero-medial border of the lung is due to the paravertebral tuberculous abscess. It is important because it may be detected before any change in the bone has become apparent. The abscess may extend the whole length of the dorsal spine, is usually fusiform in shape, and is sometimes of the width of the heart shadow; indeed, it has occasionally been missed because it was mistaken for the latter. It may show progressive expansion from the time it is first detected. Because from the level of the reflection of the pleura on to the diaphragm there is below no contrast in the densities of the adjacent tissues, the outline of the abscess cannot be traced in the abdomen—i.e., we are dependent upon the lateral displacement of the postero-medial border of the radiotransparent lung for visualization of the abscess. Consequently, though the abscess may have arisen from caries of one of the lumbar vertebrae, if no bone changes are yet recognizable its origin may not be apparent.

Incidentally, it may be stated that in the abdomen a lateral deviation of the line of the psoas muscle may help us to detect the site of an acute abscess, or a chronic abscess before calcium has been deposited within it. For anatomical reasons this line of the psoas may appear to be continuous with that of the postero-medial border of the lung.

Points in Differential Diagnosis

Because of this common appearance of the dorsal tuberculous paravertebral abscess there is a tendency to diagnose all fusiform deviations of the postero-medial border of the lung as tuberculous in origin, but appreciation of the anatomical features I have described which give rise to these shadows induces further discrimination. Thus neoplasm, both primary and secondary, may cause collapse and an increase in the transverse diameter of the diseased vertebral body: this will cause a lateral deviation of the postero-medial border of the lung, which will be increased, if there is proliferation of neoplastic cells or if associated with haemorrhage, to such an

extent that it may resemble a paravertebral abscess. It should be remembered, however, as a feature in differential diagnosis, that neoplasm appears to spare the disks even though all the vertebral body is destroyed.

Fusiform paravertebral expansions have been seen at the site of pneumococcal, typhoid, and paratyphoid abscesses. In these the destructive changes are more frequently present in the disk, which appears to collapse, and within a few weeks the postero-medial border of the lung is straightened out again. The vertebral bodies ultimately fuse. In the acute phase of vertebra plana the involved body is surrounded by fusiform expansion, which within a few weeks gradually disappears, leaving the disk apparently intact but the elements of the vertebral body compressed to a quarter or a third of the normal depth. In Paget's disease that is localized to one or more dorsal vertebral bodies the postero-medial border of the lung is deviated laterally by the expansion of the bodies. In osteochondritis of the spine in infants and adolescents some degree of thickening of the tissues adjacent to the vertebral bodies occurs, and the medial border of both lungs may become apparent just lateral to the border of those bodies; but in this there is no localized fusiform expansion.

In one case rupture of the posterior wall of the aorta was associated with haemorrhage into the areolar tissue surrounding the vertebral bodies: an appearance simulating a paravertebral abscess was produced. Any inflammatory or neoplastic proliferation within the vertebral body or the areolar tissue surrounding it may produce changes such as have been described, and it may be necessary to seek elsewhere for a clue to the identity of the organisms and cells producing the displacement of the lung. Empyema localized to this site would also push the lung laterally.

Summary

The line of demarcation formed at the flattened postero-medial border of the lung by the sharp contrast between the relative radiotransparency of the lung and the radio-opacity of the vertebral tissues is of valuable diagnostic significance in cases of backache.

The anatomical investigations which contributed to the discovery of the cause of this radiographic sign were carried out in the anatomy department of the University of Birmingham with the kind permission and assistance of Prof. C. V. Smout.

Medical Memoranda

A Case of Bilateral Bell's Palsy

Double Bell's palsy has always been looked upon as an uncommon phenomenon. Jelliffe and White (1935) consider it rare, as do Osler and McCrae (1935), Price (1941), Savill (1939), and Walshe (1941). Numerous cases of facial diplegia have been quoted, but the majority of them have been due to some condition such as epidemic encephalitis, trauma, or a lesion at the base of the brain. Bashford and Wilson (1918), Holmes (1917), and Viets (1927) have recorded numerous cases of facial diplegia due to other causes. Currier (1928) discusses facial diplegia and records two cases, but these are not of the type commonly called bilateral Bell's palsy. Most authorities now agree with Patrick (1916) that Bell's palsy is due to an infection elsewhere plus exposure to cold, although the infection is not always apparent.

CASE HISTORY

An electrician aged 17 was seen at the out-patient department of the Royal Infirmary, Sunderland, on Dec. 9, 1940. About a fortnight previously he was unable to whistle; food had a tendency to drop out of his mouth; and when he washed himself the soap got into his eyes. He stated that he had suffered from a catarrhal "cold" about a week previously. There was no history of ear trouble, whether deafness, pain, or discharge.

On examination both sides of the mouth were drooped, furrows of the brow were indefinite, naso-labial folds on both sides tended to be obliterated, and there was a general loss of expression in the face. Both eyes could be opened against the patient's efforts to keep them closed, the left side perhaps more so than the right. He was unable to close both eyes completely, and wrinkling of his brow was poor. He was unable to whistle or blow out either cheek. Attempts to show his teeth were poor, and the mouth tended to be drawn to the right side to a slight degree. No loss of sensation was detected over the face. Taste on the anterior two-thirds of the tongue was normal. There was no marked sensitivity to loud noises. The other cranial nerves were normal, as were the fundi. The central nervous system showed no other abnormality, and the circulatory, respiratory, alimentary, and urinary systems were normal. Radiographs of the skull revealed nothing unusual.

Examination of the cerebrospinal fluid showed: No cellular increase; protein 50 mg. per 100 c.cm.; negative Wassermann reaction; Lange curve, 0123444421. A blood count gave Hb 84%; R.B.C. 4,300,000; C.I. 1; W.B.C. 7,200—polymorphs 52%, lymphocytes 35%, eosinophils 6%, large mononuclears 7%, basophils 0. The red blood cells were normal in size. The electrical reactions were as follows: Brisk faradic response in both sides of the face; galvanic response normal; slightly sluggish on right side of face. Mr. Jowett of the E.N.T. department reported that no abnormality was detected in the ears and that hearing was normal.

The patient's condition improved gradually, and by Dec. 21 it had practically cleared up. The fact that his electrical reactions remained normal, the paralysis was not complete, and sensation in the tongue was normal bears out the usually accepted idea that recovery will occur rapidly. This was so in the present case.

Re-examination, March 21, 1942.—During the past year he has felt well and has noted no abnormality with regard to his face. He has continued at work without a day's absence. The pupils are equal, the eye movements full, and the power in both eyelids normal. He can wrinkle his brows normally, and the lines of his face on both sides are well marked. He can whistle, and the sensation of his face and tongue is normal.

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X-ray Investigation of Stridor in a Child

Histories of illness in childhood are of necessity obtained second-hand. Although parents are keen observers, it is inevitable that vital facts in a story may be omitted simply because the child was alone when some particular episode occurred. This applies particularly to the habit children have of inserting all sorts of objects into their various orifices. Then it is that x-ray examination may reveal an unsuspected foreign body, and provide the answer to a puzzling train of symptoms and signs. The following case shows how radiographs revealed the cause of a tracheal stridor which had been present in a child for a period of over one year.

CASE HISTORY

A boy of 2½ years was brought to the Princess Elizabeth of York Hospital for Children on account of stridor. The history was that he was well until 13 months before admission, when he had choked over the first mouthful of his afternoon meal and had vomited it. Immediately following this he had been unable to swallow anything but fluids for the ensuing 10 days, after which the power to swallow solids had gradually returned, accompanied by the development of a stridor. He had had the stridor constantly since then, and the mother had noticed that it had been more marked for the last two months. There had been no dysphagia after the initial 10 days, and in all other respects he seemed well.

Ten months after this episode the child was admitted to another hospital for radical cure of a hydrocele. Whilst awaiting operation he had developed otitis media, so the operation was postponed. Later his tonsils and adenoids were removed, and he was finally discharged after a total of 11 weeks, without operation having been performed. Radiological investigation of the stridor was not carried out, although its presence at that time was noted, and was, in part, the reason for postponing operation.

On investigation, 13 months after its onset, the stridor was found to be constant, chiefly inspiratory, and increased by activity. The child was well nourished, and no abnormality was to be seen in the pharynx. The chest was clear. There were a bilateral chronic otitis media and a right hydrocele. Radiographs showed a cog-wheel ¾ in. in diameter lying in the coronal plane at the level of the sterno-clavicular joint and posterior to the trachea, which was narrowed to a slit at this point. The supposition that the wheel was in the lumen of the oesophagus was confirmed by a barium swallow, which showed the barium stream being divided into two by the hub of the wheel. Under general anaesthesia the wheel was successfully removed by grasping it in crocodile forceps passed down an oesophagoscope. Its removal was followed by the discharge of a few c.cm. of foul-smelling pus and some fresh blood.

Post-operative progress was satisfactory. The child was given no fluid by mouth for 24 hours, an intravenous drip glucose-saline being maintained for this time. Thereafter fluids by mouth were started in small quantities and rapidly increased. There was no dysphagia, and no pyrexia after the first 24 hours. The stridor gradually decreased, and was practically absent when the patient was discharged three weeks later. A barium swallow given two weeks post-operatively showed considerable residual deformity of the oesophagus, but no obstruction. A follow-up is to be carried out to detect the possible onset of a stricture.

I am much indebted to Mr. C. P. Malley for permission to report this case, and to Dr. C. Hilliard of Hertford County Hospital for the radiographs taken.

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Reviews

ANAESTHETICS AFLOAT

Anaesthetics Afloat. By Surg. Lieut.-Commander Ronald Woolmer, R.N.V.R., B.M., B.Ch., D.A. With an introduction by Surg. Capt. H. D. Drennan, D.S.O., R.N. (Pp. 120; illustrated. 6s.) London: H. K. Lewis. 1942.

A surprising amount of sound and useful information is collected here. The author has a pleasant style and gives essential information without weighing this down with unnecessary details. From his own experience of anaesthesia at sea he deals clearly with special problems that may be met with there, and this practical teaching is invaluable to those who must work under emergency conditions. The chapters on "Mishaps and Difficulties in Anaesthesia," on "Anaesthesia in Relation to War Wounds, Injuries, and Shock," and on "Mode of Anaesthesia for Various Operations" are especially good. Full details of the preparation of patients for general anaesthesia are given, and the chief methods of spinal analgesia and the most useful types of local analgesia are described and illustrated.

Among the few controversial points are these: Many anaesthetists would consider that this author's advice that morphine for premedication be given three-quarters of an hour before operation would lead to respiratory depression during the induction, in cases where ether is used, and it is now more commonly taught that morphine should be given one and a half hours before operation. The instruction that a patient who has developed ether convulsions should be placed in the Trendelenburg position is contrary to the usual teaching, which advises the head-up (anti-Trendelenburg) position. Then, whereas the correct warning is given not to use general anaesthesia for operation for peritonsillar abscess, yet it is surprising to read that local anaesthesia should be achieved with the ethyl chloride jet. One wonders why a cocaine spray is not advised, since the average patient would inhale enough ethyl chloride to become unconscious.

The usefulness of intravenous barbiturates is rightly stressed, but the statement about evipan and pentothal, that "both drugs, however, have a very high margin of safety," true though it is for the experienced person, may give an inexperienced anaesthetist undue confidence with them. Very sound advice is included as to the care with which endotracheal intubation should be performed. The instructions on the treatment of a patient during operation under spinal analgesia are excellent, and could be read with great advantage by those anaesthetists, and also surgeons, who habitually disturb the patient by asking him if he is feeling pain.

This book will really fit into the pocket; it has a special usefulness in time of war, and the author is to be congratulated on this valuable addition to the literature.

PROBLEMS OF BLOOD TRANSFUSION

Blood Substitutes and Blood Transfusion. Edited by Stuart Mudd, M.A., M.D., and William Thalheimer, M.D. (Pp. 407. \$5.00 post paid or 27s. 6d.) Springfield and Baltimore: Charles C. Thomas; London: Baillière, Tindall and Cox. 1942.

This "symposium monograph," edited by Stuart Mudd and William Thalheimer, is based on papers read at the annual meeting of the American Human Serum Association held in 1941 and revised and brought up to date in the spring of 1942. The subjects discussed include aetiology and mechanisms of secondary shock; methods of preservation of plasma and serum; haemoglobin, serum albumin, and casein digests as blood substitutes; storage, transportation, and administration of whole blood with special reference to iso-agglutinins and group-specific substances; and therapeutic experiences with serum and plasma. There is little reference to work carried out in England in both the preparation and use of blood derivatives. In the last two years literally thousands of litres of both serum and plasma have been prepared and administered in this country. It is odd, therefore, to read papers devoted to an account of the administration of 250 c.cm. of concentrated serum to one patient.

In spite of the extensive experiences of observers here showing that haemoconcentration does not occur to any marked degree in traumatic shock, Virgil Moon still insists on its