Medical Memoranda

Spontaneous Pneumothorax and Subcutaneous Emphysema

Spontaneous pneumothorax and subcutaneous emphysema are both rare complications of asthma, and the combined conditions even more rare. So far as I know, no case of subcutaneous emphysema recurring during a subsequent asthmatic attack has previously been reported.

CASE REPORT

A boy aged 1 year 9 months was admitted to hospital on November 13, 1955, with a history that three weeks previously he had had a coryza with "wheezing," which had cleared in a week. Five days before his admission he developed another cold in the head, and later began coughing and wheezing. On admission the child was miserable and frightened. He cried lustily while being examined, and then quite suddenly swellings began to appear on each side of his neck, and these gradually increased in size. The crackling sensation elicited on palpation showed these to be due to subcutaneous emphysema. Clinical examination of the chest was impossible, as the child was very apprehensive, but a chest x-ray examination showed that he had a right pneumothorax and also demonstrated the subcutaneous emphysema of the neck.

During the next two or three days this subcutaneous emphysema spread up into his face and down over the anterior and posterior chest walls, and then gradually disappeared. Treatment consisted in sedation and a course of oxytetracycline.

A month later the child was readmitted for four days with a mild attack of asthma, which gradually subsided without treatment.

On May 12, 1956, the boy was again admitted, this time with a 24-hour history of breathlessness and wheezing, for which he had been given ephedrine, without improvement. His temperature was 99.6° F. (37.6° C.), his right tonsil was injected, and there were generalized rhonchi in the chest. On sedation he settled down well. The following morning the child, although generally much better, seemed to have a rather puffy face. This was found to be due to subcutaneous emphysema which had developed overnight and involved the neck and chest wall as well as the face.

Over the next 48 hours this increased in extent and then disappeared during the following week. An x-ray examination of the chest on this occasion showed consolidation of the anterior segment of the right lower lobe. There was no pneumothorax. Treatment was again sedation, and he was also given a course of sulphadimidine. At no time was the child particularly distressed by the presence of the subcutaneous emphysema.

Family History.—The patient was an only child. His mother suffered from hay fever. His father gave a rather interesting story: he had been in the R.A.F., and described how, when undergoing an examination in which he had to perform the Valsalva manoeuvre, he had suddenly developed a swelling in the neck. In his case, however, further investigations proved that he had a laryngocele.

COMMENT

Various theories on the mechanism of subcutaneous emphysema complicating asthma have been suggested, but the most generally accepted seems to be that an alveolus ruptures and air escapes into the lung tissues and tracks along perivascular spaces to the hilum and then along the fascial planes into the neck. It is possible for the pressure of this escaping air on the large vessels at the hilum and in the mediastinum to cause cardiac and respiratory embarrassment. Some air may track towards the periphery of the lung and collect under the pleura. If the pressure of this causes

the pleura to rupture a pneumothorax will result. Alternatively, an emphysematous bulla may rupture during an attack and produce this complication.

Air is usually absorbed quickly from the tissues, and it is only if the rate of escape *into* the tissues is exceptionally great that any distress is likely to occur. In a crying, frightened child conditions exist for this to take place—hence the importance of sedation.

Only two fatal cases have been reported. In one of these (Peterson, 1947) the subcutaneous emphysema was thought by the pathologist at necropsy to be incidental, the outstanding feature being the blocking of respiratory passages by tenacious mucus. The other patient (Wagner and Wagner, 1937) had, in addition, a localized spontaneous pneumothorax, and the final asthmatic attack lasted five months.

Two cases have been reported in which incision of the subcutaneous tissues has been necessary to allow the escape of air. One case occurred in a man of 21 who had suffered from asthma most of his life (Schrire and Schrire, 1952). During an asthmatic attack he developed gross subcutaneous emphysema, which spread down his arms to his hands and over the trunk as far as the groins. His breathing was laboured and his pulse rate rose to 150 a minute and was irregular. Following a transverse incision in his neck with exposure of the trachea, he made a good recovery.

Thirty cases of subcutaneous emphysema complicating asthma have been reported in the literature, but as this condition is usually benign, requiring no specific treatment, it is possible that it may be far more common.

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Action of Senna

On February 23, 1957, two reports were published in the *British Medical Journal* (Browne *et al.*, 1957; Duncan, 1957) demonstrating the effectiveness of senna preparations as laxatives in controlled clinical trials. At that time the precise mode of action of senna and similar compounds was attracting our attention. It is traditional in pharmacological teaching to classify a number of purgatives as "irritant." Not only senna, but cascara, rhubarb, aloes, mercury, and sulphur come into this group—a diverse collection. The assumption seems to have been made that these substances directly "irritate" the intestinal mucosa, as a result of which the activity of the gut and, in particular, peristalsis is augmented so as to eject the offending material. The obvious inflammatory changes visible on, say, sigmoidoscopy in any patient with violent bowel activity, from whatever cause, lends support to this theory. However, the question is, which is cause and which the effect?

The manufacturers of the standardized senna preparation ("senokot") used in the two clinical trials quoted go so far as to claim that their product "stimulates bowel peristalsis by the same neuromuscular mechanism that operates in the normal reflex of defaecation. There is no mucosal irritation." Another firm declares of an allied product that it "stimulates peristalsis and the secretions of the colon solely by contact with the mucosa." Search as we may in the literature, we can find no enlightenment on the fundamental mode of action of the so-called irritant purgatives.

The results of a preliminary experiment that we have completed may perhaps be worthy of notice. Ten albino mice each weighing about 20 g. were dosed individually by stomach tube with 180 mg. of standardized senna preparation (senokot) each day, this dose being considerably in excess of that used in medical practice. Fluid intake and diet were regulated. Moderate purgation was obvious within 12 hours. At intervals ranging from 2 to 11 days after the beginning of treatment the mice were sacrificed, post-mortem examinations were made, and sections of the ileum

and colon of each animal were prepared for histological examination. In no case was there seen any macroscopical or microscopical evidence of inflammatory change in the gut.

A similar experiment was next carried out, but five of the mice each received 180 mg. of senna preparation daily and the other five were given 360 mg. each day. Treatment was continued daily for four weeks in all, but the diet otherwise was not controlled. At the end of the experimental period all the mice were sacrificed and examinations made as previously. Again, no evidence whatever was obtained of anatomical "irritation," structural damage, or inflammatory reaction, despite the mild purging that was observed while the animals were under treatment.

There is a *prima facie* case, therefore, for believing that senna at least, and probably other "irritant" purgatives as well, act not so much by direct damage to the mucosa as functionally upon some mechanism of the gut. Pharmacological experiments to elucidate this are in progress. The findings so far are suggestive, but remain to be repeated and confirmed. Details will be published when the experiments are concluded.

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Spontaneous Ulnar Nerve Paresis

When a peripheral nerve lesion in the arm is first noticed after an operation there is a tendency to attribute it to mismanagement in the theatre. The thing speaks for itself—*res ipsa loquitur*—as may be said later. The following three cases of ulnar nerve paresis which developed during in-patient treatment are interesting in that two of the patients did not visit the operating theatre. In these two cases the paresis was noted during recovery from a serious illness; in the third case it was noticed after an operation. In other respects the pareses were essentially similar. Almost complete recovery has taken place in about six months in each case. There was no apparent cause for the ulnar nerve paresis in any of these cases.

CASE REPORTS

Case 1.—A housewife aged 56 was admitted to hospital in April, 1955, suffering from acute cholecystitis which resolved slowly under expectant treatment, including rest in bed and opiates. During the third week in hospital she complained of weakness and paraesthesia in the right hand. Examination revealed a partial ulnar nerve lesion, there being hypoaesthesia and muscular weakness in the hand. The sensory changes affected both ventral and dorsal areas of ulnar distribution, but the flexor carpi ulnaris was not affected. No cause for the paresis could be found, and recovery took place slowly over the next three months. The patient volunteered the information that for many years she has, at intervals, been awakened at night with numbness and tingling in the right ulnar area which passes off on moving the arm into a more comfortable position. Right lateral recumbency is the position which provokes these attacks.

Case 2.—This patient, a married woman aged 45, was a civil servant. She was treated in hospital for extensive second-degree burns of the back by the exposure method. For this she lay prone for a total period of three weeks, frequently putting her arms above her head on the pillow. A fairly large amount of sedation was required. About a week after admission she noticed a weakness in the right hand which examination showed to be an ulnar nerve paresis. Almost complete recovery was noted six months after her discharge from hospital.

Case 3.—A fitter aged 54 was admitted to hospital for a left ureterolithotomy. Two days later he complained of weakness in the left hand which proved to be an ulnar nerve paresis. Careful review of his theatre management failed to

reveal any cause for his paresis. Almost complete recovery was noted six months after his discharge from hospital.

COMMENT

The first patient's experience of waking in the night with the whole or part of an arm paralysed or anaesthetic is a common one. The arm has been uncomfortably placed and a slight move in bed allows it to recover, perhaps with some tingling, and sleep to be resumed. I have questioned about 30 presumably normal people—mostly colleagues and nursing staff—about this, and the comments which follow are based on their answers. The first feature of note is the difficulty in getting accurate information. It is often sound sleep which is disturbed by the phenomenon, it is rapidly resumed, and memory is blurred. Subjects have denied the experience only to come forward a few days later to tell of it. These features make statistical analysis worthless, and none is offered.

Nocturnal episodes of paralysis occur at all ages, and in a very high proportion of people. Only one person questioned had never experienced such episodes; amongst the others the frequency of occurrence varied from once only to as many as five times a night. Few people are troubled more than once a month. Two postures are particularly apt to produce attacks; one is lateral or semiprone recumbency in which it is the under arm that is affected, and the other is supine recumbency with the arm above the head on the pillow. There is considerable variation in the severity of the attacks. In the grossest form encountered there is complete paralysis and anaesthesia of the hand, forearm, and elbow. The victim has to draw the arm from its uncomfortable position with the other hand; recovery then occurs, the last noted abnormality often being paraesthesia in the ulnar side of the hand. Less severe attacks are similar in nature. Over half the people questioned had noticed differential involvement of the ulnar side of the hand; a fair number could definitely draw the line of abnormality down the middle of the ring finger. No one had noticed a differential involvement of the radial side of the hand.

Very few of those questioned were able to offer any explanation of why they woke during the episode—it is not uncomfortable until recovery starts—but there was a general agreement that they woke because of the attack. Some had experienced bad dreams. Occasionally people are woken by the alarm clock, to find their arm affected. This observation suggests that while the attack usually wakes the patient it does not do so with urgency.

The most probable explanation of the attacks is that they are ischaemic, that the posture adopted occludes some vessel, and that the paresis results from the ischaemia. Where there is a definite peripheral nerve distribution, the ischaemia must be presumed to act, at least in part, by causing ischaemia of the nerve itself. In any case there can be little doubt, firstly, that the termination of the attack depends on altering the position in bed, and, secondly, that if an episode remained uninterminated in this way, then in due course damage would result to the nerve, which would be noticed as a paresis for some time subsequently. While the sequence of events leading to waking is undetermined it is very probable that it is in the nature of a defence mechanism involving higher centres, which would be interfered with by narcotics or analgesics.

As already indicated, the frequency of the episodes is not great, but if one considers the possible sequel to a narcotic or sedative having been given on the occasion when the attack occurs, then a temporary paresis is a distinct possibility, and is offered here as the probable explanation of the three cases of ulnar nerve paresis described. Most sedated patients lie supine, but there is a growing tendency for the semiprone (or "tonsil") position to be used because of its advantages to the airway.

Permission to publish the above case reports has kindly been given by members of the surgical staff of the Manchester Royal Infirmary.

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