INTERSTITIAL EMPHYSEMA IN MEASLES

BY

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If judged by the nature and limited number of references to it in the medical literature, the above complication of measles would appear to be very rare. I have therefore been prompted to report the occurrence of three cases at Croydon Isolation Hospital within a period of little over a year.

Case Reports

Case 1.—A girl aged 6 developed measles on January 28, 1949, the rash appearing on February 3. Interstitial emphysema of the chest wall was recognized by the practitioner in attendance on February 5 and admission to hospital was sought. On admission the emphysema was found to be of moderate degree, extending over the greater part of the left side of the chest wall and slightly into the left side of the neck along the line of the trachea. It was not sufficient to cause any obvious swelling of the tissues affected. There was no respiratory distress. Rhonchi were audible over the entire chest, but were of a high-pitched whistling type over the upper part of the left lung. (I believe that the latter phenomenon was in some way related to the source of the emphysema.) There were no other noteworthy features except that the measles rash, which was still well marked on admission, was of the type in which haemorrhagic effusion had occurred into the skin lesions—not in itself an uncommon phenomenon. The patient had a troublesome though not exceptionally severe cough. The emphysema was maximal on admission and thereafter absorbed rapidly so as to become imperceptible within four days. Other chest signs and symptoms cleared up with similar rapidity. Sulphadimidine by mouth was the only treatment given.

Case 2.—A boy aged 5½ was admitted late at night on March 20, 1949, as an emergency case because of respiratory distress. The illness began on March 14 with the usual catarrhal symptoms. The measles rash developed on the 18th. There was a history of persistent cough following influenza in the previous December. On admission a well-marked interstitial emphysema extended over both sides of the chest wall and upwards through the neck into the tissues of the cheeks. There was obvious swelling of the neck and face, especially on the left side. A moderate degree of dyspnoea and slight cyanosis were present. The patient had a cough which was not unduly troublesome, and scattered rales were heard throughout the chest. A fading morbilliform rash and measles stomatitis conformed to the stage of the illness. Besides giving sulphonamides, treatment in an oxygen tent was considered advisable. As in Case 1, there was immediate and rapid improvement. The oxygen was discontinued the next morning, and five days later no detectable emphysema was present. Except for the subsequent development of tonsillitis and otitis media, recovery was uneventful.

Case 3.—A boy aged 5½ was admitted on February 17, 1950, as a case of measles and pneumonia. The measles began on February 7, the rash appearing on the 11th. On admission the skin still showed the staining of a morbilliform rash and the mouth was still inflamed. Interstitial emphysema extended over the entire left side of the chest and upwards on each side of the trachea. The patient had an irritable cough and a cyanotic tinge, but was not dyspnoeic. I could not definitely determine whether there were any adventitious sounds apart from those arising from the emphysema. Again the subcutaneous emphysema cleared up rapidly from the time of admission and was fully absorbed by February 23. Rales were audible in the left base until March 2 and may have been related to the emphysema-producing lesion. An x-ray photograph of the chest showed no further abnormality apart from the appearance of air in the chest wall.

Discussion

Some textbooks on infectious diseases make no reference to interstitial, subcutaneous, or surgical emphysema as a complication of measles, while those that do refer to it treat it as a very rare complication.

Goodall (1928) states: "As very rare complications [of measles] I have met with cases of nephritis, endo- and peri-carditis, parotid abscess, cellulitis with or without sloughing, and extensive (surgical) emphysema of the trunk and neck. (One case—the patient a boy of 6 years—had bronchitis with severe attacks of coughing: he recovered.)"

Rolleston and Ronaldson (1939) state: "Subcutaneous emphysema is another infrequent occurrence. It may be associated with bronchopneumonia or develop apart from any gross pulmonary lesion, when it has been attributed to rupture of the alveoli, bronchi, or larynx as the result of violent coughing. Ronaldson (1927), in reporting two cases in which cough was not a feature, suggested that weakening of the alveolar wall by toxic action rather than by over-distension was the usual explanation of this occurrence in measles."

Price (1947) states: "Rarely, mediastinal and subcutaneous emphysema have occurred."

In none of my cases was the cough particularly severe, which would confirm Ronaldson's suggestion that the cough can produce this effect only when combined with some pathological process in the respiratory tract. In this connexion, I think that the description of "autemphysema" in the *British Medical Journal* of January 14 and February 4, 1950 (pp. 105, 308), is suggestive. It is described how surgical emphysema is self-induced by malingerers by wounding the buccal mucosa and strongly distending the cheeks with air.

Measles has a well-known capacity for producing ulceration of mucous membranes, and such ulcers could act similarly to the self-induced trauma of the malingerer. I feel that some such discrete ulceration in the respiratory tract is a more probable source of air entry than a weakening of the alveolar walls by toxic action as suggested by Ronaldson. Cases in which measles toxæmia damaged the alveolar walls might be expected to produce more general evidence of lung involvement than was present in the cases I have described. The involvement of the chest wall in each case would indicate that the air did escape from some part of the respiratory tract within the lung, the subsequent spread being via the root of the lung and mediastinum to the tissues of the chest wall and neck, extending in one case to the face.

In the one case in which the face was involved, I think that origin from the buccal mucosa can be excluded. There was no evident lesion in the mouth. Also, in the description of cases produced by mouth injuries to which I have referred, the chest wall is not mentioned as being involved, though there was marked generalized swelling of the face and neck. I think, therefore, that in my three cases the causative lesion was intrapulmonary, probably a small ulcer in one of the larger bronchi. A common feature in all three cases was that the amount of air in the tissues rapidly reached a maximum and thereafter was progressively absorbed. This also would favour the idea of a single small lesion capable of sealing up quickly. So far as it is permissible to draw any generalized conclusion from three cases, it would seem that a favourable prognosis can be given even when the initial condition is somewhat alarming.

REFERENCES

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Medical Memoranda

Contamination of Caesarean Wounds by Vernix Caseosa

The vernix caseosa, though one of the potential contaminants of the operative field in the caesarean operation, has for various reasons received as such little or no attention. Some of the neglect has been due to a belief that because the vernix is sterile it is therefore innocuous, some to a proper preoccupation with the problems of contamination by pyogenic organisms. That the vernix is not, however, an entirely innocuous contaminant is illustrated by the events that follow the rupture of an ovarian dermoid cyst. Such an accident, whether spontaneous, puerperal, or operative, is succeeded quite rapidly by a severe peritoneal reaction, and this usually in turn by a secondary invasion of the peritoneum by pyogenic organisms. Even a small amount of free sebaceous material may determine the reaction; in a recent case a severe lower abdominal peritonitis followed the rupture of a cyst no larger than a pullet's egg.

If the dermoid cyst that bursts is large, or if at operation much of the contents escape into the peritoneal cavity, a diffuse plastic peritonitis is certain unless a thorough cleansing is carried out at once. If any pockets of sebaceous material are left behind, then at the best the accident may be followed by the development of localized residual abscesses here and there throughout the peritoneal cavity, even under the diaphragm. Indeed, it may fairly be stated that a dermoid cyst is the one ovarian tumour which must not be tapped at operation.

Both the vernix caseosa and the contents of dermoid cysts, allied as they are in constitution, have similar properties as contaminants; both are unabsorbable and can be removed from the operation area only by a slow phagocytosis, a process carrying the certainty of adhesions and the chances of secondary infection. It seems likely that the escape of vernix into the peritoneal cavity or its inclusion in the abdominal incision is a potential cause of caesarean morbidity. This may be one of the reasons why caesarean section wounds seem more apt than others to heal by second intention; after a period of apparent healing by first intention pus is discharged from their lower end. On seeing an ugly caesarean scar it must be a common experience to draw the inference that the uterine scar must be equally unsound, an inference indeed almost expected from an examination candidate, yet to find at the repeat section that the secondary scar tissue is confined to the abdominal wall, the uterine scar itself being quite sound.

The amount of vernix soiling the operative field varies a great deal. For one thing the amount found covering the foetus varies for reasons that are as yet not understood. Again, the method by which the child is extracted has a bearing on the amount escaping into the wound. More escapes when the child is delivered by the breech or when

any difficulty arises in the extraction. Large flakes escaping into the operative field are of course readily seen and removed. It is, however, important to look for and remove the smallest fragments and to recognize the potential contamination coming from the vernix which adheres to the gloves and swabs. Inspection of the surface of water used for rinsing the hands or the swabs will often show a film of liquefied vernix. All these are sources of contamination.

Once the importance of vernix spill is appreciated, suitable precautions come readily to mind. Packing off the peritoneal cavity before the uterus is opened is not a real protection, for once the foetus is extracted these packs always lie loose. "Tetra" cloths as usually employed do not protect the deep layers of the incision. The real protection is twofold; (1) a change of gloves, swabs, and rinsing water immediately the uterine incision is closed and before the peritoneum covering the lower segment is sutured; and (2) a meticulous removal of every fragment of the vernix caseosa from the operative field, the most likely places being the front of the broad ligaments and the lower angle of the abdominal incision behind the rectus sheath. If a transverse abdominal incision is used a careful inspection of the recess behind the lower flap of the sheath should be made. It is well, too, to wipe all the raw surfaces with a clean swab before any attempt at closure.

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Pethidine as an Adjunct to Nitrous Oxide and Oxygen Anaesthesia

Mushin and Rendell-Baker, in 1949, published a memorandum on the use of pethidine as a supplement to nitrous oxide and oxygen anaesthesia, a method which they had seen used successfully in America and had themselves adopted on 100 occasions. It appeared that this method might well be a useful innovation, and such has proved to be the case in the 150 administrations here recorded.

METHOD

The method suggested by Mushin and Rendell-Baker was adopted and has been adhered to throughout this series, although they themselves have on occasion given increased doses of pethidine.

The routine in the present series has been as follows: (1) Premedication has been by "omnupon" 1/3 gr. (22 mg.) and scopolamine 1/150 gr. (0.43 mg.) for all adults between the ages of 17 and 60 years; over 60 years, omnupon 1/6 gr. (11 mg.) and scopolamine 1/300 gr. (0.21 mg.). (2) Induction has been by a "sleep" dose of sodium thiopentone—usually between 0.2 and 0.3 g. is given in 5% solution. (3) Anaesthesia is begun with nitrous oxide and oxygen. (4) Pethidine is given in a dose of 25 mg. intravenously as soon as anaesthesia is smoothly established. (5) Tubarine or "flaxedil" is given at the requisite time should there be any undue movements of the patient or should great relaxation of the muscles be necessary.

This routine has been the standard in all cases except three. Of these, two were tough farm-labourers, and each had to have 0.5 g. of sodium thiopentone followed by 37.5 mg. of pethidine. The third was a girl who received 0.5 g. of sodium thiopentone originally, as she was to be examined under an anaesthetic before operation was decided upon.