Sputum retention was rarely observed, and no useful information could be deduced from the small number investigated in this series.

The character of the chest pain too afforded no help in detecting cases of bronchial obstruction.

The first of the examination findings—stridor—was equivocal and bore no clear relation to the presence of bronchial obstruction. Some patients with generalized bronchitis, and even one in whom there was no known abnormality, could produce this sign. As interpreted by us, the sign was of no value in detecting bronchial obstruction. On the other hand, a local reduction of breath sounds was an important finding. Investigation of the further five group B cases showed that in four the lesion was on the same side as the sign (in one of these a bronchogram showed confirmatory narrowing of a segmental bronchus). The fifth group B case suffered from secondary deposits in both lungs, and our finding is unexplained. This sign, therefore, though present in less than half of the cases of bronchial obstruction, was a good pointer to this condition in the absence of fluid.

Localized rhonchi were found in 26 of the group A cases. As in the case of a wheeze, this sign was accentuated in more than half by lying on one side. Accentuation and, indeed, discovery of localized rhonchi by forced expiration were also notable findings. In 20 of the 26 cases forced expiration made this sign more obvious. One observer (T. M. W.) noted, further, that in nine cases forced expiration was necessary for the discovery of the sign, whereas in the remaining 11 forced expiration accentuated a sign already present. These localized rhonchi were in most instances maximal over the anterior chest at the level of the hilum. They were ascribed to the correct side in all but one case. Attempts to define involvement of one particular lobe were unsuccessful. Both partial and complete bronchial obstruction were apparently not related to the incidence of this sign. Thus localized rhonchi were found in 14 of the 35 cases of complete block (40%) and in 51 of the 115 cases of partial block (44%). Likewise this sign was not related to the cause of the stenosis, since the distribution between cases of tuberculosis and carcinoma showed little difference. Localized rhonchi were also found in 12 of the group B cases and probably arose from distal bronchial obstruction. The only evidence in support of this was the site of the principal lesion as shown by x-ray examination; in 10 the clinical and x-ray findings agreed; the last two cases suffered from bilateral pulmonary tuberculosis.

It is interesting to compare the symptom localized wheeze with the finding of localized rhonchi. Both were present together in only 12 patients. At first sight this low figure seems surprising. When it is remembered that both are intermittent and may vary from day to day, it is understandable that in only a small proportion will the symptom and sign be simultaneously present.

Conclusion

Two symptoms and two signs have proved of some value in assessing bronchial obstruction—a cough which has altered in character, a localized wheeze, a local reduction of breath sounds, and localized rhonchi. The effect of change of posture in accentuating localized wheezing and rhonchi has been confirmed. However, these symptoms and signs were inconstant, and even the most frequent localized rhonchi were noted in just over half the cases of proved bronchial obstruction. Nevertheless we would suggest that these symptoms and signs are important enough to be included in routine clinical assessment of the chest, and their presence, especially localized wheezing and localized rhonchi, should call for further investigation.

Summary

One hundred patients (50 with bronchial obstruction and 50 in whom bronchoscopy was negative) have been studied independently by three observers to assess the diagnostic value of certain symptoms and signs.

A cough which has altered in character, a localized wheeze, a local reduction of breath sounds, and localized rhonchi were more commonly found among patients with bronchial obstruction.

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Medical Memoranda

A Case of Acute Hydramnios

Acute hydramnios is a very rare condition, and, according to Browne (1946), every case should be reported.

CASE REPORT

The patient was an Ibo woman, aged about 37, who reported to the antenatal clinic at the Maternity Hospital, Aba, Nigeria, complaining of severe abdominal pain and distension. She said she was seven months pregnant. She had had eight previous pregnancies, the first ending in abortion at three months, but the remainder went to term and ended in apparently normal deliveries of normal babies—six female and one male. All her confinements were at home, and she had never attended hospital in her life before; but her condition on this occasion was so serious that she felt her life was in danger, and decided, after a certain amount of consideration, to go to the hospital.

Her pregnancy had apparently proceeded without incident until the evening before her attendance, when she felt that her abdomen was becoming acutely distended. The swelling continued throughout the night and also while on her way to hospital the following morning. She had severe pain all over the abdomen and was vomiting. She also complained of shortness of breath.

On examination she was seen to be of average build but rather thin. There was no pallor of the conjunctivae or any sign of shock. Her pulse rate was 84, regular, and of good volume. The temperature was normal. There was oedema of the lower limbs, which she stated had been present since the previous day. There was also oedema of the abdominal wall below the level of the umbilicus. The blood pressure was 120/80, and a catheter specimen of the urine contained a trace of albumin but was otherwise normal. Orthopnoea was present, but examination of the chest revealed no lesion of the heart or lungs to account for this. The abdomen was greatly distended and the skin over it shining and tense. Coils of small intestine could be seen outlined in the flanks when they were pressed against the thin abdominal wall by the tumour within. On palpation it was difficult to outline the uterus owing to the tenderness present, but the fundus was at the level of the xiphisternum and there was marked tenderness over the liver where it was being squeezed against the diaphragm. A fluid thrill could be readily detected, but no foetal parts

were felt and no foetal heart was heard. The uterus was contracting every 10 minutes. On vaginal examination the cervix was high up and admitted two fingers. The membranes were tense and bulging, and a foetal head was ballotted without difficulty.

The patient was admitted to the ward and given pethidine hydrochloride, 100 mg. intramuscularly. After routine preparation she was taken to the theatre and the membranes ruptured. Nine pints (5 litres) of fluid was removed, the symptoms being almost immediately relieved. On examination the abdomen was still slightly tender, the uterus had contracted down on to the foetus, and a foetal heart could then be heard. An abdominal binder was applied, but the patient objected to this and it was removed after an hour. She went into labour and was delivered 12 hours later quite normally. There was no post-partum haemorrhage. The patient felt so well that it was with difficulty that she was persuaded to stay in hospital for three days. When she left her condition was good and there was no albuminuria or oedema. I have not seen her since.

The foetus was a stillborn male, and, as in most of these cases, it was abnormal. It was estimated to be about eight weeks premature (the patient gave a history of seven months' amenorrhoea, but this discrepancy is common in our practice here), it had a rather cretinous appearance, it had no ears except for small rudimentary knobs of skin, the external auditory meatus was absent on both sides, and the right foot was clubbed. No other congenital abnormalities were detected, and the oesophagus was patent.

My thanks are due to the Inspector-General of Medical Services, Nigeria, for permission to publish.

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Fatal Case of Agranulocytosis following Phenylbutazone

The following case is of interest because of: (1) the small dosage and duration of phenylbutazone administration (14 g. over 27 days); (2) the appearance of a rash six days after cessation of treatment; (3) the long interval between the cessation of the drug and the recovery of marrow function (19 days); and (4) the tragic consequence of the administration (even though every precaution was taken) of a toxic drug, instead of an inert one, as a placebo to a patient whose symptomatology was largely functional.

CASE REPORT

A woman aged 49 was admitted to hospital on June 22, 1953. She had complained of "arthritis" for many years, and for the preceding two years had been under treatment by the general practitioner who had sent her to hospital. During the two years' treatment he had tried most of the common symptomatic drugs—phenobarbitone, aspirin, codein. co., etc., and her husband had spent many hours massaging her various aching areas, chiefly back, lower chest, and thighs. Periodically she had complained of pain and stiffness and swelling of her fingers, knuckle-joints, and the small joints of her feet. She had begged for the "new treatment" of which she had heard. In view of reports of toxic effects her doctor had given way with misgivings, and after a blood examination on May 14 (haemoglobin, 95%; white cells, 8,400; differential count, "within normal limits"; E.S.R. (Westergren), 18 mm. in one hour, phenylbutazone ("butazolidin"), 200 mg. three times a day, was started on May 19, and she was advised to stop taking her sleeping-tablets—sodium amytal, 3 gr. (0.2 g.) nocte.

On May 28 her haemoglobin was 96%, and the white-cell count 5,200, the differential count being "within normal limits." By June 4 she had noticed no improvement, although she had not taken the tablets regularly. She was

advised to continue with them a little longer, and then to stop if they did not help. On June 14, for that reason and because she felt somewhat nauseated, she stopped taking them. On the 18th sodium amytal was again taken because she had not been sleeping well, and on the 20th she was covered with a rash. This was reported to her doctor the next day (Sunday), when he made sure that she had stopped the tablets, and arranged for a blood test next morning. This revealed haemoglobin, 81%; white-cell count, 1,600 (polymorphs 2%, small lymphocytes 92%, large lymphocytes 4%, monocytes 1%, Türk cells 1%); E.S.R., 20 mm. in one hour (Westergren). She was immediately sent to hospital.

Examination showed her to be an anxious alert woman, with a temperature of 101° F. (38.3° C.), a generalized maculo-papular rash, a slightly injected throat, and no clinical evidence of arthritis. Crystalline penicillin, 1,000,000 units intramuscularly three-hourly; pyridoxine, 50 mg. intramuscularly daily; liver extract ("livogen"), 2 dr. (7 ml.) three times daily; and vitamin-B complex, one tablet three times daily orally, were given. Sodium amytal, 3 gr. (0.2 g.) nightly, was prescribed as a sedative, to be taken as desired.

Clinically she was progressing well on June 26, the rash had nearly faded, and the white-cell count was 4,000 (polymorphs 1%). Next day the white-cell count was 4,200 (polymorphs 2%, small lymphocytes 93%, large lymphocytes 5%); platelets 150,000. On the 28th her temperature, which had been averaging 100° F. (37.8° C.), was elevated to 104° F. (40° C.). She had a cough and complained of pain in the left side of her chest; there were signs of consolidation at the left base. Oxytetracycline ("terramycin"), 750 mg. six-hourly, and oral vitamin K, 10 mg. twice daily, were added to the previous treatment. The white-cell count was 1,600 (small lymphocytes 98%, large lymphocytes 2%, no polymorphs). On June 30 she was delirious and irrational. The white-cell count was 1,000 (small lymphocytes 96%, large lymphocytes 4%, no polymorphs). Next day there was scattered consolidation over both lungs; she was dehydrated, and it was necessary to begin intravenous therapy cautiously.

A myelogram revealed: Neutrophils—polymorphs 0, metamyelocytes 6%, myelocytes 43%, promyelocytes 3%; blast cells, 2%; lymphocytes, 25%; monocytes, 2%; megakaryocytes, 1%; nucleated red cells and normoblasts, early 2%, intermediate 15%, late 1%. The myeloid-erythroid ratio was 1.5 : 1 (normal 8 : 1 to 2 : 1), the leuco-erythrogenic ratio was 1.5 : 1 (normal 1.5 : 1 to 2.5 : 1). *Interpretation:* This is a cellular marrow in which there is evidence of active erythropoiesis and leucopoiesis. The granular cells are all immature.

The white blood cell count was 800; no polymorphs seen. On July 2 the white-cell count was 600 (polymorphs 1%). Next day her temperature, which for the previous five days had remained between 103 and 104° F. (39.4 and 40° C.), had dropped to 100° F. (37.8° C.), and the white-cell count was 4,300 (polymorphs 67%, small lymphocytes 28%, large lymphocytes 1%, monocytes 1%, eosinophils 1%, metamyelocytes 2%). However, she was still very confused mentally, and was cyanotic; there was evidence of diffuse bronchopneumonia.

On July 4 the white-cell count was 11,200 (polymorphs 74%, metamyelocytes 4%) and her temperature was within normal limits, but she died early the next morning.

Post-mortem Examination.—The chief findings were:—Respiratory: some adhesions in the left pleural sac; some frothy mucus, slightly purulent, in the air passages, chiefly the right bronchus. Lungs: multiple small abscesses and hypostatic oedema. Spleen: enlarged (230 g.), soft, diffuent. Liver: a few small abscesses. In other words, findings of septicaemia.

I am indebted to Dr. J. Fine for his pathological reports, and to Dr. E. Grahame Jones, under whose care the patient was admitted, for permission to publish the case.

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