as those which occur in children as a result of infection with poliomyelitis virus. These findings are similar to those already reported from America by Salk (1956) and Brown and Smith (1956).

It should be noted, however, that during the period between the primary course of immunization and the third injection a decline in antibody level occurred which was especially marked in the case of types 1 and 3, resulting in a high proportion of children with titres of 4 or less at the time of the third dose. In some of these children the response to the third dose of vaccine was poor, especially with type 1, and it is proposed to study, with those children in the original group who did not receive the third injection, the question of whether the chance of a poor response is increased the longer one waits before giving a third dose.

It is also intended to follow up those children who have already received the third dose, to determine to what extent their antibody levels are maintained over the next few years.

Summary

A study was made in a group of 100 children who initially had no detectable poliomyelitis antibodies, to determine the decline in antibody level after immunization with two doses of poliomyelitis vaccine, and the response to a third dose of vaccine given eight to eleven months after the second dose.

The decline in antibody level was greatest with types 1 and 3, resulting in a high proportion of children with low titres at the time of the third injection.

In the majority of children a substantial antibody response to all three types was obtained to the third injection of vaccine.

Those responsible for this study are grateful to the parents who consented to allow their children to take part and also thank the Ministry of Health for the supply of poliomyelitis vaccine.

REFERENCES

- Biological Standards Control Laboratory (1957). *Brit. med. J.*, 2, 124.
Brown, G. C., and Smith, D. C. (1956). *J. Amer. med. Ass.*, 161, 399.
Medical Research Council (1957). *Brit. med. J.*, 1, 366.
Salk, J. E. (1956). *J. Amer. med. Ass.*, 162, 1451.

UNHERALDED PULMONARY EMBOLISM

BY

H. COHEN, M.B., M.R.C.P.

Senior Medical Registrar, Royal Hospital, Sheffield

AND

J. J. DALY, M.B., M.R.C.P.

Medical Registrar, Royal Hospital, Sheffield

Pulmonary embolism is a common disease which accounts for 2 to 3% of all deaths in hospital (Brooks, 1952). It is suspected when a patient has acute chest symptoms either post-operatively or after prolonged bed rest. It can, however, occur in patients who appear to be fit, are active, and have no signs of venous thrombosis (Hampton *et al.*, 1945), and in these patients diagnosis is difficult, as the clinical picture frequently resembles pneumonia.

The object of this report is to describe 10 cases, eight of which had unheralded pulmonary embolism as described above. The remaining two patients had no signs of venous thrombosis when first seen, but had been at rest in bed for about a week before they developed chest symptoms. Such a short period of bed rest is not generally associated with venous thrombosis.

Case 1

A woman aged 21, who worked as a machinist, was admitted to hospital on November 23, 1955, with a history of left-sided chest pain for eight days. One month previously she had a similar pain on the right side of her chest for three days. Fever was present on both occasions and there was no history of haemoptysis. A day before admission she complained of a dry cough. Her weight had been steady. At the onset of the last attack penicillin was given. She had been working between the two episodes of pleurisy. Her past health had been good and there was no history of chest illness in the family.

Physical examination revealed a pale girl, not distressed, with a temperature of 100° F. (37.8° C.). There was no evidence of phlebitis in her legs. Examination of the chest disclosed signs of collapse at the left base. The cardiovascular system appeared normal with a blood pressure of 110/80. Treatment was started with analgesics and intramuscular penicillin.

Investigations.—Chest x-ray examination showed elevated left leaf of diaphragm. Blood count: Haemoglobin, 50%; red cells, 4,290,000 per c.mm.; white cells, 7,000 per c.mm. (normal differential); E.S.R. (Westergren), 98 mm. in one hour. Mantoux positive at 1/10,000. Specimens of gastric juice were collected for examination for tubercle bacilli.

The pain subsided but the fever continued. On November 27 there were signs of a left pleural effusion. The fluid was turbid, contained polymorphs and lymphocytes, and culture was sterile. Streptomycin, 5 g. twice daily, and isoniazid, 200 mg. twice daily, in divided doses, were substituted for penicillin. On December 3 she called for a bed-pan, complained of feeling faint, collapsed, and died.

Necropsy Findings.—There was a left pleural effusion of 1,200 ml. This was yellow but mixed with blood. Straddling the bifurcation of the pulmonary artery was a coiled mass of ante-mortem thrombus showing branching. Further down the right pulmonary artery a small thrombus was noted adherent to the vessel wall. A yellowish-brown firmly adherent clot was seen in the left pulmonary artery. Both were associated with established infarcts. Extensive dissection of the leg veins revealed no ante-mortem thrombus.

Comment.—This case illustrates several important features. The patient had been active before the onset of her symptoms. Between the episodes of pleurisy she was working. At no time was there any clinical evidence of phlebitis. Her cough had never been productive and there appeared to be no response to antibiotics. At necropsy the appearance of the ante-mortem clot suggested an embolus, and there was no evidence of thrombosis in the calf veins.

Case 2

A miner aged 54 was admitted to hospital on May 5, 1955, with a history of intermittent chest pain for one year. The first attack was right-sided and lasted three days. He then remained well until March, 1956, when one night he awoke with severe retrosternal pain radiating to the left and associated with breathlessness and tightness in his chest. Three weeks later he experienced pleural pain on the left side. The next incident, 11 days later, was right-sided and accompanied by haemoptysis. Two days before admission he experienced right-sided chest pain, again with haemoptysis. During the five weeks before admission he was breathless on moderate exertion. At no time was cough a prominent feature. During each illness he was febrile, and with the last three attacks he had been given penicillin, oxytetracycline, and streptomycin. There was no family history of chest illness, and his general health in the past had been good.

Examination revealed an ill, cyanosed, breathless man, in pain and with a temperature of 101° F. (38.3° C.). Movement was diminished and rales were heard over both lung bases. There was no evidence of heart failure and the blood pressure was 120/70. Examination of his legs revealed no evidence of thrombophlebitis.

Investigations.—Chest x-ray report (Dr. N. A. Lewtas) was as follows: "Lung fields: There are bilateral pleural effusions. There is an opacity lying peripherally in the left mid-zone which does not appear to be of segmental distribution and is very suggestive of an infarct." The E.C.G. was normal. Blood count: Haemoglobin, 96%; white cells, 12,000 per c.mm., with normal differential; E.S.R. (Westergren), 90 mm. in one hour.

Treatment was started with anticoagulants, penicillin, and streptomycin. The patient improved over the course of a week and became afebrile. Prior to discharge on June 15 he was up and about and symptom-free. Chest x-ray examination at that time revealed a small residual shadow at the left costophrenic angle.

Comment.—This patient had five acute attacks of chest pain in a year. Between attacks he was well apart from breathlessness on moderate exertion just before admission. He had never had clinical evidence of venous thrombosis. His sputum was not purulent on any occasion. There appeared to be no response to antibiotics, and it was not until he was given anticoagulants that his condition improved and no further attacks occurred.

Case 3

A man aged 76 was admitted to hospital on May 30, 1955, complaining of sudden onset of abdominal pain 12 hours previously. The pain was generalized, but was more severe in the left loin and worse on breathing. Two weeks previously he had experienced a similar attack. Prior to this his health had been good.

Physical examination revealed an ill man with a temperature of 100° F. (37.8° C.). Moist sounds were present at both bases. The blood pressure was 190/90. There were no abnormal signs in his abdomen and no evidence of venous thrombosis in his legs. Pleurisy was diagnosed and the patient was treated with penicillin and sulphadimidine. X-ray examination of the chest showed shadowing at both bases and evidence of an effusion in the region of the horizontal fissure.

The patient continued to experience pain, which had spread to his left shoulder. An intermittent fever was present. On June 13 he collapsed and died.

Necropsy Findings.—There were fibrinous adhesions over the lower lobe of the right lung. Both pulmonary arteries contained coiled pieces of ante-mortem thrombi. An infarct was present in the upper part of the right middle lobe. There was congestion at both bases. No evidence of thrombosis was found in the legs.

Comment.—This patient's illness consisted of two attacks of pleurisy associated with persistent fever. Abdominal pain was the presenting feature. There was no response to chemotherapy. No evidence of venous thrombosis was found at necropsy.

Case 4

A man aged 43 was admitted to the Royal Infirmary, Sheffield, on April 30, 1952. He had been well until two weeks previously, when pain developed in the left side of his chest and in his left loin. He had recurrent attacks of pain and haemoptysis during these two weeks. The past history was not recorded.

Physical examination revealed that he was shocked. B.P. 90/70. There were no signs of heart failure. He was febrile—temperature 100° F. (37.8° C.). The breath sounds were diminished at the left base. There was no tenderness of the calves or swelling of the legs. An E.C.G. showed a Q wave and an inverted T wave in standard lead III. He collapsed and died shortly after admission.

Necropsy Findings.—The right ventricle was dilated. At the bifurcation of the pulmonary artery, and extending for a short distance into the right branch and for a long way into the left branch and its subdivisions, lay a greyish-red, firm, coiled pulmonary embolus completely occluding the vascular lumen. There was ante-mortem thrombus in the

left femoral vein. At the base of the left lung there was a dark-red, friable haemorrhagic infarct. Both pleural cavities appeared normal.

Comment.—The clinical picture, consisting of peripheral circulatory failure, chest pain, and an electrocardiographic abnormality in a man of 43 who was active until the onset of his illness, was suggestive of coronary thrombosis, and quite unlike that usually associated with pulmonary embolism.

Case 5

A male clerk aged 56, previously fit, developed pain in his left groin, radiating to the right groin, and shooting down his right leg. His left testicle became swollen, but not his legs. He was admitted to the surgical wards of another hospital on July 20, 1956, and a diagnosis of acute epididymo-orchitis was made. *Staphylococcus aureus* was grown on culture of his urine, and he was treated with chloramphenicol. He remained in bed in hospital for one week and was discharged on July 28, the pain and testicular swelling having gone. However, treatment with chloramphenicol was continued. At home, he stayed in bed for about half the day and for the remainder rested in an armchair. Four days after discharge from hospital he developed a severe pleural pain in the left side of his chest. The onset was not dramatically sudden: it was about four hours before the pain became severe. He had a slight cough and produced a small quantity of tenacious blood-stained sputum. He was seen by his general practitioner, who diagnosed pneumonia and treated him with oxytetracycline. He improved, but four days later had another attack of pain in the left side of his chest, this time associated with breathlessness and signs of consolidation in the left lower lobe. Following this attack the antibiotic was changed to erythromycin and he again improved. Three days later he had an attack of pleural pain in his right side: he became breathless, cyanosed, and collapsed, and was admitted to hospital on August 9.

Inquiry revealed that four years previously he had had an operation on varicose veins in his left thigh. There was no history of previous respiratory disease or other illness.

Physical examination on admission revealed a thin cyanosed man whose breathing was rapid, shallow, and painful. He was febrile—temperature 101° F. (38.3° C.). The pulse was regular, rapid (130 a minute), and the blood pressure 160/110. Jugular venous pressure was raised about 2 cm. above the sternal angle. There was no oedema of the legs or sacral pad. The heart was not clinically enlarged, but there was a sternal heave. The pulmonary second sound was split. There were no murmurs. Dullness to percussion was present over both lung bases with crepitations in these areas and bronchial breathing over the right lower lobe posteriorly between the eighth and tenth ribs. The legs were not tender and there were no visibly inflamed veins. Homan's sign was negative. There were no other abnormal physical signs.

He was treated with phenindione, penicillin, 500,000 units six-hourly, and streptomycin, 0.5 g. twice daily.

Investigations.—Chest x-ray examination (August 10) showed bilateral pleural effusions and patchy inflammatory changes at both bases. The final x-ray examination, on August 21, showed, in the left lateral film, fluid or pleural thickening along the posterior chest wall and linear atelectasis just above the diaphragm. The conclusion was that the appearances could be due to embolism, inflammatory change, or possibly pleural thickening. Blood count: Haemoglobin, 92% (14.1 g./100 ml.); red cells, 5,030,000; white cells, 11,000 (polymorphs 66%, lymphocytes 30%, monocytes 4%); E.S.R. (Westergren) 92 mm. in one hour. E.C.G. showed no abnormality other than tachycardia and no evidence of acute cor pulmonale.

The patient improved greatly, but 10 days after admission, while still on anticoagulants, he developed thrombophlebitis of the left femoral vein in the femoral canal. The left leg became swollen, tender, and painful. Anticoagulant therapy was continued for four weeks, by which time his

leg had returned to normal except for a little oedema round the ankle. Before he was discharged from hospital he had recovered and showed no signs of pulmonary hypertension.

Comment.—Unlike the others, this patient was not active up to the time when he first had chest symptoms. Thrombosis of a pelvic vein may have followed epididymo-orchitis. Alternatively, epididymo-orchitis might have been mimicked by thrombophlebitis in the pampiniform plexus. The history of recurrent attacks of pleurisy not responding to treatment with antibiotics was characteristic of pulmonary embolism. In this case signs of pulmonary hypertension were an aid to diagnosis.

Case 6

A storekeeper aged 59 was in good health and at work when he had an attack of shivering. He had a non-productive cough, with a fever, and went to bed for eight days. He was active but convalescent, when, almost three weeks after the start of his first illness, he had a sudden severe stabbing pain in the left side of his chest posteriorly. He was breathless, and the pain was worse on coughing or deep breathing. He was admitted to hospital on March 3, 1952, two days after the onset of this pleuritic pain. There were no other symptoms, and, apart from bronchopneumonia in 1949, no past illness of note.

Physical examination revealed that he was breathless at rest but not cyanosed. He was febrile—temperature 100.4° F. (38° C.). He had clubbing of the fingers and toes. Chest expansion was poor and there were signs of consolidation at the left base. There were no signs of heart failure or of peripheral venous thrombosis. The blood pressure was 135/90.

Lobar pneumonia was diagnosed and penicillin, 240,000 units six-hourly, was given. On the day after admission to hospital he had a haemoptysis, and this persisted for four days. He was improving generally and the signs of consolidation were diminishing, when on March 10 he had a recurrence of pain in the left side of his chest. He improved again, becoming afebrile, but on March 22 his temperature rose.

Treatment with penicillin was stopped, and instead he was given chloramphenicol, 0.5 g. six-hourly. On March 23 he developed pleuritic pain in the right side of his chest and there were rales at both lung bases.

It was not until March 31 that he had pain in his right leg, with some swelling, but still without tenderness. Anticoagulant therapy was started on that day and he slowly improved, but still had swelling of his right leg on discharge from hospital.

Investigations.—The sputum was mucoid and no pathogen was grown from it. Chest x-ray films, which were repeated, showed shadowing (inflammatory changes) at both lung bases, with, on April 4, a shadow at the left base suggestive of segmental collapse. Blood count on March 5 showed: Hb, 83%; white cells 8,000, with 65% polymorphs. E.S.R. (Westergren), 47 mm. in one hour.

Comment.—Again in this case the symptoms and signs of pulmonary embolism appeared well before the signs of venous thrombosis.

In the cases so far described a definite diagnosis could be made because of the post-mortem findings, the radiological findings, or the later development of venous thrombosis. In the following four cases the diagnosis could not be proved, but the clinical picture presented by each patient was that which we have come to associate with pulmonary embolism and each recovered after treatment with anticoagulants and antibiotics.

Case 7

A sales manager aged 36 was well and at work, when he developed a "head cold." The next day he had pleural pain in the left side of his chest and produced grey sputum streaked with blood. He went to bed and was treated by his general practitioner with sulphonamides and penicillin.

The haemoptysis continued. Six days after the start of his illness he had pleural pain in the right side of his chest. There was a past history of a football injury to his right leg with subsequent swelling and tenderness in the calf. This had occurred 10 years previously and he had been well since.

He was admitted to hospital on December 17, 1956, nine days after the start of his illness. He was afebrile. Physical examination revealed a pleural rub at the right lung base and signs of collapse at the left base. There were no signs of peripheral venous thrombosis. X-ray examination showed a small effusion in the left costophrenic angle. No pathogen was grown from the sputum, which contained blood for the first week that he was in hospital. The E.C.G. was normal. He was treated with anticoagulants and penicillin and made a rapid recovery.

Comment.—The occurrence of a second attack of pleurisy while the patient was being treated with penicillin and the persistent haemoptysis suggested a diagnosis of pulmonary embolism, possibly from the site of old thrombophlebitis.

Case 8

A mine-worker aged 53 was recovering from an operation for arthrodesis of his right hip performed six months previously for osteoarthritis. He was well and had been walking one to two miles a day for over a month. Two days before admission to hospital on February 23, 1957, he had a sudden sharp pain in the right side of his chest. He had difficulty in breathing because of the pain, which was pleural in character. He had no cough or haemoptysis. Six months previously whilst in hospital following his operation he had two similar attacks of pleural pain. Except for a history of injury to his right hip there was nothing of note in the past history.

Physical examination revealed that he was hypertensive (blood pressure 145/105). There were no signs of heart failure. The percussion note was impaired at the right lung base, in which area the breath sounds were diminished and there were a few crepitations. There was no evidence of peripheral venous thrombosis. He was febrile on admission, temperature 100.5° F. (38.1° C.). He produced no sputum. Chest x-ray examination showed that the right diaphragm was raised and there was some ill-defined shadowing at the right base. The blood count and E.C.G. were normal. He was treated with penicillin and anticoagulants, and made a satisfactory recovery.

Comment.—The previous attacks of pleural pain and the sudden onset of the illness described above suggest pulmonary embolism. He may have had phlebothrombosis in his pelvis following the operation on his hip.

Case 9

A miner aged 56 was admitted from the casualty department as an emergency case on March 15, 1957. He had collapsed at work, having had a sudden pain in his back and the left side of his chest. The pain was worse on deep respiration. One year previously he had been treated for a papilloma of the bladder; this had been removed by diathermy. A cystoscopy had been performed on February 5, 1957.

Physical examination revealed that respiration was shallow, being restricted because of pain. The percussion note was impaired at the left lung base and there were crepitations at both bases. He was not shocked (blood pressure 150/80). His temperature was normal on admission but rose to 101° F. (38.3° C.) on the fourth day. There were no signs of venous thrombosis. The E.C.G. and blood count were normal. The sputum was mucopurulent, and on culture a moderate growth of *H. influenzae* was obtained. Chest x-ray examination showed elevation of the right diaphragm with segmental shadows in both lower zones. The shadows one week later had cleared, leaving the well-defined markings of linear collapse.

Because of the nature of the sputum he was treated at first with penicillin and streptomycin, but not with anti-

coagulants. He did not improve, and three days after admission the sputum became stained with bright blood, and at times was pure blood. A diagnosis of pulmonary embolism was made. He was treated with anticoagulants and slowly improved.

Comment.—The sudden onset of the symptoms and the lack of response to antibiotics suggest a diagnosis of pulmonary embolism. In this case pelvic veins, thrombosed during diathermy of a bladder papilloma, were a probable source of the embolus.

Case 10

A lorry driver aged 38 was admitted to hospital on February 1, 1957, with a history of pleuritic pain on the left side of his chest with shortness of breath for one week. He had complained of a dry cough for three days. Two weeks before admission he had abdominal pain, colicky in nature, situated in the hypogastrium, together with diarrhoea which persisted for 48 hours. The stools were of normal colour and were not blood-stained.

In 1944, while in the Army, he had recurrent attacks of diarrhoea lasting nine months in all, for which he had been fully investigated at a military hospital in Germany. The diarrhoea settled completely on return to this country.

Physical examination revealed a breathless man in pain, with a temperature of 101° F. (38.3° C.). Respiratory system: Diminished movement; dullness to percussion and reduced breath sounds, with rales at the right lung base. The pulse rate was 100 a minute, regular. B.P. 120/80. Heart sounds normal. No evidence of cardiac failure. The central nervous system was normal in all respects. There was no abdominal tenderness; the spleen and liver were not palpable.

Investigations.—Urine contained no albumin or sugar. X-ray examination of the chest initially showed a triangular shadow in the right lower zone consistent with consolidation. Haemoglobin, 14.8 g./100 ml.; white cells, 8,000 per c.mm. (normal differential). E.S.R., 51 mm. in one hour. Sputum: No pus cells, no pathogens grown; culture for acid-fast bacilli was sterile.

Forty-eight hours after admission he experienced a similar attack of pain on the left side of his chest. Physical examination revealed a pleural rub with rales at the right lung base. Following this he coughed up a small quantity of bright red blood. Three days later there was a recurrence of pleurisy on the right side. A second chest x-ray examination showed a triangular opacity at the left base.

He was treated with penicillin and anticoagulants and made an uninterrupted recovery. Radiological resolution of both shadows at the lung bases had occurred three weeks after admission. A bronchogram at that time was normal.

Comment.—In view of the repeated attacks of pleurisy, the absence of purulent sputum, plus the haemoptysis, this man was regarded as having had pulmonary emboli although at no time was there evidence of venous thrombosis.

Discussion

Thrombosis of the calf veins may not produce pain or tenderness. The severity of the symptoms may be related to the extent of the clot (Marks *et al.*, 1954). Thrombosis may occur intra-abdominally, and Marks *et al.* noted that in 12 out of 70 fatal cases pulmonary emboli originated from sites other than the calf. Thus the absence of physical signs does not exclude the diagnosis of deep venous thrombosis, and indeed Grundersen (1955) observed that clinical evidence of venous thrombosis was present in less than 10% of 119 cases of pulmonary embolism. Of these 119 patients, 7 were ambulant and in good health just before they had a pulmonary embolus. Hampton *et al.* (1954) described 10 cases of pulmonary embolism in which no signs of peripheral venous thrombosis were found clinically, although thrombosis in deep veins of the leg was later demonstrated by

venography in three patients, and another patient had a thrombosed haemorrhoid. All 10 patients were at work and had no heart disease when they developed chest symptoms. Renner (1949) reported a similar case. This patient presented with the features of a myocardial infarction. The electrocardiographic changes were interpreted as those of auricular flutter with marked right axis deviation. Later, clinical and radiological signs suggestive of pulmonary embolism became evident. A well-documented case of pulmonary embolism occurring in a fit and active man has also been described (Hollister and Cull, 1956, Case I).

In cases where embolism cannot be proved it is possible that occlusion of the pulmonary artery is due to thrombosis. Pulmonary embolism cannot always be distinguished pathologically from autochthonous pulmonary-artery thrombosis, particularly as thrombus may form around an embolus. The appearances are difficult to interpret if the patient died of cor pulmonale a long time after the embolism occurred. The common experience is that peripheral venous thrombosis precedes occlusion of the pulmonary artery, and pulmonary embolism is thought to be much commoner than thrombosis of the pulmonary artery. As peripheral venous thrombosis may be "silent" it is sometimes clinically impossible to distinguish between pulmonary embolism and thrombosis. It is our intention to emphasize the clinical syndrome; for the exact differentiation is not important, the treatment for both conditions being the same.

None of the cases we have described had signs of venous thrombosis when first seen, and eight of these patients had been well and active up to the time when they first developed symptoms. Almost all had recurrent attacks of pleurisy which did not respond to treatment with antibiotics, although temporary improvement after each acute attack was attributed to the antibiotic given at the time. An important feature was the absence of purulent sputum. The patients treated with anticoagulants recovered and have not developed cor pulmonale.

As anticoagulant treatment is sometimes effective, the diagnosis of pulmonary embolism is important. It should be considered in cases of recurrent pleurisy or pneumonia not responding to antibiotic therapy, particularly if symptoms and signs are bilateral, even in the absence of signs of venous thrombosis.

Summary

Ten cases of pulmonary embolism are described. None of the patients had signs of peripheral venous thrombosis when first seen, and eight had been active until they developed chest symptoms. Pulmonary embolism occurring in patients who appear to be fit, are active, and have no signs of venous thrombosis has been termed "unheralded pulmonary embolism." The patients treated with anticoagulant recovered. Three patients died, and the necropsy findings are reported.

In nine of the cases the clinical picture was that of recurrent attacks of pleurisy which did not respond to treatment with antibiotics. The sputum was blood-stained but not purulent—an important aid to diagnosis.

Reference is made to similar cases that have been reported.

We wish to thank Dr. T. E. Gumpert, Dr. H. P. Brody, and Professor C. H. Stuart-Harris for permission to use the records of cases under their care, and Dr. J. L. Edwards for his help and advice.

REFERENCES

- Brooks, W. D. W. (1952). In *Diseases of the Chest*, edited by G. Marshall and K. M. A. Perry, vol. 2. London.
- Grundersen, P. (1955). *Nord. Med.*, 53, 565.
- Hampton, A. O., Prandoni, A. G., and King, J. T. (1945). *Bull. Johns Hopk. Hosp.*, 76, 245.
- Hollister, L. E., and Cull, V. L. (1956). *Amer. J. Med.*, 21, 312.
- Marks, J., Truscott, B. M., and Withycombe, J. F. R. (1954). *Lancet*, 2, 787.
- Renner, W. F. (1949). *Ann. Intern. Med.*, 31, 1090.