AN EPIDEMIC OF 'ACUTE EOSINOPHILIC PNEUMONIA' FOLLOWING 'BEER DRINKING' AND PROBABLY DUE TO INFESTATION WITH *ASCARIS LUMBRICOIDES*

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Patz[1] has recently described an epidemic of 'acute eosinophilic pneumonia' which occurred during November 1956 in 12 adult Bantu males at Middelburg, Transvaal. All the patients had attended a beer drink, illicitly held at a nearby brickfield, 6 days before the onset of symptoms. The illness started with a feeling of malaise, headache and generalized body pains, soon followed by rigors, pyrexia, and a severe cough productive of scanty white sputum. The patients were extremely breathless, and wheezing was a prominent feature. Rhonchi and fine crepitations were audible throughout the chest. From the 12th to the 17th day after drinking the beer the patients underwent the worst of their illness, but they then improved rapidly and were able to leave hospital after a further 5 days.

At least 3 patients had had a generalized urticarial rash

TABLE I. RELEVANT FEATURES OF THE 13 PATIENTS

Case and sex	*Date of drinking*	*Onset of respiratory symptoms*	*Date and place of examination*	*Stool for ascaris*	*Urticaria*	*Pyrexia*	*Leucocyte count*		*Eosinophils*		*Chest X-ray*
							Date	*c.mm.*	*%*	*Wet count*	
1 M	15.1.59	20.1.59	25.1.59 N.E.H. (admitted)	Negative 17.2.59. Negative 15.5.59. Negative 29.5.59	Present	Present	26.1.59 18.2.59	29,000 6,700	10 36	 2,200	Extensive bilateral mottling. Right interlobar effusion
2 M	15.1.59	20.1.59	24.1.59 C.H. admitted)	Positive 11.2.59	Present	Present	27.1.59 9.2.59	39,900 20,400	44 66		Extensive bilateral mottling. Interlobar effusions. Probable Kerley 'B' lines
3 F	15.1.59	22.1.59	25.1.59 C.H. (admitted)	Negative 10.5.59. Positive 29.5.59	Present	Present	26.1.59 18.2.59	16,400 6,300	4 12	 770	Extensive bilateral mottling. Interlobar effusion
4 M	18.1.59	25.1.59	29.1.59 N.E.H. (admitted)	Positive 18.2.59	Present	Present	30.1.59 16.2.59	9,600 6,100	3 21	 1,023	Increased hilar shadows. Right interlobar effusions. Mild mottling
5 F	15.1.59	20.1.59	30.1.59 (Ferndale)	Positive 18.2.59	Present	Not known	18.2.59	11,800	21	2,200	Not done
6 M	15.1.59	20.1.59	28.1.59 N.E.H.	Positive 18.2.59	Present	Present	29.1.59 18.2.59	19,500 4,400	36 12	 407	Mild bilateral mottling
7 M	18.1.59	25.1.59	1.2.59 B.H.	Positive 29.5.59	Present	Not known	21.2.59	8,900	7	638	Increased hilar shadows. Mild bilateral mottling
8 M	18.1.59	25.1.59	1.2.59 B.H.	Positive 18.2.59	Present	Not known	21.2.59	6,500	15	1,166	Increased hilar shadows. Interlobar effusions. Probable Kerley 'B' lines
9 F	15.1.59	24.1.59	31.1.59 N.E.H.	Negative 10.5.59. Positive 29.5.59	Present	Present	Not done				Normal (31.1.59)
10 F	18.1.59	21.1.59	1.2.59 C.H.	Positive 18.2.59	Present	Not known	21.2.59	6,500	15	990	Not done
11 M	15.1.59	Not known	5.3.59 (Ferndale)	Negative 17.2.59. Negative 15.5.59. Negative 29.5.59	Present	Not known	Not done				Not done
12 F	15.1.59	19.1.59	Not seen	Positive 16.2.59	Present	Not known	Not done				Not done
13 M*	15.1.59	±22.1.59	Not seen	Not done	Present	Not known	Not done				Not done

C.H. = Coronation Hospital, B.H. = Baragwan th Hospital, N.E.H. = Non-European Hospital.

* Patient died on or about 27 January 195[illegible], without receiving medical attention.

before the onset of other symptoms. Blood counts showed an eosinophilia which ranged from 7% of 9,100 WBCs in a mild case to 63·5% of 24,000 WBCs in one of the most severe. Radiological signs varied from increased bronchovascular markings to bilateral diffuse mottling of the lung fields. Eight patients were passing ascaris ova at the start of the illness and all but one of the remainder had ova in their stools 8 - 16 weeks later. Patz[1] considered that infestation, in some cases probably massive, with *Ascaris lumbricoides* was the most likely cause of this illness. He thought it probable that the patients were infected at the party although he was unable to offer any explanation of how the food or drink had become heavily contaminated with ascaris ova.

In January 1959 we encountered a remarkably similar epidemic in the Ferndale district of Johannesburg. To our knowledge, 13 Bantu adults were involved and the only common factor was that they had all drunk beer from the same source. It is the purpose of this paper to describe the features of this illness and to discuss briefly the possible aetiology.

FEATURES OF THE EPIDEMIC

Thirteen adult Bantu, 5 of whom were females, drank beer from the same source between 15 and 18 January 1959. Most of the witnesses were suspicious of us and their stories were unreliable. It was only by repeated questioning of the 10 whom we were able to contact, and of several relatives or friends who did not drink the beer, that we could ascertain some of the facts. A summary of the relevant features of all the patients is shown in Table I.

Of the 13 patients, 3 (cases 1 - 13) apparently received no medical treatment. One of these (case 13) died on a neighbouring farm on approximately the 7th day of his illness. Another (case 11) was apparently very ill, but was only seen by us some 5 weeks later when he had fully recovered. The third (case 12) was mildly affected. The remaining 10 patients all received medical treatment and our knowledge of their illnesses is fairly complete. Six of them were seen by us personally during the acute stage of the illness and of these 2 were admitted to the Non-European General Hospital (cases 1 and 4), 2 (cases 2 and 3) to Coronation Hospital, 1 (case 5) was examined in her room at Ferndale, and 1 (case 6) was treated by us in the outpatient department of the Non-European General Hospital. Of the remaining 4 patients, 2 (cases 7 and 8) were treated at the Baragwanath Hospital casualty department, 1 (case 9) was treated for 4 days, unfortunately without our knowledge, in the casualty department of the Non-European General Hospital and the last (case 10) received outpatient therapy at Coronation Hospital.

Clinical Features

All patients had developed an itchy skin rash preceding the respiratory symptoms. The rash was still present in 2 (cases 4 and 5) when seen by us and had the appearance of a generalized urticaria.

Respiratory symptoms developed 5 - 7 days after the beer was drunk and consisted of cough and breathlessness with prolonged expiration. The cough was initially dry or productive of a little mucopurulent sputum, but in at least 3 patients (cases 1 - 3) whom we observed, the sputum became frothy, blood-stained and copious 5 - 8 days after the onset of the respiratory symptoms. Five of the 6 patients seen by us (cases 1 - 5) were cyanosed. Dyspnoea was marked in 3 of them (cases 1 - 3) and was moderate in 2 (cases 4 and 5). All 6 had prolonged expiration with rhonchi and crepitations audible over both lung fields. They were pyrexial, the temperature reaching 104° F. in 1 patient (case 2). Similar clinical signs were described in the 4 patients (cases 7 - 10) examined by other medical colleagues. The patients were most ill 5 - 8 days after the onset of respiratory symptoms and about 11 - 14 days after drinking the beer. The man

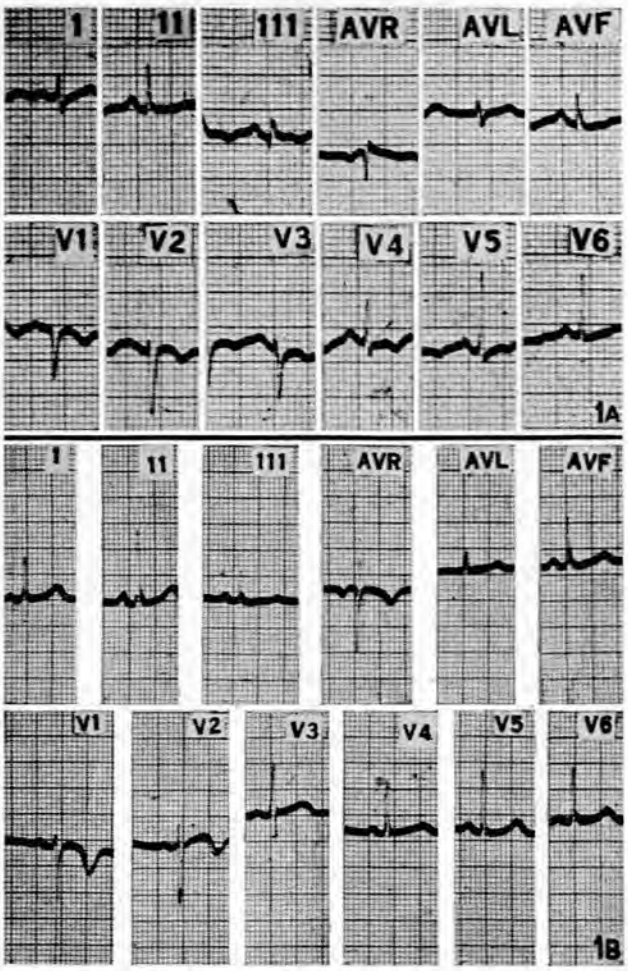

Fig. 1. (*a*) Electrocardiogram of case 2 taken 6 days after the onset of respiratory symptoms when the patient was in mild congestive cardiac failure. The Q in standard lead 3, Qr in AVR, RS in AVL, and inverted or flattened T waves in all the V leads are compatible with acute right ventricular strain.
(*b*) Electrocardiogram 2 weeks later when all symptoms and signs had disappeared. The T waves in V_1 and V_2 are normal for an adult Bantu.[23]

who died (case 13), according to our rather unreliable witnesses, had been coughing and breathless for 5 days. Two patients (1 and 2) developed mild congestive heart failure 6 days after the onset of the respiratory symptoms, with gallop rhythm and electrocardiographic changes compatible with acute cor pulmonale (Fig. 1). Treatment included antibiotics, bronchial antispasmodics, and, in 3 patients (cases 1 - 3), steroids. The 2 patients who developed congestive cardiac failure were digitalized and given mercurial diuretics. None of this therapy seemed to alter the course of the illness. The patients improved spontaneously about 10 days after the onset of respiratory symptoms and all seemed completely well a week later.

Chest X-rays were taken of 8 patients (cases 1 - 4 and 6 - 9). During the acute stage of the illness, radiological changes varied from increased hilar shadows in the mild cases to extensive mottling of both lung fields in the severe (Fig. 2). Small interlobar effusions were present in 5 cases and probable Kerley 'B' lines in 2. Serial X-rays showed a gradual return to normal within 3 weeks of the onset of the respiratory symptoms.

Special Investigations

Nine patients had at least 1 blood count within 5 weeks of the onset of symptoms. An eosinophilia was present in all

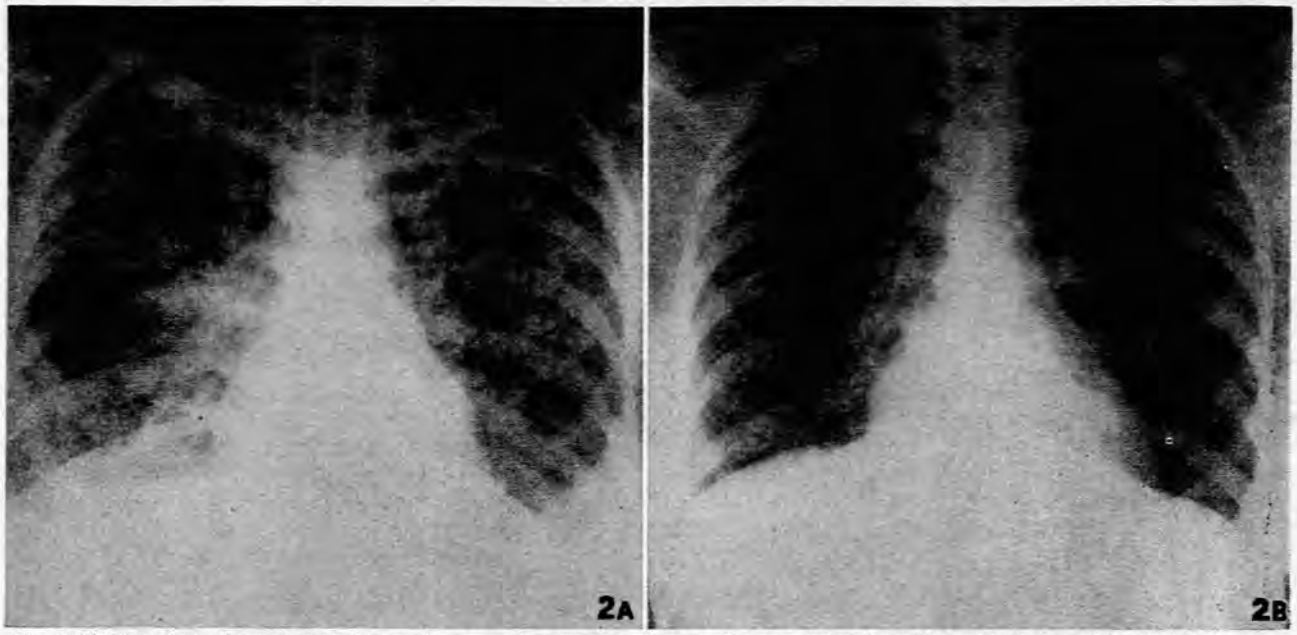

Fig. 2. (*a*) Chest X-ray of case 3 taken 4 days after onset of respiratory symptoms. Extensive bilateral mottling of both lung fields is clearly (*b*) Chest X-ray 8 days later.

and ranged from 7% of 8,900 WBCs to 66% of 20,400 WBCs (Table I). The stools were examined for ascaris ova in all except the man who died (Table I). Seven of the 8 patients whose stools were examined within 4 weeks of the onset of symptoms were passing ascaris ova. Three of the remaining 4 patients were passing ascaris ova when their stools were examined for the first time 4 months later. In 2 patients (cases 1 and 11) we failed to find ova.

The urine of 9 patients was negative for bilharzia ova. The bilharzia complement-fixation test was negative in 8 and doubtful in 2 (cases 5 and 6). The skin test for histoplasmosis was negative in the 8 patients on whom this was performed. Virus studies, including psittacosis, were negative in 4 patients. The sputa of 5 patients (cases 1 - 4 and 6) were sent for microscopic examination and culture. Various, possibly pathogenic, organisms were grown in 4 cases. Nothing of significance was reported on the microscopic examination (performed at the South African Institute for Medical Research) though unfortunately a request for specific search for eosinophils and ascaris larvae had not been made.

DISCUSSION

Ascaris lumbricoides infestation in man starts with the swallowing of embryonated eggs by the host; these eggs then hatch in the small intestine. On the seventh or eighth day after inoculation the larvae migrate in the blood stream through the liver to the lungs where growth continues. They then return to the small intestine, *via* the bronchi, trachea and oesophagus, and reach maturity in 8 - 10 weeks producing unsegmented, non-infective eggs which leave the host in the stools. Further development takes place in a favourable atmosphere, usually moist soil, and within 3 weeks the eggs are infective.[2]

The passage of the larvae through the lungs often produces no symptoms, but radiological signs of transient pulmonary infiltrations may be present at this stage.[3-5] Where respiratory symptoms do occur, however, the clinical syndrome described by Loeffler is the one most frequently seen.[6-11]

Loeffler's syndrome consists of a mild pyrexia lasting about 1 week, associated with a slight productive cough and an eosinophilia. There are minimal signs on clinical examination of the chest, though X-ray examination reveals transient pulmonary infiltrations. Loeffler[7] believed that this syndrome was a manifestation of an allergic reaction to various allergens, but that the migration of ascaris larvae through the lungs was one of the commonest causes. The patient is essentially only slightly ill and spontaneous recovery is invariable.

The condition known as tropical eosinophilia may also be related to some parasitic infestations. This condition, however, which is characterized by pyrexia, weight loss, cough, asthma, radiological signs of pulmonary infiltrations, and a marked eosinophilia, is a more severe illness and runs a protracted course.[12,13] *Toxascaris canis*,[14] mites,[15] and filariae[16] are the parasites most commonly incriminated, and a good response to therapy with organic arsenicals usually occurs.

Pulmonary ascariasis is known to be associated sometimes with a severe acute respiratory illness,[17-21] and the clinical picture is then similar to that described by Patz[1] and to our own cases. In 1922, Koino[19] experimentally swallowed 2,000 mature human ascaris ova. Two days later he developed anorexia and headache, followed 6 days after swallowing the eggs by a severe pyrexial illness with rigors, rapid pulse, cyanosis, extreme dyspnoea, and paroxysmal cough productive of increasing quantities of sputum which later became blood stained. Râles were present throughout the chest and ascaris larvae were recovered from the sputum. The illness lasted 10 days. Beaver and Danaraj[20] described an adult male who presented with a 10-day history of very severe bronchial asthma. On examination the patient was critically ill, coughing, and very dyspnoeic with marked bronchospasm. A blood count showed a leucocytosis of 21,400 with 37% eosinophils. The patient died a few hours after admission; at autopsy there was marked dilatation of the right ven-

tricle and ascaris larvae were found in the bronchioles. Hemming[21] observed that severe respiratory symptoms were common among malnourished Indian children in the Fiji Islands. Within a period of only 40 days he saw 100 cases of 'ascaris pneumonitis'. His patients, one of whom died, were all seriously ill and had presented with severe cough, asthma, cyanosis and pyrexia.

The epidemic we have described seems to us virtually identical to that reported previously by Patz.[1] The illness itself, including the urticaria, small interlobar effusions, eosinophilia and severe asthma, is strongly suggestive of an acute allergic reaction and it seems likely that the causative agent was contained in the beer. Certainly everyone who drank this particular brew became ill to a greater or lesser extent, whereas at least 1 person, to our knowledge, who was present at the party but did not drink, remained completely well. Neither Patz[1] nor ourselves, however, were able to ascertain the exact constituents of the beer, and in neither instance is there conclusive evidence that the swallowing of ascaris ova was the cause of the subsequent illness. Possibly due to the death of one of their number, those who had attended the party were suspicious, particularly the man Arthur (case 1) and his wife who brewed the beer. Originally, all the witnesses had denied that there was anything unusual about the beer, but after repeated questioning and some direct suggestion by us, several of them later volunteered that it had contained sand. Since the beer had been hidden for 3 days on the bank of a nearby stream in order to avoid detection by the police, it might have been contaminated accidentally or by someone ill-disposed towards the brewers. Perhaps another possibility is that it was made originally with impure river water.

Of the 12 cases described by Patz,[1] 8 had ova in the stools at the start of the illness and were thus almost certainly already infected when they drank the beer. Three of his remaining 4 patients had ova in the stools 8 - 16 weeks later. Patz[1] considered that the patients already infected had a higher eosinophilia and more severe illness than the others. Since it usually takes at least 8 weeks for the worm to reach maturity and therefore produce eggs,[2] however, it is debatable whether his 3 patients who had ova in their stools at 8 - 16 weeks were not also already infected at the time of the beer drink. Of our 13 cases, the man who died was never examined, 7 patients had ova when the stools were examined 4 - 5 weeks after drinking the beer, and 3 others had ova present 3 months later. The probable existence of intestinal ascaris infestation in the majority of Patz's and our patients at the time of the respective 'parties', when infection or re-infection is thought to have occurred, does not weaken the argument that the acute pulmonary illness could also have been due to ascariasis. Beaver and Danaraj,[20] and Baumann,[8] believe that the most severe pulmonary reactions occur either in patients already infested with ascaris, and consequently more liable to be allergic to the larvae passing through the lungs, or in those where a massive infestation takes place for the first time.

In 2 patients (cases 1 and 11), one of whom had been extremely ill (case 1), we were unable to find ova at any time. Absence of ova in the stools, however, is not incompatible with ascaris infestation.[3,9,10,22] Minteer *et al.*[22] described 11 patients in whom adult ascaris worms were demonstrated by barium-meal examination, yet only 3 had positive stools. In their remaining 8 patients, no ova were found in a total of 20 stool examinations.

We are unable to offer any alternative explanation for the cause of this epidemic. The only common factor to all our patients was that they drank beer from the same source. Four of them (cases 4, 7, 8 and 10) bought the beer and drank it elsewhere, and at least 1 person, in fact the wife of Arthur (case 1), who was present at the party but did not drink, remained well.

If an allergic reaction to the passage of ascaris larvae through the lungs was the cause, however, then it is surprising that this syndrome is not seen more frequently. Infestation with ascaris is extremely common in this country especially among the Bantu population. A possible explanation is that our patients swallowed an unusually large number of ova and, especially as at least 7 of them were already infected, were probably more liable to have a severe reaction.[8,20] How or why the beer became contaminated with these ova remains a problem unsolved by us.

SUMMARY

An epidemic is described of an acute respiratory illness in 13 adult Bantu (5 females) which occurred during January 1959 in the Ferndale district of Johannesburg. Features of the illness included a generalized urticarial skin rash, cough, breathlessness with prolonged expiration, cyanosis and pyrexia. Two patients developed congestive cardiac failure with electrocardiographic evidence of acute cor pulmonale. Radiological changes varied from increased hilar shadows in the mild cases to extensive mottling of both lung fields in the severe. An eosinophilia was present in all 9 patients on whom a blood count was done. The stools of 12 patients were examined for ascaris ova and were positive in 10. One patient died without receiving medical attention.

This epidemic is thought to be remarkably similar to that reported previously by Patz,[1] which took place during November 1956 in 12 Bantu males at Middelburg, Transvaal, and which was attributed to infestation with *Ascaris lumbricoides*.

The common factor to our patients was that they had all drunk beer from the same source. It is postulated that the respiratory illness resulted from infestation with *Ascaris lumbricoides* and that the beer was the source of infection.

The pulmonary manifestations of ascariasis are briefly discussed.

ADDENDUM

Since this paper was written, a case of pulmonary ascariasis was reported by Simson and Heinz.[24] The patient, a 3-year-old Bantu female, died after a severe respiratory illness of four days' duration. On histological examination of the lungs, numerous nematode larvae, resembling *Ascaris lumbricoides*, were found in the bronchioles. As in our cases, the source of the infestation was unknown.

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REFERENCES

1. Patz, I. M. (1959): Cent. Afr. J. Med., **5**, 399.
2. Chandler, A. C. (1955): *Introduction to Parasitology*, 9th ed., pp. 440-447. New York: John Wiley.
3. Spühler, O. and Kartagener, M. (1944): Schweiz. med. Wschr., **74**, 1144.
4. Lagarde, C. (1956): Quoted in Trop. Dis. Bull., **53**, 499.
5. Pokorny, M. and Klausová, L. (1951): Quoted in *Ibid.*, **48**, 177.
6. Loeffler, W. (1936): Schweiz. med. Wschr., **66**, 1069.
7. *Idem* (1945): Presse méd., **39**, 516.
8. Baumann, H. (1944): Schweiz. Med. Wschr., **74**, 326.
9. Muller, R. W. (1938): Dtsch. med. Wschr., **64**, 1286.
10. Vogel, H. and Minning, W. (1942): Quoted by Spühler, O. and Kartagener, M. (1944): *Loc. cit.*[3]
11. Crofton, J. W., Livingstone, J. L., Oswald, N. C. and Roberts, A. T. M. (1952): Thorax, **7**, 1.
12. Frimodt-Moller, C. and Barton, R. M. (1940): Indian Med. Gaz., **75**, 607.
13. Ball, J. D. (1950): Trans. Roy. Soc. Trop. Med. Hyg., **44**, 237.
14. Chaudhuri, R. N. and Saha, T. K. (1959): Lancet, **2**, 493.
15. Van der Sar, A. (1946): Amer. Rev. Tuberc., **53**, 440.
16. Webb, J. K. G., Job, C. K. and Gault, E. W. (1960): Lancet, **1**, 835.
17. Adams, A. R. D. and Maegraith, B. G. (1953): *Clinical Tropical Diseases*, p. 233. Oxford: Blackwell Scientific Publications.
18. de Silva, C. C. (1957): J. Trop. Pediat., **3**, 62.
19. Koino, S. (1923): Quoted in Trop. Dis. Bull., **20**, 235.
20. Beaver, P. C. and Danaraj, T. J. (1958): Amer. J. Trop. Med. Hyg., **7**, 100.
21. Hemming, G. R. (1956): Med. J. Aust., **1**, 501.
22. Minteer, D. W., Hayes, J. D. and Youngstom, K. A. (1954): Amer. J. Roentgenol., **71**, 416.
23. Grusin, H. (1954): Circulation, **9**, 860.
24. Simson, I. W. and Heinz, H. J. (1960): Leech, **30**, 140.