That outbreak afforded an opportunity for a more general application of the method by reason of the fact that ten of the fifteen city districts had been invaded by epizoötic plague. During the course of this work (1907-08) observations were made and materials gathered with a view to improving the technique, and of adding new features for future studies. Several new features were added, only one of which need be mentioned in this paper.

In the course of the epidemic cases appeared among the refugees of the great fire, who had been housed by the city in cottages on the public squares. These cottages rested upon the ground without intervening spaces between the floors and the soil, thereby affording excellent harborage to rats. The usual methods of getting rid of infection were employed but without success. Finally the cottages were raised eighteen inches from the ground, and no further cases occurred. This experience gave rise to a form of ratproofing which, for lack of a better title, has been called ratproofing by elevation. It is a modified form adapted to the construction of cheap frame buildings for suburban districts.

* * *

Ratproofing may be defined as a method of insulating structures against the ingress and egress of rodents. It is necessary because of the general use of wood as building material in this country, as well as because of certain forms of construction, both of which lend themselves to the harboring of rats. If concrete could be substituted for wood on ground areas, and if hollow walls could be sealed from below, the rodent menace would be brought under control. At all events, building ordinances should be based upon a knowledge of the habits of our common rodent species. A retaining wall of concrete extending entirely around a building, eighteen inches below and twelve inches above the ground, will protect it against the burrowing rat, and wire screens of the proper mesh, placed over ventilators and low windows, will exclude the climbing species.

* * *

In those days nobody knew anything about fleas, the intermediary agents in the dissemination of plague. The subject apparently had not merited scientific investigation up to that time. Books on entomology had nothing to offer as to bionomics or the distribution of species which factors seemed important in view of the rôle assigned to insects as vectors of disease. In order to cover this field, and to obtain information of value, a student of entomology of the University of California was employed and put to work in the laboratory. A flea survey was made of San Francisco, specimens removed from live rats were identified as to species and much useful data were collected. It was shown that X. Cheopis, the plague flea of India, as described by the British Plague Commission, was one of the common species found in San Francisco; C. fasciatus, however, predominated throughout all seasons of the year. Both fleas should be considered as important factors in the spread of plague infection from rat to rat and from rat to man.*

United States Public Health Service,
Washington, D. C.

* References to articles and items on bubonic plague control, which have been printed in CALIFORNIA AND WESTERN MEDICINE in recent years, follow:

1. The Bubonic Plague—Does History Repeat Itself? 27:666 (November), 1927.
2. An article under Current Comment, 27:683 (November), 1927.
3. The 1924 Los Angeles Plague Epidemic, 27:684 (November), 1927.
4. Predicting Epidemics of Plague in the Punjab, 27:826 (December), 1927.

Other references in the "Twenty-five Years Ago" department of CALIFORNIA AND WESTERN MEDICINE include the following:

5. 36:69, second column, first item; and 36:70, first column, first item (January), 1932.
6. 36:142, second column, first two items; and 36:143, first column, first item (February), 1932.
7. 36:213, first column, first two items; and second column, first two items (March), 1932.
8. 36:301, second column, first two items; 36:302, first column, first item (April), 1932.
9. 36:372, second column, first two items; 36:373, first column, first item (May), 1932.
10. 36:477, second column, first two items (June), 1932.
11. 37:69, second column, first two items (July), 1932.
12. 37:142, second column, first item (August), 1932.
13. 37:215, first column, first two items (September), 1932.
14. 37:284, first column, first item (October), 1932.
15. 37:358, first column, first three items (November), 1932.
16. 37:429, first column, first four items (December), 1932.
17. 38:69, first column, first two items (January), 1933.

CLINICAL NOTES AND CASE REPORTS

PRIMARY CARCINOMA OF THE LUNG WITH UNUSUAL MANIFESTATIONS*

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THAT primary carcinoma of the lung is definitely on the increase is amply proved by clinical and postmortem studies during the last three decades. While older statistics, quoted by Fried [1] show an incidence of less than 0.2 per cent, more recent reports [2] demonstrate that primary carcinoma of the lung is responsible for from 5 to 10 per cent of all deaths from cancer.

The object in presenting this case study is to emphasize the peculiar manifestations under which primary carcinoma of the lung may mask itself. While our acuity in the diagnosis of this malignancy has increased extensively, still many an expert clinician admits that it presents a baffling problem. Over 80 per cent of Fishberg's [3] patients entered his clinic with diagnoses other than lung tumor. Since this growth exhibits a very vigorous and early metastatic power in about 75 per cent of the cases,[4] it is evident that the clinician has two different pictures to keep in mind: the typical form, wherein there are localizing symptoms to the chest, and an atypical one where the symptomatology is referable entirely to the metastatic

* Case in General Hospital of Fresno County.

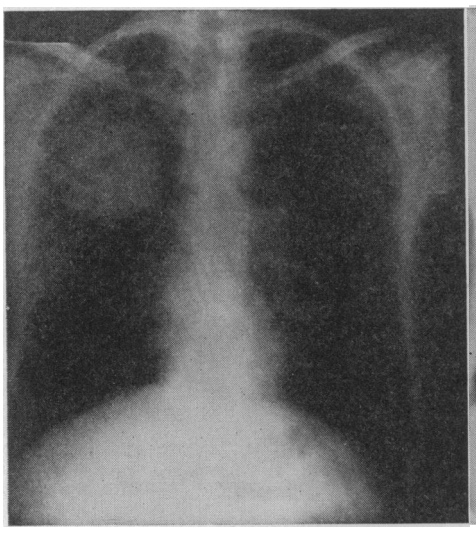

Fig 1.—X-ray plate of chest, July 19, 1932, showing opacity in upper right lung field.

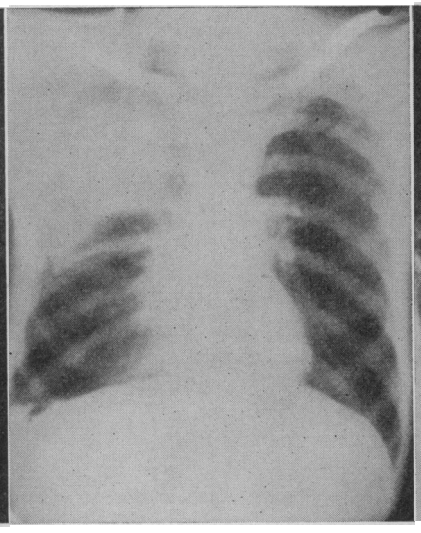

Fig. 2.—Chest plate, October 24, 1932, showing advance of lesion.

Fig. 3.—X-ray of left middle finger, August 2, 1932, showing defect (metastasis) of middle phalanx and periostitis.

involvement. It is interesting that our patient presented neither one of the above clinical syndromes, but that he complained of secondary pulmonary osteo-arthritic changes in his fingers. Many reports in the literature on this subject fail to mention such findings. In a review of the frequency of the different symptoms and signs of primary carcinoma of the lung, the California Medical Association Cancer Commission cites them as being 25 per cent. Other symptoms are as follows: cough, 90 per cent; pain, fever, dyspnea, 53 per cent; cyanosis, 50 per cent; hemoptysis, 40 per cent; effusion, 50 per cent; loss of weight, 35 per cent; clubbing of fingers, 25 per cent; cavitation, 14 per cent; and stridor.

M. Fishberg[3] claims an early clinical diagnosis can be made if we bear in mind that the disease manifests itself in three forms:

One, the pulmonary form, simulating tuberculosis;

Two, the pleural form, simulating pleurisy, dry or with serous, sanguineous, or purulent effusions;

Three, the excavating form, simulating abscess and gangrene of the lung.

Polerski[7] emphasizes the following characteristic physical findings:

High position of the diaphragm on the affected side;

Paradoxical respiration with a see-saw movement of the diaphragm;

Pendulum movement of the heart;

Infraclavicular flatness fusing with cardiac dullness.

Our interest in our own patient arose from the fact that he had nonlocalizing symptoms in the chest. The secondary pulmonary osteo-arthritic changes of the fingers and toes, and the marked degree of periostitis of the long bones, with their accompanying severe pain and tenderness, presented a very misleading clinical entity. A very similar case was presented in 1926 by H. Brunn.[6] Rogers,[8] in his series of fifty cases of primary carcinoma of the lung, states that 44 per cent of his patients gave as their first symptoms one that was not connected with the chest, and that 12 per cent went through the entire course of the illness without a pulmonary symptom.

REPORT OF CASE

History.—T. B., age forty-seven, an Armenian farmer, was admitted to the Medical Service of the Fresno County Hospital on July 7, 1932. The only complaint at that time was pain in the finger-joints, with moderate tenderness and swelling.

It was difficult to obtain a detailed history, as the patient's command of the English language was very limited. The onset of the disease was ushered in five months previously with multiple joint involvement, settling finally in the interphalangeal joints. The pain in these joints had so progressed that the patient became bedridden the last four months of his illness. He was unable to feed himself, and even the slight touch of bedcovers would bring forth an outcry of pain.

Past history was of no import, the patient enjoying very good health up to the onset of the present illness. Family history was irrelevant.

Physical Examination. — The most striking observation at first sight was a marked degree of clubbing of both fingers and, to a lesser degree, of the toes.

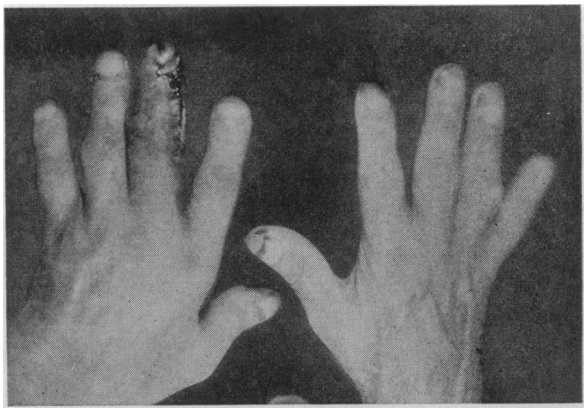

Fig. 4.—Photograph of fingers showing clubbing and the metastatic lesion of finger.

Inspection revealed a fairly emaciated, middle-aged man, sitting in bed, and holding his swollen fingers in front for protection. The temperature was 99.6 degrees. Respiration, 20. The pupils were equal, and reacted to both light and accommodation. All teeth were absent (removed on advice of former physician for "arthritis"). The supra- and infraclavicular fossae were prominent. No limitation of thoracic breathing was noted. The diaphragmatic excursion on both sides was of normal range. No changes were noted in the vocal fremitus. There was questionable dullness and somewhat diminished breath sounds over the right infraclavicular region. No râles were present. The area of cardiac dullness was of normal position and size. The heart sounds were distant and scarcely audible. No murmurs were present. The pulse was 88. The blood pressure was 120/70.

The abdomen presented a peculiar formation of cicatricial tissue due to a previous skin infection of undetermined etiology. No abnormal masses were palpable. No tenderness or deformity of the spine was noted. Moderate hyperesthesia of all extremities and marked clubbing of both fingers and toes, with swelling and tenderness of the interphalangeal joints. There were no abnormal reflexes elicited. Rectal examination revealed good sphincter tone with no prostatic enlargement.

Laboratory Findings. — Blood: hemoglobin, 67 per cent; red blood cells, 3,680,000; white cells, 5,850. The urinalysis was repeatedly negative. The sputum examinations were negative for acid-fast bacilli, elastic fibers, and abnormal cells. Repeated blood cultures were negative. A mantoux using 1 milligram gave a negative reaction. Blood, spinal and provocative Wassermanns were negative. The spinal fluid was clear, under no increased pressure and no pleocytosis.

Roentgenograms.—It was purely of academic interest alone that a roentgenogram of the chest was ordered because of the secondary pulmonary osteopathic changes noted in the fingers. Much to our surprise, a circular dense area was revealed occupying the right upper lobe in the periphery. Figures 1 and 2 demonstrate the progress of the lesion during the patient's five months' stay in the hospital.

There were diversified opinions among us as to the interpretation of the first chest plate. The following diagnoses were considered: Pulmonary abscess; neoplasm (primary or secondary), foreign body reaction, and echinococcus cyst. At this time x-ray studies of the extremities revealed secondary pulmonary hypertrophic osteo-arthropathy, namely, a marked periostitis of the long bones and well-defined spurring of the interphalangeal joints. Figure 3 shows a view of the middle finger of the left hand, demonstrating the hypertrophic bony and periosteal changes as well as a small area of decalcification of the middle phalanx. We now strongly suspicioned malignancy with metastasis, and had a readily accessible area for biopsy study. Roentgenographs of the spine, pelvis, skull and other extremities failed to reveal any evidence of further metastasis. Other diagnostic procedures employed were: artificial pneumothoraces, bronchoscopic examinations, fluoroscopic studies, and aspiration of the tumor mass. All gave negative findings. An exploratory needle with a hooked end was used successfully in securing small bits of the tumor mass. An intubator attached to a high frequency current was used in the tract to secure hemostasis.

Course.—Hospitalization extended to approximately five months. The patient's symptoms progressively became worse. Pain and tenderness gradually involved the right shoulder joint and chest. The appetite grew poorer every day. Emaciation was becoming marked. It is remarkable that even in advanced stages the patient had no cough, expectoration, hemoptysis or dyspnea; in fact, the only localizing subjective symptom was intractable pain in the right chest radiating to the shoulder on the same side. The frequent use of opiates was necessitated in the late stages. The affected finger had to be amputated due to enlargement. Metastatic subcutaneous nodules now made their appearance over the right axilla.

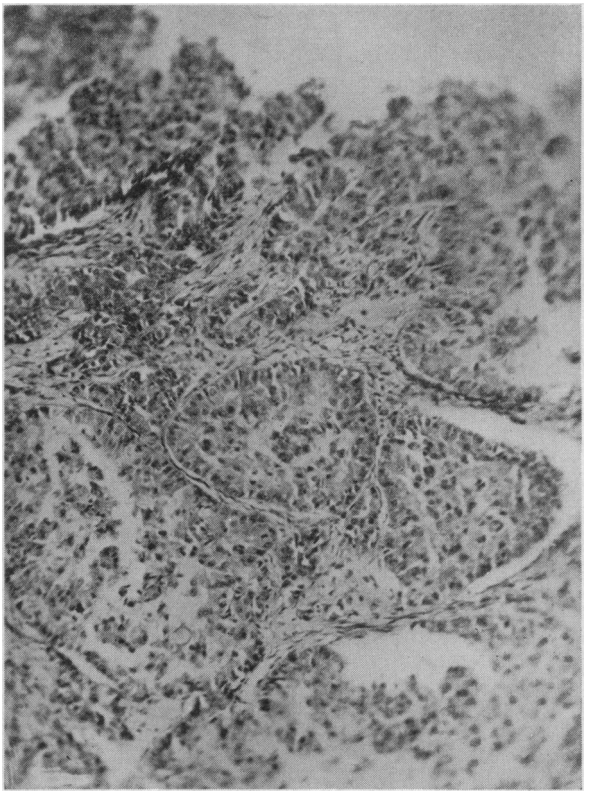

Fig. 5.—Microphotograph of section of biopsy from finger.

The patient died on November 27, 1932, following a progressive loss of weight, cachexia, and marked pain. Surgical interference was not considered, because of the early metastatic phenomena. An autopsy was performed, which verified the clinical diagnosis of a primary carcinoma of the lung.

COMMENT

We are in full accord with M. Fishberg's statement that clubbed fingers appearing in a patient of middle age should urge careful examination of the lungs for tumor.

General Hospital of Fresno.

REFERENCES

1. Fried, B. M.: Arch. Int. Med., 40:340-363 (Sept. 15), 1927.

2. Bonner, L. M.: J. A. M. A., 94:1044-1049 (April 5), 1923.

3. Fishberg: Arch. Int. Med., 37:745-772 (June 15), 1926.

4. Seyfarth: Deutsche Med.-Wchnschr., 50-1491-1499 (Oct. 31), 1924.

5. Fried, B. M.: Arch. Int. Med., 35:1-41 (Jan.), 1925.

6. Brunn, H.: Arch. Surg., 12:406-436 (Jan.), 1926.

7. Polerski, J.: Arch. Int. Med., 48:1126-1137 (Dec.), 1931.

8. Rogers, W. L.: Arch. Int. Med., 49:1058-1077 (June), 1932.