

LUNG ABSCESS AS A COMPLICATION OF SHOCK THERAPIES

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Shock therapy has been accepted today as one of the most successful methods for the treatment of the affective psychoses. The shock may be induced by insulin coma or by convulsions produced either electrically or by leptazol ("cardiazol"). Pulmonary suppuration has been a major complication of insulin coma therapy and, except for vertebral compression fractures, the most frequent sequel of convulsion therapy.

INCIDENCE

INSULIN COMA THERAPY.—From the initial report of Sakel (1934) on the use of insulin until 1938 the aim has been to produce stupor with the smallest dosage and the smallest number of convulsions. Later, longer periods of deeper stupor were induced by increasing the dosage, and convulsions were regarded as a beneficial accompaniment of therapy. At present a course of insulin shock treatment consists of 30 to 60 comas. The duration of maintained coma proposed by different authorities varies from one to four hours. Most authorities terminate coma by administering glucose either orally, intravenously, or by a combination of these routes.

Pulmonary complications, including lung abscesses, pneumonia, pulmonary embolism, and activation of quiescent tuberculous infiltration, have been mentioned in several reports. Delgado, Valega, and Gutierrez-Noriega (1938) were the first to report such complications. They recorded three cases of pulmonary gangrene, one of suppurative pneumonitis, and one of pulmonary oedema in a group of 76 patients treated by insulin shock. In the same year O'Neill published two cases of lung abscess and four of pneumonia which developed in a group of 100 treated patients. Ross (1939) reported three deaths attributed to aspiration pneumonia in a group of 1,356 patients. Kinsey (1941) collected from the literature and from a survey in the United States and Canada 22 cases in which death was attributed to pulmonary complications; three of these were ascribed to lung abscesses, nine to aspiration pneumonia, seven to other types of pneumonia, one to

tuberculosis, and two to miscellaneous causes. Kolb and Vogel (1942) in a statistical report from 300 mental hospitals in the United States estimated the incidence of lung abscess complicating insulin therapy at 0.5 per thousand patients.

LEPTAZOL TREATMENT.—Meduna (1935) first introduced convulsive therapy as a treatment for schizophrenia. Intravenous leptazol was the popular method of inducing convulsions until the introduction of electrically induced seizures by Cerletti and Bini in 1938. Today leptazol treatment has become outmoded in favour of electrically induced shock. Meduna and Friedman (1939) reviewing 3,046 cases treated by leptazol convulsion, found that 0.1% developed abscesses. The same incidence was obtained by Trinch (1939), who reported 13 cases of lung abscess. The survey of Kolb and Vogel (1942) placed the incidence of lung abscesses at 0.5 per thousand with leptazol therapy. Other authors have noted a much higher incidence. Crosa and Viviano (1951) described six cases of lung abscess in a series of 623 patients, i.e., 1%. Polon (1946) observed six cases in 300 patients, an incidence of 2%.

ELECTRICALLY INDUCED SHOCK.—Electrical shock has become the most commonly employed method in shock therapy today. Surprisingly no reports of lung abscesses following electrically induced shock were found in the English literature, and only three reports have been published in the American literature. Kalinowsky and Worthing (1943) first mentioned the occurrence of one lung abscess in a series of 200 cases. Altschule and Tillotson (1945) reported one case. Kwalwasser, Monroe, and Neander (1950), in a review of 2,562 cases treated by electric shock, found 25 cases of pulmonary abscess, i.e., 1%.

Many more cases of lung abscess have been reported in the French and Italian literature. Lalanne (1944) from Algiers reported 46 cases of lung abscess following electrical shock, 40 of them among Arabs. The incidence of pulmonary abscesses among

the Arabs was 15%, the highest figure published. Jaquel (1945) collected 20 cases among 744 patients treated with electrical shock, i.e., 2.6%.

Perret and Nespoulous (1943) and Nespoulous and Constans (1948) reported 12 cases, an incidence of 4.7%. Pons Balmes (1944) in Spain presented a series of 450 patients with seven cases of pulmonary abscess, i.e., 1.6%. Poloni (1946) in Italy published 10 cases of lung abscess with an incidence of 1%. Crosa and Viviano (1951) recorded seven cases in 590 patients, an incidence of 1%. Kultcher and Goldschlager (personal communication) in Israel observed five cases of lung abscess in 1,950 patients treated by electric shock (33,280 treatments), an incidence of 0.4%.

To summarize, we have found records of a total of 250 cases of lung abscess after shock therapy: 33 after insulin treatment, 46 after leptazol, and 171 after electrical shock. The large number following electrically induced shock probably indicates the more frequent use of this method rather than a higher incidence. The present paper reports six cases of lung abscess as a result of shock treatment, two after insulin therapy and four after electrical shock.

CASE REPORTS

CASE 1.—A 58-year-old woman had had involuntional psychosis since 1948. Except for short periods of maniacal overactivity she was fairly cooperative. She was admitted to hospital in April, 1953, and received 43 electrical shock treatments without amelioration of her mental condition. Insulin coma therapy was then started. After the sixth coma she developed fever of 39.0° C. (102.2° F.) and cough, with rales at the base of the right lung. One month later she suddenly expectorated copious foul sputum. A radiograph at this time showed two abscesses in the right middle and lower lobes (Fig. 1). The sputum was negative for acid-fast bacilli. After treatment for three months with penicillin, streptomycin, and chlortetracycline, the pulmonary condition improved and expectoration ceased. The last radiographs showed notable clearing and a zone of fibrosis in the posterior basal segment (Fig. 2). Clinically she is cured.

CASE 2.—A 38-year-old man with paranoid schizophrenia since 1948 was admitted to hospital in February, 1953. One week after insulin therapy, which consisted of nine periods of light and 12 of deep coma, he developed fever and cough and complained of pain in the left thorax. At this time a massive infiltration in the left parahilar region was seen radiographically (Fig. 3). After a sudden copious expectoration of foul sputum a cavity with a fluid level was observed in the apex of the lower lobe (Fig. 4). The patient received 15 million units of penicillin followed by 30 g. of chlortetracycline. After two months the cavity was completely healed.

CASE 3.—A 51-year-old woman suffered her first attack of involuntional psychosis with melancholia

agitans in 1944. In April, 1952, she had an acute relapse. She was admitted to hospital in December, 1952. A first course of 25 electrical shocks was administered which greatly improved her mental condition, but during this treatment she developed a fracture of the neck of the right femur. In July, 1952, the fracture was treated surgically and this precipitated an acute relapse of psychosis. A second course of 26 electrical shocks was administered in August and September, 1952, and a good remission was obtained. This treatment, however, caused a dental fracture and the patient developed extensive suppurative pneumonia in the right upper lobe (Fig. 5). Her symptoms consisted of bouts of fever and a heavy cough with production of 80 to 150 ml. of foul, purulent sputum daily. The radiographs showed a dense almost homogeneous opacity in the entire right upper lobe, and tomography revealed that it consisted of many areas of consolidation situated at different levels. Streptomycin, chlortetracycline, chloramphenicol, and oxytetracycline were of little value, and penicillin had a favourable but transitory influence upon the fever and the quantity and odour of the sputum. Right upper lobectomy was decided upon, but a few days before operation the patient developed an acute psychosis again and the lobectomy was postponed. A new series of electrical shocks improved her mental state, but caused the development of a new abscess in the right lower lobe.

CASE 4.—A 34-year-old woman with catatonic schizophrenia since 1938 was admitted to hospital in February, 1952. In July and August, 1952, a course of 27 electrical shocks was administered without good response. Three months after this treatment she began to expectorate foul sputum. This patient was completely silent and apathetic and made no complaints concerning her chest. Radiographs showed a larger area of pneumonia in the apex and basal posterior segment of the right lower lobe (Fig. 6). The administration of penicillin, streptomycin, and chlortetracycline influenced favourably the clinical symptoms, but the radiographic findings remained unaltered. A right lower lobectomy was performed in September, 1953, by Dr. Milvidsky, and the patient is now well.

CASE 5.—A 39-year-old man with paranoid schizophrenia since 1948 was admitted to hospital in March, 1953. In 1948 he had received seven electrical shocks, after which there was a remission of his psychosis until January, 1953. He received five electrical fits. Nine days after the last he developed a fever of 38.0° C. (100.4° F.). Radiographs showed two abscesses in the apex of the left lower lobe (Fig. 7). After six weeks of treatment with penicillin, streptomycin, and chlortetracycline the condition resolved clinically and radiologically.

CASE 6.—(This case is included by courtesy of Dr. Shlesinger from Shaarei Zedek Hospital, Jerusalem.)

A 45-year-old woman with catatonic psychosis since 1952 was treated in April and May, 1953, by a course of 11 electrical shocks. One day after the last shock she developed a fever of 39.0° C. (102.2° F.). The leucocyte count was 16,000 per c.mm. A radiograph showed an abscess in the apex of the right lower lobe (Fig. 8). After

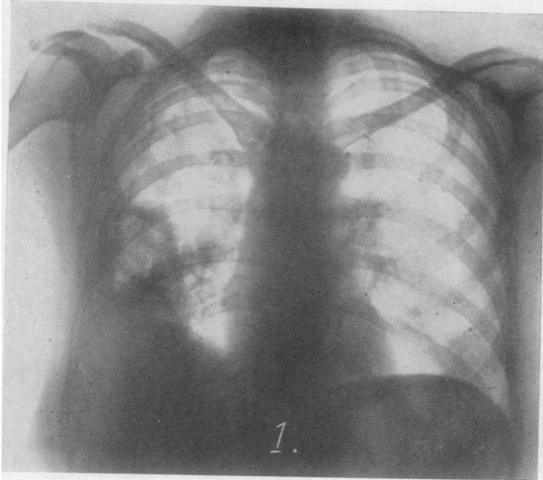


Fig. 1

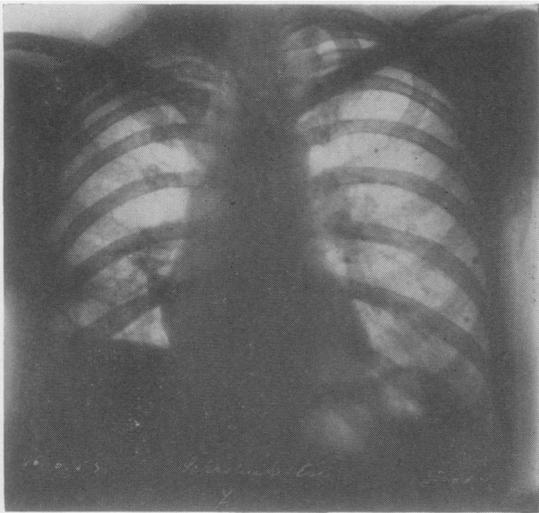


Fig. 2

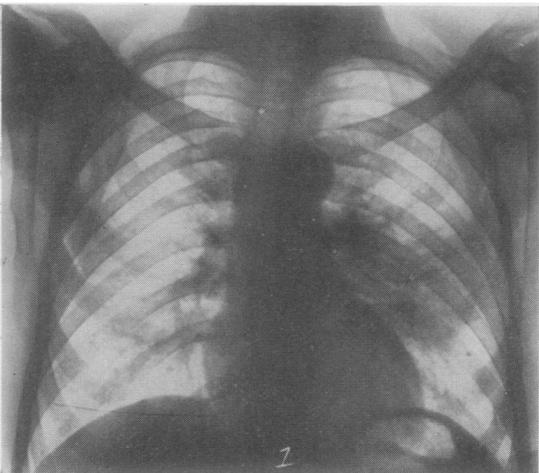


Fig. 3

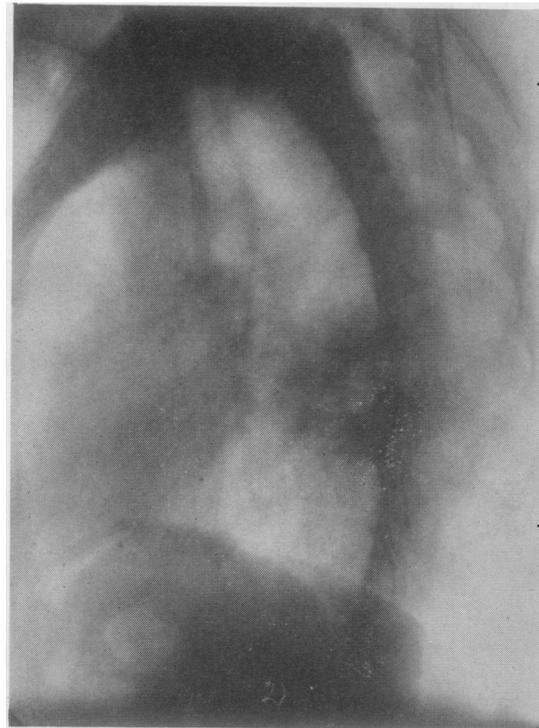


Fig. 4

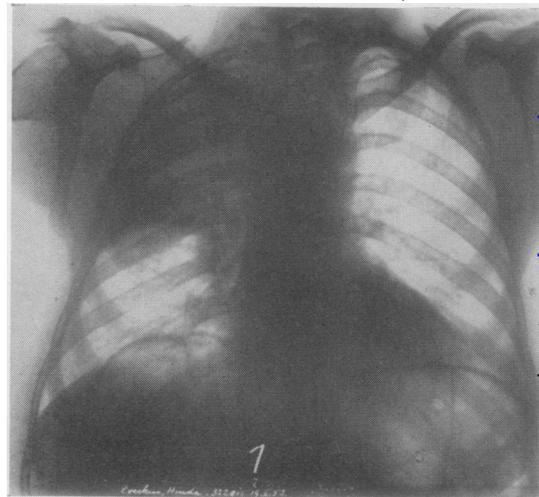


Fig. 5

- FIG. 1.—Case 1: two abscesses in the right lower and middle lobes.
 FIG. 2.—Case 1: zone of fibrosis in the right lower lobe, remnant of previous abscess.
 FIG. 3.—Case 2: abscess in the apex of the left lower lobe.
 FIG. 4.—Case 2: appearance of the cavity following sudden expectoration.
 FIG. 5.—Case 3: suppurative pneumonitis of the right upper lobe.

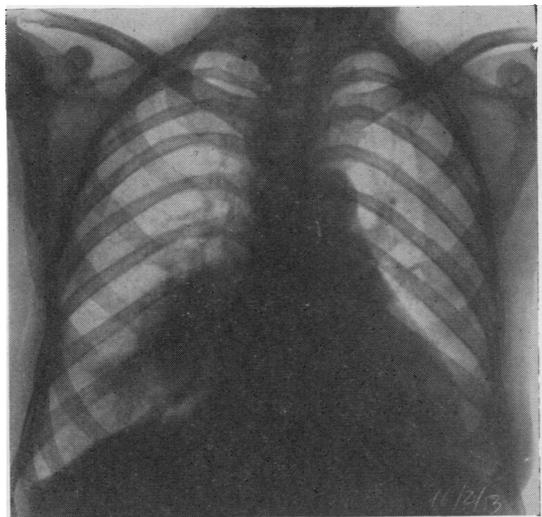


FIG. 6.—Case 4: suppurative pneumonitis of the apex of the right lower lobe.

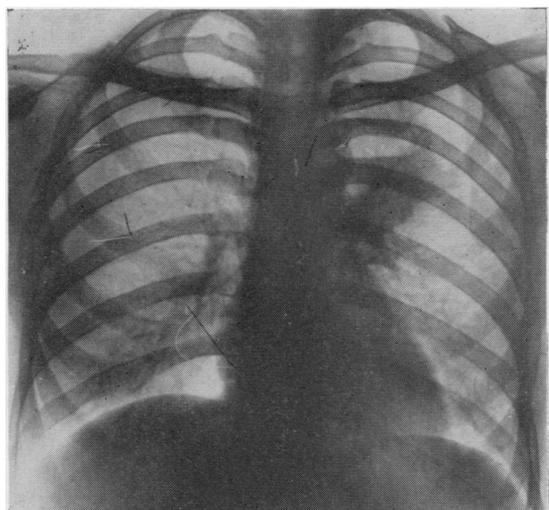


FIG. 7.—Case 5: abscess of the apex of the left lower lobe.

treatment with penicillin, complete radiographic and clinical healing was noted after 40 days.

PATHOGENESIS

A number of theories have been proposed to explain the pathogenesis of pulmonary abscess after shock therapy. Roubier (1951) suggested "pulmonary shock" as a cause, the result of excitation of medullary respiratory and cardiac centres. Altschule and Tillotson (1948) suggested that insulin hypoglycaemia interfered with the physico-chemical

kinetics of cardiac muscle contraction and this led to pulmonary oedema. Poloni (1946) thought that hyperglycaemia, often observed in patients with mental diseases, favoured the development of abscesses, as it does in diabetes. Cerletti's (1941) investigations showed that during the electric shock the glottis was closed, the muscles of the thorax and abdomen were contracted, and there was an increase of intrathoracic pressure. The vagal excitation induced contraction of the bronchiolar muscles and occlusion of the airways. Air was compressed in the alveolar spaces under high pressure, which could cause a rupture of the alveolar walls and minute haemorrhages, with local stasis, thrombosis or necrosis, followed by the formation of an abscess. These investigations were supported by several radiologists who observed in some patients disseminated patches of pulmonary infiltration or irregular consolidations one hour after insulin shock therapy and complete resolution 24 hours later (Breton and Devallet, 1942; Gross and Schaefer, 1950). These phenomena were interpreted as due to vascular stasis or bronchospasm. Other authors (Kalinowski and Worthing, 1943; Conway and Osmond, 1948; Zeifert, 1939) have suggested that the appearance of lung abscess after leptazol treatment was due to septic emboli which arose at the site of the intravenous injection. Zeifert (1939) maintained that lung abscess was not observed when sodium citrate was added to the leptazol solution, but that seven cases appeared when this precaution was not taken. It is obvious, however, that the development of pulmonary supuration after electrical shock therapy is not explicable on this basis.

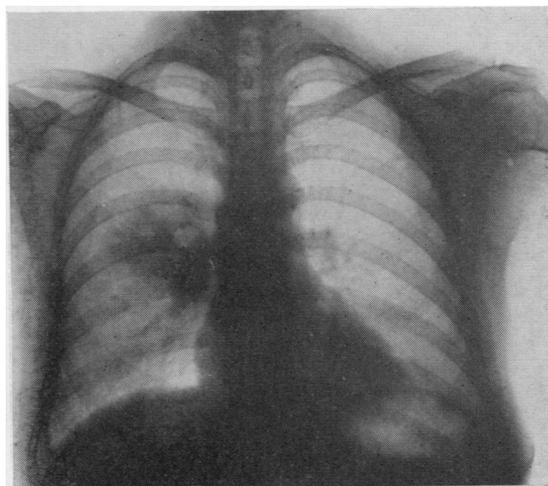


FIG. 8.—Case 6: abscess of the apex of the right lower lobe.

The most likely explanation for the development of lung abscess after shock therapy is that of aspiration. During the stage of tonic convulsions the thoracic muscles are in tetany and the patient is apnoeic. During the clonic stage there is a period of forced expiration. At the beginning of the stage of coma a violent, deep and prolonged inspiration takes place. Salivation is profuse, apparently the result of autonomic stimulation. It is at this last stage that large quantities of saliva with its bacterial contents may enter the lower respiratory tree. With loss of consciousness and depression of the cough reflex, the aspirated material is not removed. The infected saliva occludes the small bronchi and causes atelectasis distal to the occlusion. Atelectasis is an indispensable condition for the development of putrid lung abscess. It has been shown experimentally that anaerobic bacteria in the lung without atelectasis fail to produce the disease (Rubin and Rubin, 1947). Brock (1952) has coined the term "bronchial embolism" for this aspirational process, and it may explain the transient patches or consolidations which are observed one hour after electrical shock. Unconsciousness and convulsions appear both in insulin coma and electrical shock, but not in the same proportion. In insulin coma the patient is unconscious from one to four hours, in convulsive therapy from five to ten minutes only. On the other hand, the violent aspiration at the end of clonic convulsions, which is rare in insulin coma, seems likely to cause more bronchial embolism in electrical shock. Aspiration of food or liquid as a result of intubation or regurgitation of gastric contents may also produce lung abscess. Vomiting frequently accompanies insulin coma and electrical shock. Kwalwasser and others (1950) have reported seven cases of pulmonary suppuration which developed after tracheal intubation during the shock treatment. Aspiration of infected material from the nose, sinuses, throat, tonsils, and particularly the teeth has been emphasized by Nespoulous and Constans (1948) and Delay (1950). Dental hygiene in psychotic patients is often very poor. Brock (1952) has reported that 40 to 80% of the patients in a general hospital who developed lung abscesses had dental abscesses or infections. During the convulsive stage of shock the violent closure of the mandible causes partial fractures of carious teeth and particles of dentine, enamel, or tartar may be aspirated into the lungs. Case 3 of this report developed pulmonary suppuration after dental fracture, and Delay's (1950) patient aspirated a tooth during electrical shock. Opening the mouth to prevent biting the tongue also favours aspiration. A distance of 1 cm. or more between mandible and

maxilla hinders deglutition and permits saliva to enter the lower respiratory passages (Titeca, 1946).

The location of the abscess also indicates that aspiration is the chief cause of pulmonary suppuration. The majority of lung abscesses are monolobar and are found in the right lung, either in the apical segment of the lower lobe or in the posterior segment of the upper lobe. The more direct course of the right main bronchus is responsible for its greater vulnerability to aspiration. Brock has demonstrated that the apical segment of the right lower lobe is most vulnerable when the patient is lying on his back and the posterior segment of the right upper lobe when the patient is on his side. In our six cases, five abscesses were located in the lower lobes and one only in the upper lobe. Of Poloni's 10 cases, nine were located in the apical segment of the right lower lobe. In Lalanne's 46 cases all abscesses were in the lower or middle lobes, but none in the upper lobes. In one case an abscess in the right upper lobe communicated with a much larger cavity in the lower lobe. In Kwalwasser's series, however, the majority of abscesses were located in the upper lobes.

A number of factors have been proposed as predisposing to the development of pulmonary suppuration after shock therapy. Roubier (1951) suggested that the poor physical condition of psychotic patients predisposed to a higher incidence of lung abscess than in the normal population, since a large number of pulmonary suppurations were observed in France during the war years of 1941-1945 when food was restricted. Kwalwasser and others (1950), however, found the incidence of lung abscess to be five times higher in a group of 2,562 psychotic patients receiving shock treatment than in a group of 16,438 psychotic patients without such therapy. These latter authors suggested severity of the mental condition as a predisposing factor. Only three of 25 psychotic patients with lung abscesses were clean and tidy and the remainder were disturbed, over-active, and untidy. Poloni (1946), however, was unable to correlate poor physical and severe mental state with the development of pulmonary suppuration. Some authors have suggested that lung abscess after shock therapy is more prone to appear in older patients (Kwalwasser and others, 1950) and in men (Lalanne, 1944). Lalanne has suggested that there is a racial factor, emphasizing an incidence of 15% in Arab patients as compared with 1% in Europeans. The number of shocks has been suggested as an important predisposing factor (Poloni, 1946). It seems obvious that the more the shocks, the more the chances of developing a lung abscess. In corroboration of this assumption lung abscess has rarely been found complicating

epilepsy in which the convulsive seizures are relatively infrequent (Kwalwasser and others, 1950; Lalanne, 1944; Pons Balmes, 1944). However, no close correlation has been found between the number of shocks and the number of patients developing abscess. In this series the lung abscess appeared after the seventh shock in Case 5, and after the fifty-first in Case 3.

The clinical course of lung abscess in psychotic patients is much the same as that observed in non-psychotic individuals. There are, however, a few minor differences. The onset may be unrecognized for a longer period of time, particularly in those mental patients who are apathetic or do not complain. There have been several cases reported in which the abscess was found four to 12 months after the treatment (Kwalwasser and others, 1950).

The prognosis of pulmonary suppuration in psychotic patients is poor. The mortality is approximately 45%. In Lalanne's series of 46 cases 54% died. Pons Balmes (1944) reported six deaths in seven cases and Kwalwasser and others (1950) five in 25 patients. Of this last series, 10 remained unhealed and 10 were cured. With the advent of the antibiotics and excisional surgery the prognosis has improved noticeably. Of the six cases presented here five were cured, four with antibiotics and one after lobectomy. It is important to use surgical treatment after two to three months if there is no response to antibiotics. The treatment of choice is surgery, which nevertheless still presents problems in psychotic patients, who are difficult to prepare for operation, generally are uncooperative, and difficult to care for post-operatively. Furthermore, a surgical procedure may precipitate a crisis or relapse in a previously quiescent patient.

Prophylactic measures should be directed towards the prevention of aspiration. To accomplish this the following procedures are recommended. (1) Atropine, before shock therapy, should be administered. Suction should be employed in cases of copious salivary secretion during the treatment. (2) The stomach should be empty before treatment is started. If vomiting occurs readily dimenhydrinate ("dramamine") should be administered before the fit. (3) Close attention should be paid to dental hygiene. Caries should be treated, roots and shaky or isolated teeth extracted, tartar removed, and the mouth thoroughly cleaned before instituting therapy. (4) During the shock the patient should be placed on his back and the head inclined to one side. A thin gag should keep the mouth open to prevent tongue

biting and dental fractures. (5) During a series of shocks the temperature should be recorded at least once daily. At the first sign of respiratory symptoms shock therapy should be discontinued and radiographs of the chest be done, as they should before and at the completion of treatment. (6) An adequate nutritional state should be maintained.

SUMMARY

Lung abscess is a major and one of the most frequent complications of shock therapy. The incidence varies in reported series from 0.05 to 2.5%. Abscesses which occur during or shortly after shock therapy are of the putrid type and are due to aspiration of infected material from the upper respiratory passages. Unconsciousness and abolition of the cough reflex are predisposing factors. The infective particles originate mostly from broken carious teeth and from alimentary remains in the mouth. The abscess is located most frequently in the right lower lobe, particularly in its apical segment. Primary resection is the treatment of choice for those cases which do not respond readily to antibiotic treatment. The prophylactic measures aim at preventing aspiration, and a number of effective procedures are recommended.

REFERENCES

- Altschule, M. D., and Tillotson, K. J. (1948). *New Engl. J. Med.* **238**, 113.
- Breton, A., and Devallet, J. (1942). *Bull. Soc. Méd. Nord.* Cited by Roubier, C. (1951).
- Brock, R. C. (1952). *Lung Abscess.* Blackwell, Oxford.
- Cerletti, U. (1941). *Riv. sper. Freniat.*, **65**, 542.
- and Bini, L. (1938). *Boll. Accad. med., Roma*, **64**, 136.
- Conway, H., and Osmond, H. (1948). *J. ment. Sci.*, **94**, 653.
- Crosa, G., and Viviano, M. (1951). *Sistema nerv.*, **3**, 304.
- Delay, J. (1950). *Méthodes biologiques en clinique psychiatrique.* Masson, Paris.
- Delgado, H., Valega, J. F., and Gutierrez-Noriega, C. (1938). *Rev. neuro-psiquiatr.*, **1**, 463.
- Gross, R. J., and Schaefer, F. H. (1950). *Amer. J. Roentgenol.*, **63**, 191.
- Jacquel, M. (1945). Thèse, Nancy. Cited by Roubier, C. (1951).
- Kalinowsky, J. B., and Worthing, H. J. (1943). *Psychiat. Quart.*, **17**, 144.
- Kinsey, J. L. (1941). *Arch. Neurol. Psychiat.*, Chicago, **46**, 55.
- Kolb, L., and Vogel, V. H. (1942). *Amer. J. Psychiat.*, **99**, 90.
- Kultcher, S., and Goldschlager, E. Personal communications.
- Kwalwasser, S., Monroe, R. R., and Neander, J. F. (1950). *Amer. J. Psychiat.*, **106**, 750.
- Lalanne, R. (1944). Thèse, Alger. Cited by Roubier, C. (1951).
- Meduna, L. van (1935). *Psychiat. neurol. Wschr.*, **37**, 317.
- and Friedman, E. (1939). *J. Amer. med. Ass.*, **112**, 501.
- Nespoulous, J., and Constans, J. (1948). 11è Congrès français de Stomatologie, Paris.
- O'Neill, F. J. (1938). *Psychiat. Quart.*, **12**, 455.
- Perret, —, and Nespoulous, J. (1943). *Presse méd.*, **51**, 341.
- Poloni, A. (1946). *Riv. Neurol.*, **16**, 395.
- Pons Balmes, J. (1944). *Med. clin., Barcelona*, **3**, 466.
- Ross, J. R. (1939). *Amer. J. Psychiat.*, **95**, 769.
- Roubier, C. (1951). *J. méd. Lyon*, **32**, 707.
- Rubin, E. H., and Rubin, M. (1947). *Diseases of the Chest.* Saunders, Philadelphia.
- Sakel, M. (1934). *Wien. med. Wschr.*, **84**, 1211.
- Titeca, J. (1946). *J. belge Neurol. Psychiat.*, **44-46**, 52.
- Trinch, G. (1939). *Rass. studi psichiat.*, **28**, 315.
- Zeifert, M. (1939). *Psychiat. Quart.*, **13**, 303.