periments have shown that this is the case, although the effect is not very large. . . .

"A study of the calcium salts of organic acids seemed advisable because of the considerable irritation produced by intramuscular injection of calcium chloride. . . . In our experiments the irritant action of calcium chloride was distinctly noticeable. . . . It must be added, however, that such an inefficiency of calcium gluconate is observed only when it is added to and injected simultaneously with the local anesthetic. If calcium gluconate is injected separately it is not entirely inactive. . . .

"In an attempt to explain the slight anticonvulsive action of calcium gluconate on simultaneous injection it seemed reasonable to assume that the calcium content *per se* was not so important as perhaps the calcium ion content. . . . The conclusion is that calcium gluconate does not contain any calcium ions at all; obviously this is the reason why it completely fails to relieve procaine convulsions. . . .

"This absence of electric conductivity, or of ionization, of calcium gluconate is quite a rare and peculiar property. So far as known today no other calcium salt is completely non-ionized. The other organic calcium salts investigated so far are all ionized completely or nearly so. . . .

"Another extensive experimetal series was carried out, using butyn sulphate in the place of procaine as a local anesthetic. As is well known, butyn is a higher homologue of procaine, differing from it in possessing a butyl group in the place of the ethyl group of procaine and a propanol group in place of the ethanol. Accordingly butyn is much more toxic. It is convulsant in doses as low as 20 mg./kg.: 18 animals were injected; all had convulsions, yet survived. With doses as high as 50 mg. or more, all of the injected animals died, since their convulsions were extremely violent. Nevertheless calcium salts were seen to inhibit these violent butyn convulsions quite efficiently. . . .

"All the anticonvulsive agents mentioned so far act by rendering the tissue membranes or the brain centers less permeable. All these have no effect if injected separately or later. They are inactive after procaine has reached the brain. It is known that barbiturates and other centrally depressing drugs also check procaine convulsions. (Tatum and others.) However, this action, in contrast to that of calcium salts appears only if the barbiturate is administered 15 to 30 minutes before the local anesthetic. It was found that if pentobarbital or phenobarbital is mixed with procaine solutions and injected simultaneously, the incidence of expected convulsions is even higher than if procaine is given alone. . . . If the same amounts of these barbitals were injected half an hour earlier than the procaine, no convulsions appeared at all. . . .

"Another depressing drug was tested, calcium bromide. This was found to act exactly like calcium chloride. The same anticonvulsive action was seen which undoubtedly is due to its calcium content, while the bromine, in spite of depressing effect, plays no part.

"These observations show that the barbiturate or bromide has no effect on cell permeability. Its central depressing action is considerably slower than the central stimulating, or convulsant, action of the local anesthetics." Bibliography—5 references.

J. C. M. C.

E. FRIAS AND F. FERNANDEZ. *Postoperative Plate-like Atelectasis.* Current Researches in Anesth. & Analg. 19: 98–101 (March–April), 1940.

"Aside from infection of the bronchi, aside from massive and lobar

atelectasis and infarction, which are not included in this discussion, we meet with complications of a definite pneumonia type. Among our patients this is the most frequent form of post-operative pulmonary complication. It is in this type of complication that partial atelectasis is of great etiological importance. When we speak of partial atelectasis we refer to plate-like atelectasis. In this study we desire to report the clinical and radiological characteristics of this postoperative complication. . . .

"The signs [of plate-like atelectasis] develop mostly in the base of the lung, hence the greatest frequency of plate-like atelectasis in this situation. In any portion of the base of the lung, extending horizontally and being near the costal region, where the finer bronchi end, there is a respiratory and circulatory disturbance and in this bronchiolar system some mucus accumulates. After a certain time we have blocking of that part of the bronchial system and the air in the alveoli is re-absorbed. Soon that area of the lung does not exchange air and tends to collapse. This is possibly only in one direction — lengthwise — because transversely the parenchyma is supported by the hilus, mediastinum, and by the negative intrathoracic pressure that keeps the lung contiguous to the thoracic wall. . . .

"In massive and lobar atelectasis the collapse or retraction is so great that it even acts on the mediastinum and costal wall, giving a characteristic radiological picture. In plate-like atelectasis the retraction is not sufficient to displace the mediastinum and ribs, these parts remaining in situ. Thus Fleischner explains the formation of plate-like atelectasis and that is the reason he also calls them 'directed lung collapse.' . . .

"One sign is always present in plate-like atelectasis, elevation and reduced movement of the diaphragm. In the majority of our cases, not only has atelectasis been present, but we have also found a pneumo-peritoneum which would seem to be of some importance as an etiological factor.

"It is possible to mistake plate-like atelectasis for incisuritis, it is less likely to confuse the atelectasis with pleural adhesions. But careful radiological study will clarify the diagnosis. . . .

"Plate-like atelectasis is generally an early complication and does not affect the general condition of the patient. The clinical picture is that of one recently operated, with some cough, dyspnea of the expiratory type, slight increase in temperature and moderate tachycardia. In almost all cases there is a seromucoid or mucoid expectoration. This condition lasts three or four days and gradually subsides. In severe cases there is thoracic pain, higher temperature and great dyspnea, cough with mucopurulent expectoration, all of which produces a marked effect on the general condition of the patient. These symptoms indicate that infection is complicating a pure atelectasic process. . . .

"In cases of moderate severity there is slight dullness over the respective bases, diminished breath sounds, bronchial breathing and pectoriloquy. In some cases bronchial râles are heard.

"In more severe cases there is dullness, râles, bronchial breathing, bronchophony and pectoriloquy. This clinical picture lasts for five or six days and then gradually subsides. The last signs to disappear are the râles and the aphonic pectoriloquy.

"The X-ray findings persist for some time and in all of our cases, although there were no clinical signs at the time, X-ray examination revealed the persistence of the atelectasic shadow for fifteen or even thirty days after. . . .

"We have followed the clinical post-operative course of 566 cases, in 17 of which we found plate-like atelactasis

by X-ray examination. Apart from these cases of atelectasis we found a pleuropneumonic process in two others and in still another a mediastinal diaphragmatic pleural process. All cases with pulmonary or clinical signs were subjected to X-ray, as well as a series of over fifty operated patients who had no lung signs. Among the latter we discovered a case of plate-like atelectasis after a cholecystectomy.

"In over 95 per cent. of our cases ether was used as the anesthetic. It was administered with Ombredanne's apparatus or as nitrous oxid-oxygen-ether in open circuit. Only rarely was local or spinal anesthesia employed.

"Of the 17 cases of plate-like atelectasis, 16 followed laparotomy, 8 of which were supra-umbilical and 8 were infra-umbilical. After only one extra-abdominal operation (abscess of the liver) did this complication occur and in this instance the transthoracic method was used with novocain as a local anesthetic."

MOUSEL, L. H.: *Bronchoscopic treatment of postoperative atelectasis.* Proc. Staff Meet. Mayo Clin. **15**: 261–264 (Apr. 24) 1940.

"Atelectasis is perhaps one of the most frequent postoperative pulmonary complications. It is true that the majority of pulmonary complications, as reported in the literature, are cases with bronchopneumonia; however, it is my belief that most cases reported as postoperative bronchopneumonia are actually cases of postoperative atelectasis. Several hypotheses have been advanced as to the etiologic factors involved in producing atelectasis. . . .

"It is my opinion that most postoperative atelectasis is caused by an actual plugging of a bronchus by tenacious mucous secretion which has collected in the tracheobronchial tree during anesthesia, by tenacious mucopurulent material which was present preoperatively, or by mucus, blood or vomitus which has been aspirated into the trachea either during or immediately following anesthesia. The onset of actual atelectasis is probably gradual. The bronchus becomes plugged, causing a preliminary emphysema. If the patient is unable to remove the plug by coughing or change of position, the air in the involved region will be slowly absorbed in the blood stream until collapse is complete. Usually the patient will complain of dyspnea which is frequently out of proportion to the degree of pulmonary involvement. There is usually a sense of discomfort on the side of involvement in association with the atelectasis; the pulse becomes rapid, there is a sudden rise of temperature and cyanosis becomes apparent, the degree of cyanosis depending on the amount of lung tissue involved.

"In massive atelectasis the heart and mediastinal structures are shifted toward the side involved; breath sounds become diminished or absent, and the respiratory excursion on the affected side becomes diminished. The success of bronchoscopic aspiration depends on early recognition of the condition and early treatment, for if the condition is allowed to exist for any length of time mucopurulent material collects in the bronchus distal to the point of obstruction and with secondary infection a true pneumonitis or pulmonary suppuration develops.

"The usual procedure for the treatment of postoperative atelectasis has been the frequent changing of the patient's position in bed in order to encourage gravity drainage, the inhalation of 5 or 10 per cent. carbon dioxide with oxygen to promote deep respiration, and encouraging the patient to cough. While these measures seem to be of benefit in some cases, in many