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THE PREVENTION AND TREATMENT OF ATELECTASIS BY THE CONTROL OF BRONCHIAL SECRETIONS*

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THE POSTOPERATIVE DEVELOPMENT of atelectasis and pneumonitis, despite extensive investigation of its mechanism and treatment, still remains one of the common and serious complications in the surgical patient. Most of the discussions of the subject appearing in the literature concern themselves with the pathology and treatment of atelectasis. A more logical approach has appeared to be an investigation of the basic causes and consequently a better understanding of its prevention.

Postoperative pulmonary atelectasis is produced by the retention of highly viscous secretions which occlude bronchi with subsequent absorption of air from the segment involved.[1-4] It is important to realize that it is viscid respiratory tract fluid which occludes small bronchi or bronchioles. The concept of large branchi being occluded by discrete, almost solid, cork-like plugs—the conception which motivates most attempts at bronchoscopic aspiration in the control of atelectasis—is rarely encountered in actual practice.

Normally the bronchial tree is cleansed by the elaboration of a respiratory tract secretion. This secretion is evacuated continuously by ciliary activity and the expulsive action of bronchial inspiratory and expiratory movement. In the presence of excessive secretions removal is aided by the cough mechanism and lymphatic absorption.[5-11] It is this normal respiratory tract fluid, altered by pathologic changes in secretory physiology, which produces the bronchial occlusion leading to atelectasis. Control of this secretion is consequently the key to the prevention of bronchial blockade.

The bronchial secretion is elaborated by the goblet cells and the bronchial glands. These are mucous, serous and mixed.[12] A variable amount of transudate, together with exudate from the bronchial mucosal capillaries, is contributed in the presence of inflammation.[9]

The goblet cells which elaborate mucus are found interspersed between the ciliated epithelial cells.[12] These cells appear to be activated by local stimulation and not by nervous control.

The bronchial glands are tubuloalveolar in type and secrete both mucin and serous fluid.[9, 12] They are situated in the submucosa and are under nervous control. Vagus as well as direct stimulation activates them and they are inhibited by sympathetic stimulation.

The normal bronchial secretion forms a two-layered covering for the mucosa, the outer layer being a mucus blanket which is normally tenacious and adhesive, yet not extremely viscid. The underlying layer is a serous coating, which because of its more fluid character, enables the entire covering to be moved rapidly along the mucosal sur-

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face.[8, 13, 14] In the presence of chronic irritation, inflammation or allergy, there is an increased production of mucin by the bronchial glands and by increased numbers of goblet cells.[9] This greatly increases the viscosity and adhesiveness of the covering secretion. Buhrmester[15] has shown that when the mucin content (normally 2-3 per cent in nasal secretions) is increased above 3 to 5 per cent, there is a sharp rise in viscosity (Fig. 1).

Ciliary activity is a most important mechanism in moving the secretory covering, and hence in cleansing the tracheobronchial tree[16-20] The role of the ciliated epithelium is most important in cleansing the smaller bronchi and bronchioles. These ciliated cells extend outward as far as the respiratory bronchioles, which are lined with non-ciliated cuboidal epithelium.[12] The cilia are autonomous and propel the coating of mucus by a concerted wave action.[8, 13, 14] Lucas and Douglas[8] showed that the cilia moved in the underlying layer of low viscosity, and that only the tips of the cilia dip into the overlying tenacious strands of mucin. Thus, alterations in the mucin content such as occur with dehydration, inflammation or allergy greatly alter the efficiency of ciliary propulsion. When there is an excess of mucin, the cilia become entangled, and the basis for subsequent atelectasis is prepared. It would thus appear that clearance of the bronchial tree is dependent upon the maintenance of normal bronchial secretory activity.

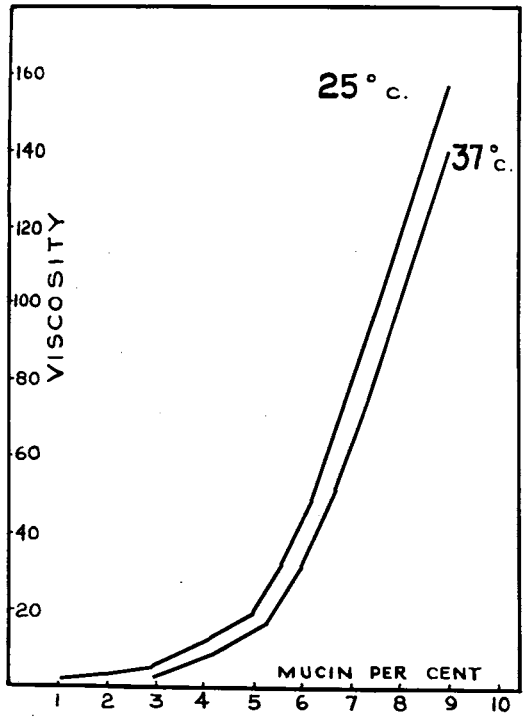


Fig. 1

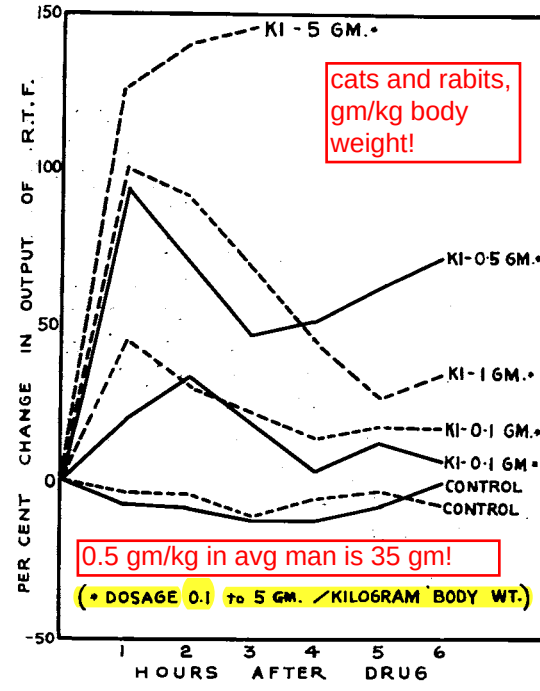


Fig. 2

Fig. 1.—Illustrating the relation between viscosity of mucin and its hydration. (After Buhrmester, Catherine C.[15], *Nasal Mucin.* Ann. Otol. Rhin. and Laryng., **45**: 687, 1936.)

Fig. 2.—The effect of potassium iodide upon the output of respiratory tract fluid (tracheobronchial secretion) in cats and rabbits. (After Boyd, E. M. and associates,[27] *The Effect of Inorganic and Organic Iodides Upon the Output of Respiratory Tract Secretion.* Can. J. Research, **23**: 195, 1945.)

The expulsive action of bronchial respiratory movements appears of secondary importance. The cough mechanism becomes necessary when there is a derangement in the secretory physiology. It appears most effective in the larger bronchi.[10, 19, 21-25]

FIG. 3.—Rabbit bronchus—lead nitrate precipitate, counterstain methylene blue. At the end of five minutes the greatest content of iodide is seen in the blood vessel lumen in the submucosal layer. The iodide is also scattered diffusely through the tissues in low concentration. A somewhat higher concentration is seen in the mucosa. The cartilage is stained an intense blue and in the photograph appears black. (Magnification 100x).

Postoperative atelectasis occurs in individuals who have an altered bronchial secretion which is highly viscid and contains increased mucin. Such factors as asthma, upper respiratory infection, chronic bronchitis and bronchiectasis stimulate an increased production of mucin. In the presence of allergy, chronic irritation and chronic infection, a metaplasia of the ciliated epithelium to squamous cells may occur.[9, 20]

When the basic cause of atelectasis is considered from the foregoing standpoint of deranged bronchial secretory physiology, it becomes of practical clinical importance to find a method whereby one can produce a more physiologic bronchial secretion in which the cilia may beat, since they play such a significant role in evacuating the small bronchi and bronchioles.

Holinger, Basch, and Poncher[26] found that in patients with bronchiectasis the mode of action of the commonly used expectorants, including potassium iodide, ammonium chloride, emetine chloride and steam, was by lowering the viscosity of the bronchial secretion.

In our search for an agent to prevent the development of postoperative atelectasis and to facilitate the evacuation of viscid secretions formed during anesthesia, our attention was focused on sodium iodide because of its ease of administration and since we noted the definite resultant stimulation of the bronchial secretion during direct examination by bronchoscopy. Moreover, the secretion produced was easily and rapidly eliminated from the bronchial tree.

Iodides have been used empirically as expectorants for many years, particularly in those diseases of the respiratory tract associated with viscous, tenacious tracheobronchial secretions such as occur in bronchial asthma and bronchiectasis. There remains, however, a difference of opinion as to the effectiveness of iodide as an expectorant, and its mode of action.

Two theories have been advanced regarding the mechanism of expectorant action of the iodides. E. M. Boyd and associates[27] have offered evidence that iodides act by a vagus reflex mechanism initiated in the stomach. They were able to show an increase in respiratory tract fluid output in anesthetized cats and rabbits after the administration of potassium iodide by stomach tube. This could not be demonstrated

what dose?

after section of the anterior and posterior gastric branches of the vagus.

Stieglitz[28] first showed by histochemical methods that iodide localizes in the epithelial cells of the bronchial mucosa subsequent to its administration. Ariel and his co-workers,[29] working with radioactive iodine, found that next to the thyroid gland the lungs collect most of the radioactive iodine injected. This has been verified in

FIG. 4.—The mucosa and blood vessel shown in Fig. 3. (Magnification 350x.)

our clinic by Lein.[30] Tuft and Levin[31] demonstrated in patients that iodides given either orally or intravenously are selectively excreted into the bronchi within 15 to 20 minutes. They regard this as accounting for the action of iodides in increasing the respiratory tract output of fluid. However, they were unable to determine if the bronchial secretion was increased. Boyd[27] believes that the excretion of iodides might explain lowering of the viscosity of respiratory tract fluid noted by Basch and Holinger, yet that the increased fluid output is resultant to vagus stimulation.

both increased output (vagus) AND less viscocity (iodine)

The effectiveness of iodides as expectorants, whether they act by increasing the volume or merely by lowering the fluid viscosity, is still not agreed. Clinically, Alstead[32] could demonstrate no benefit from potossium iodide either in regard to volume or fluidity in patients with chronic bronchitis. Basch and Holinger[26] found that potassium iodide administration decreased the viscosity of sputum of patients with bronchiectasis, but report no change in volume. Gordonoff[33] on the other hand, presented indirect evidence that iodides produced an increased volume of secretion. He noted in lipiodol bronchograms that the administration of iodides produced a dilution of the lipiodol and diminution of the shadow. Boyd[27] showed a great increase in respiratory tract fluid subsequent to large dosages of potassium iodide in animals (Fig. 2).

TABLE I.

Patient	Diagnosis	Dosage Sodium Iodide (Grams)	Time for Appearance of Secretion (Minutes)	Amount of Increased Respiratory Tract Secretion
F.G.	Tbc. bronchiectasis	0.5	1–2	0
V.S.	Normal	0.6	3.0	0
P.K.	Tbc. bronchiectasis	1.0	10.0	3+
F.P.	Normal	1.0	10.0	4+
J.M.	Normal	1.0	10.0	2+
F.G.	Tbc. bronchiectasis	1.0	10.0	0

Thus, except for the work of Holinger, Basch and Poncher, the direct evidence of iodide expectorant action has largely been based on animal experimentation. Tuft and Levin demonstrated in patients that iodides given orally or intravenously were selectively excreted in the tracheobronchial tree, but they did not report any increase in bronchial secretion. Empirically, the drug has long been used with good effect in asthma and bronchitis, yet controlled clinical observation for the most part has thus far not substantiated its effectiveness. Therefore, it was proposed *first*, to demonstrate by direct observation in man whether or not iodides have a bronchial secretory effect, and then to determine the site of action in man as well as in experimental animals.

1) secretory?
2) site of action?

To determine the effectiveness of sodium iodide as an expectorant, patients seen in the bronchoscopy clinic were divided into two groups. Group One (nine patients) were given 1 Gm. of sodium iodide intravenously. The bronchoscope was then introduced five to ten minutes later for direct observation of its effect upon the bronchial mucosa. After ten to 20 minutes occasionally a patient of this group exhibited a great increase in bronchial secretion that lasted for two or three minutes (Table I). The amount given was then increased to 2 Gm. since the earlier results were inconstant. Group Two (eight patients) received this amount. The bronchoscope was then introduced after seven to ten minutes. All but one of eight patients exhibited a great increase in bronchial secretion in from eight to 15 minutes, lasting three to four minutes.

To determine whether or not iodides are selectively secreted by the bronchial mucosa and bronchial glands, the bronchial secretion was aspirated bronchoscopically. At the time of appearance of the secretion a blood sample was drawn simultaneously. The iodide content of the branchial secretion and the blood were then determined and compared (Table II). The iodine content was determined after the method of Mathews, Curtis and Brode.[34] It will be noted that in each patient of Group Two except one, there was a much greater iodine content in the bronchial secretion than in the blood. The average iodine value for the bronchial secretions was 76.3 mg. per 100 cc.; whereas the blood iodide values averaged only 7.7 mg. per 100 cc. The normal whole blood iodine is 4.2 micrograms per 100 cc.[35] The bronchial secretion aspirated from a normal individual had an iodine content of 4.0 micrograms per 100 cc. This investigation thus demonstrated the selective secretion of iodide by the bronchial tree.

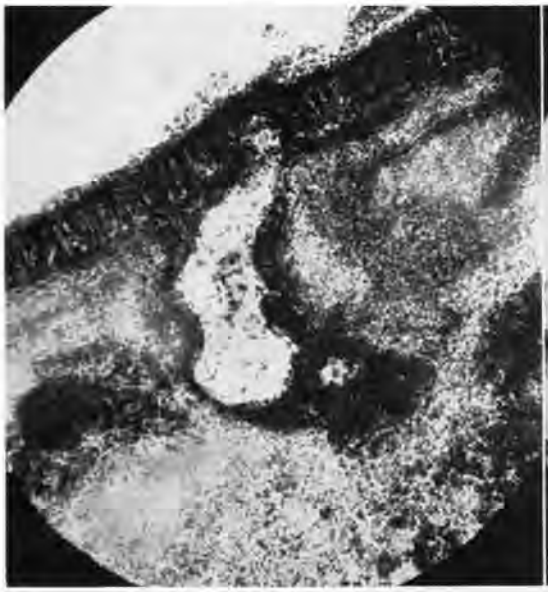


FIG. 5

FIG. 6

FIG. 5.—Twenty minutes post-injection, showing the small amount of iodide secreted into the lumen of a bronchial gland duct. Note the larger amount of iodide distributed throughout the mucosal layer. The dark areas in the submucosa are blue staining bronchial glands. (Magnification 260x.)

FIG. 6.—At the end of 30 minutes the concentration of iodide in the mucosal cells has shifted from the basal to the outer portion. (Magnification 130x.)

4.2 mcg/100 ml =0.042 ppm
76 mg/100 ml = 7.6 %=76,000 ppm

1Mg% = 1mg/dL = 10mg/L = 10 ppm

Thus, by simultaneous quantitative measurement of the blood and bronchial secretion iodine, we were able to demonstrate that there is active iodide excretion accompanying the great increase in the bronchial secretion. This in part confirmed the work of Tuft and Levin, who used qualitative methods of iodine detection. We regard this as evidence that the expectorant action of iodides is either by direct stimulation of the bronchial glands or by an alteration of the cellular permeability of the bronchial mucosa, and not as a result of vagus stimulation as maintained by Boyd and associates.

TABLE II.

Patient	Diagnosis	Dose (Gms.)	Bl. I, Sample Taken at Time of Aspiration (Mg. %)	Time for Appearance of Secretion (Minutes)	Amount of Respiratory Tract Secretion	Iodide Content of Secretion (Mg. %)
Group I						
G.S.	Bronchiectasis	1	8.2	20–30	None	6.78
P.M.	Carcinoma, bronch.	1	7.44	25	2 (bloody)	3.01
Group II						
L.N.	Hemoptysis	2	8.8	15	2+	33.3
R.K.	Carcinoma, l.l.	2	7.1	10	4+	14.1
R.B.	Pulm. Tbc.	2	10.8	10	2+	12.3
L.N.	Suppur. bronch.	2	4.7	15	3+	370.0
E.J.	Bronchiectasis	2	6.7	15	4+	62.8
T.E.	Carcinoma, r. l.	2	9.0	8	0	23.8
M.G.	Bronchiectasis	2	8.3	15	2+	44.8
L.C.	Bronchiectasis	2	6.4	11	4+	49.5
Control						
E.B.	Normal		(Bronchial aspiration without NaI)			4.0 mcg.

mg/ 100 ml

mcg/ 100 ml

TABLE III.—*Eleven Patients With Postoperative Atelectasis Treated with Sodium Iodide Intravenously.*

Surgery	Number of Cases	Time Elapsed to Clear Atelectasis
Lobectomy for bronchiectasis	9	24 hours
Cholecystectomy	1	24 hours
Exploratory thoracotomy for inoperable bronchogenic carcinoma	1	48 hours

The site of action of iodides was then studied by a modification of the histochemical technic used by Stieglitz.[28] Rabbits averaging about 2 Kg. received 10 cc. of 10 per cent sodium iodide by injection into an ear vein. Following this, at intervals of five, ten, 20, 30 and 40 minutes, a 5 per cent solution of lead nitrate was injected into the ear vein. Shortly thereafter the rabbit developed convulsions and lost consciousness. The chest was opened immediately. Two hundred cc. of lead nitrate was then injected into the apex of the left ventricle, using a large bore needle inserted near the mitral valve. The injection was continued until cessation of the heart beat. Portions of the bronchial wall and lung were then fixed in 10 per cent formalin. The tisues were washed overnight. Frozen sections were prepared, stained with methylene blue and mounted in Glycogel. Iodide reacts with lead nitrate to form a yellow precipitate of lead iodide in the tissues. Depending upon the amount of blue stain in the areas examined and the concentration of lead iodide, the lead iodide granules in transmitted light varied in appearance from yellow through green to almost black.

At the end of five minutes, it is seen, large amounts of iodide are present in the blood vessels, some is seen in the tissue, a moderate quantity is seen in the mucosal cells and just under them, and in the lumen of the trachea (Figs. 3 and 4). In addition, a small amount is present in the perichondrium. After ten minutes the concentration of iodide in the bronchial mucosa, glands

and submucosa was maximal. The amount seen in the blood vessels was somewhat less. A progressive diminution in the concentration of iodide in the various structures was seen at 20, 30 and 40 minutes (Figs. 5, 6 and 7). In the mucosal cells, site of the greatest concentrations, the iodide shifts from a basal to an apical position during this time. Bronchial glands were never seen to contain iodide in the amounts visible within the bronchial mucosa.

In view of the foregoing experimental findings, sodium iodide may be considered a specific therapeutic agent in the control of bronchial secretions. It was therefore employed in the treatment of 11 patients who had developed atelectasis postoperatively. These patients had been given pre- and postoperative penicillin and streptomycin as well as preoperative aerosol penicillin and streptomycin. One to 2 Gm. of sodium iodide were given intravenously twice daily up to three or four days after the onset of the atelectasis. The patients were ambulated early. Coughing was encouraged frequently. Under this management ten of the group cleared the atelectasis within 24 hours. The remaining patient cleared within 48 hours without resorting to bronchoscopic aspiration. Nine had undergone surgery for bronchiectasis, one had an exploratory thoracotomy for an inoperable bronchogenic carcinoma, and one had an atelectasis following cholecystectomy (Table III).

TABLE IV.—*One Hundred Consecutive Unselected Surgical Patients Treated With Sodium Iodide Postoperatively Without Incidence of Atelectasis.*

Bilateral saphenous ligation with vein stripping	2
Cavernostomy (thoracotomy)	2
Small bowel obstruction	2
Radical mastectomy	2
Cholecystectomy	4
Thoracoplasty	19
Exploratory thoracotomy	3
Exploratory laparotomy	3
Thyroidectomy	4
Radical neck dissection	1
Esophagogastrectomy	3
Bilateral anterior scalenotomy	1
Major limb amputation	4
Lobectomy	3
Ovarian cyst	3
Skin grafting (major)	7
Vagotomy	1
Herniorrhaphy	6
Hysterectomy	4
Appendectomy	9
Colostomy	3
Pneumonectomy	7
Transverse colectomy	1
Hydrocele	1
Gastric resection	3
Simple mastectomy	2

The following case reports are illustrative of the clinical use and effectiveness of the iodides in patients who have altered bronchial secretions.

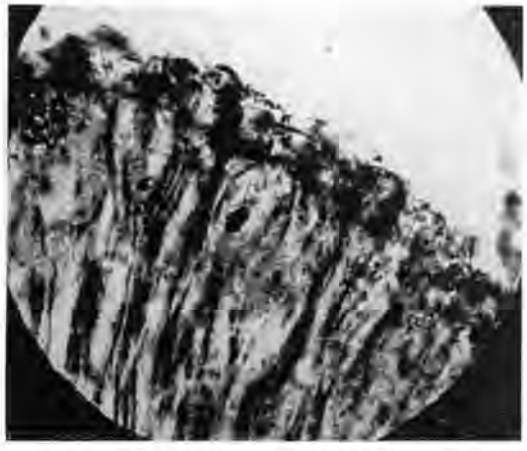


FIG. 7.—A magnification of the mucosal layer shown in Fig. 5 presents the grossly irregular dark masses and granules concentrated in the outer portion of cells, between cells, and along the free border of the cells. (Magnification 550x.)

Case 1.—D. W. (496898 A) entered the University Hospital with acute follicular tonsillitis, October 10, 1949. He was known to have bronchiectasis of the right lower and middle lobes since 1946. He was treated with penicillin for five days. His tonsillitis subsided rapidly. Alpha streptococcus was cultured from his pharynx.

On October 19, 1949, the patient was transferred to the research surgical service for lobectomy. Preoperatively he was placed on postural drainage, aerosol penicillin and streptomycin, and duracillin 200,000 units daily, together with streptomycin 0.5 Gm. twice daily. On October 21 a right middle and lower lobectomy was accomplished without incident. Postoperatively, he received penicillin, 100,000 units every 3 hours, and

FIG. 8

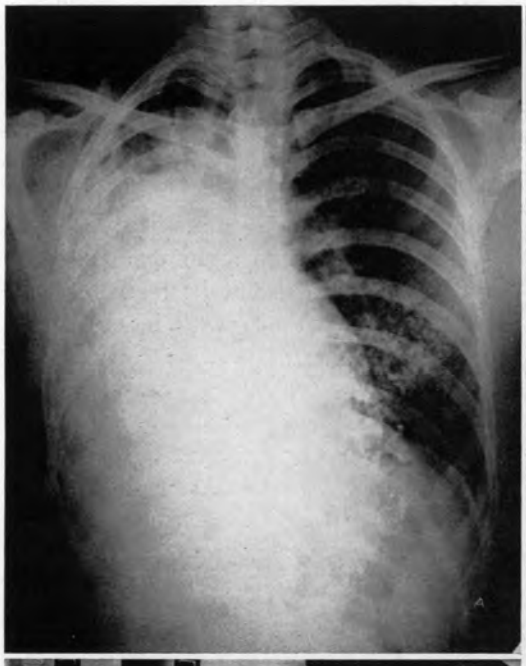
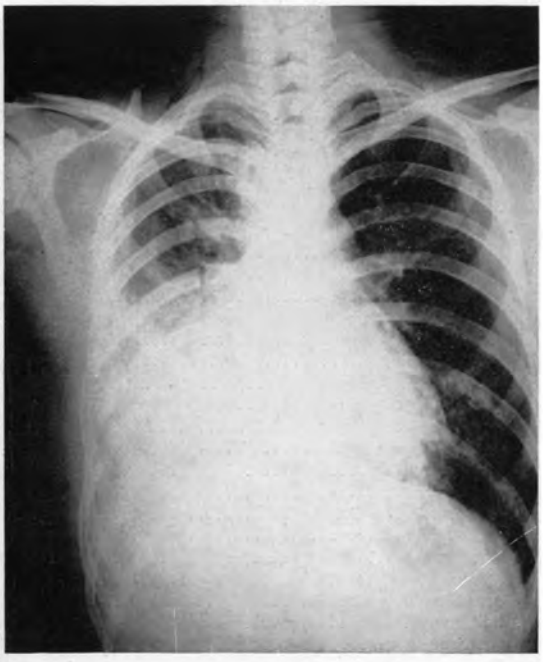


FIG. 9



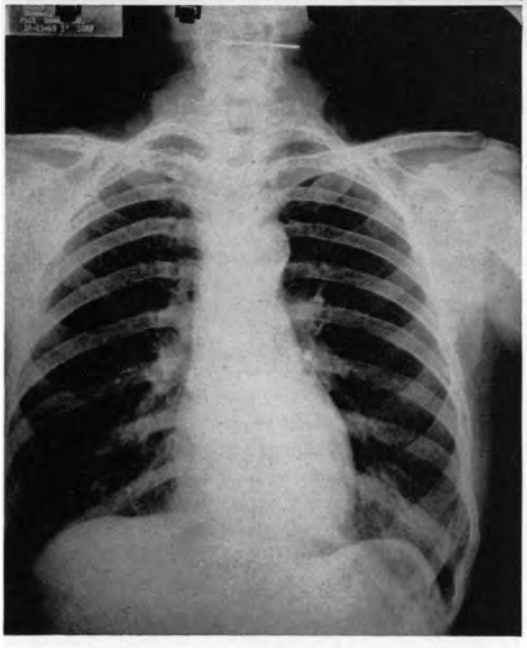


FIG. 10

FIG. 8. — Roentgen ray of D. W., 496898A, taken 48 hours after a right lower lobectomy. Note the development of atelectasis in the upper right lobe with shift of the trachea to the right and pleural effusion.

FIG. 9. — Roentgenogram of D. W., 496898A, taken 72 hours postoperatively. Note clearing of the atelectasis and that the trachea has shifted back to the midline. The diaphragm remains elevated on the right due to crushing of the phrenic nerve at surgery.

FIG. 10.—Preoperative roentgenogram of Mrs. M. B., 497281A.

streptomycin, 0.5 Gm. every 6 hours. He was ambulatory on the first postoperative day. On the second postoperative day he developed massive atelectasis of the right upper lobe with considerable shift of the trachea to the right, together with elevation of the right diaphragm and pleural effusion. His temperature was elevated to 101°, with a respiratory rate of 30 and a pulse of 140. He was given sodium iodide, 1 Gm. twice daily, morning and afternoon, intravenously. Immediately after the iodide administration he began to cough

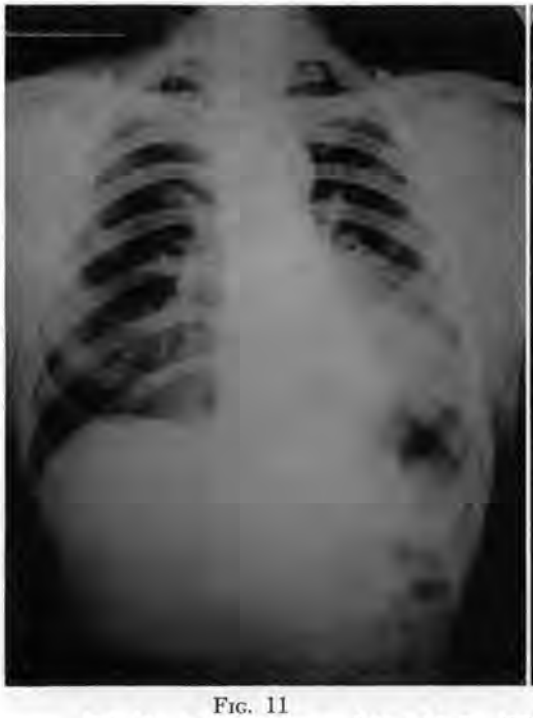


FIG. 11

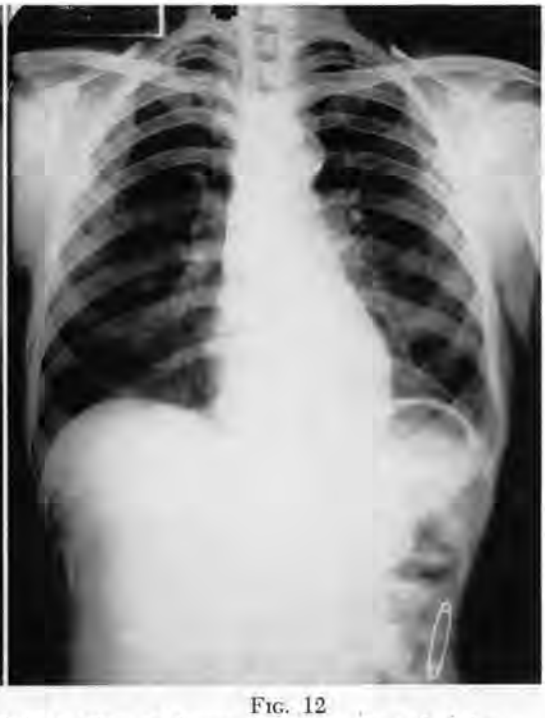


FIG. 12

FIG. 11.—Roentgenogram of Mrs. M. B., 497281A, taken 24 hours after the onset of atelectasis on the third postoperative day following cholecystectomy. Note the left lower lobe atelectasis with an elevated left diaphragm.

FIG. 12.—This roentgenogram of Mrs. M. B. was taken on the fourth postoperative day, approximately 12 to 15 hours after the administration of 1 Gm. of sodium iodide intravenously. Note the marked and rapid clearing of the atelectasis.

up with ease a large amount of viscid, dark mucoid sputum and continued to do so during the next 2 days. Coughing was encouraged. In 12 hours the physical signs of atelectasis had cleared. A roentgen ray taken in 24 hours revealed re-expansion of the lung. The temperature gradually subsided and was normal by the fifth postoperative day (Figs. 8 and 9).

Case 2.—M. B. (497281 A), a woman of 54 years, entered the University Hospital October 23, 1949, with chronic cholecystitis and cholelithiasis, symptomatic for 5 years. On October 29, 1949, a cholecystectomy was accomplished and a large stone removed from the cystic duct. Postoperatively the patient was placed on aerosol benzoin-penicillin inhalations (5000 units of penicillin per cc.) every half hour. Despite this on the second day postoperatively she developed a persistent fever up to 103°, with the physical signs and roentgen ray evidence of an atelectasis of the left lower lobe (Figs. 10 and 11). She was placed on penicillin, 100,000 units every 6 hours. In 12 hours, the patient's temperature was normal only to rise to 103° in 3 hours. She complained of shortness of breath. It was noted that despite a deep cough the patient was unable to raise sputum. On the third postoperative day, October 31, 24 hours after the onset of the atelectasis the temperature was 102° and the signs of atelectasis persisted. At this time the patient was given 1 Gm. of sodium iodide intravenously. Coughing was stimulated but only a small amount of mucus was obtained. The following morning the patient was coughing and expectorating freely. The temperature was normal 15 hours after injection of the sodium iodide and roentgen rays of the chest showed clearing of the atelectasis (Fig. 12).

The next step in the clinical use of sodium iodide was the formulation of a regimen for the prevention of postoperative atelectasis. Regulation of the fluids so as to maintain optimum viscosity and thus prevent occlusion can be achieved by the daily administration of sodium iodide intravenously. We have finally adjusted our dosage to a routine administration of 1 Gm. of sodium iodide intravenously twice daily. The first injection is made during the late afternoon or evening of the day of operation. Subsequent injections are made early in the morning and in the late afternoon. This is continued for three or four days postoperatively. The patients are encouraged to cough 15 minutes after the administration of the sodium iodide, since at this point the maximum secretory effect occurs. Patients undergoing pulmonary surgery are also placed on preoperative aerosol and parenteral penicillin and streptomycin. This program has proved unusually effective in the prevention of postoperative atelectasis.

A series of 100 unselected, consecutive surgical patients were placed on this prophylactic regimen postoperatively. Not one developed atelectasis or pneumonia (Table IV). There were no instances of iodine sensitivity. Various types of surgery are represented. All patients receive general anesthesia. Continuation of this regimen after our first series has yielded equally gratifying results.

DISCUSSION

The experimental data presented demonstrate that iodide given intravenously concentrates in the mucosa and submucosa of the bronchi and bronchioles and then rapidly passes into the bronchial lumen. This is a rapid physiochemical process, as evidenced by the high iodine content of the bronchial secretion, which is almost ten times greater than that of the blood. The maximum concentration of iodide in the bronchial mucosa occurs in ten minutes by histochemical methods. There is a great increase in fluid volume, which occurs as the maximum concentration of iodide passes into the bronchial lumen. The actual volume increase in the newly formed respiratory tract fluid could not be measured accurately because of technical difficulties. However, the sudden increase in bronchial secretion was unmistakable and grossly visible by bronchoscopy, lasting for several minutes. This is in accord with Boyd's experiments with cats and rabbits. He used much greater dosages of potassium iodide (0.1 to 1.5 Gm. per kilogram of body weight by stomach tube) and the volume of the respiratory tract fluid increased from 25 to 150 per cent (Fig. 2).

Iodides are known to lower the viscosity of sputum. This signifies a process of hydration. Since by histochemical methods the iodides are seen to pass rapidly across the respiratory tract mucosa, a process which would carry water across any semi-permeable membrane by osmotic action, a likely mechanism of secretory activity is in transudation of serous fluid into the bronchial tree. This directly provides the cilia with a serous fluid layer in which to beat, and again they can become more effective. Decreasing the viscosity of the secretion aids the cough mechanism, and evacuation by respiratory movements. The greatest secretory response obtained was apparently due to the large amount of iodide given.

The empirical use of iodides as expectorants over a period of years is evidence that they possess a certain value in thinning tracheobronchial secretions. The routine administration has been to give relatively small amounts of potassium iodide or syrup of hydriodic acid orally, repeating at three- or four-hour intervals, and maintaining this medication over a period of several days. In the light of our experimental studies, however, we feel that the true effectiveness of iodides as expectorants has been substantiated. We are of the opinion, however,

that in the treatment of unusually viscid bronchial secretions and particularly in the prevention of atelectasis in the postoperative thoracic surgery patient, larger doses administered by the intravenous route are indicated. We feel that the profuse flushing of the bronchial tree twice daily, resultant to larger intravenous dosage, is of greater value than the lesser lowering of viscosity resulting from repeated oral dosage. In a controlled clinical study of 100 consecutive surgical cases we have been able to demonstrate and report the effectiveness of intravenous sodium iodide in the prevention of postoperative atelectasis.

CONCLUSION

Intravenous sodium iodide has been shown a specific therapeutic agent to control bronchial secretory physiology. The surgeon consequently may confidently use it to prevent the development of the distressing complications of atelectasis and the resultant pneumonitis.

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Dr. Alton Ochsner, New Orleans: This presentation is certainly a valuable one, and should, it seems to me, do a great deal of good. I would like to emphasize the necessity, in many of these instances, in which there is impending atelectasis, of the performance of a tracheotomy. I am sure, in the past, we as surgeons, have used tracheotomy not frequently enough.

Recently, Dr. Lahey has called attention to the necessity of tracheotomy in patients having difficulty following thyroidectomy. Our neurosurgeons are doing them in all comatose patients, and it is particularly valuable in patients who have suppurative diseases of their pulmonary tree because only by tracheotomy can one satisfactorily perform a tracheobronchial toilet by the tracheal aspiration.

In many of these patients, I am sure, the difficulty can be prevented by the use of the expectorant which Dr. Baker has emphasized but, if there is difficulty in getting rid of the secretions which so many patients have who are critically ill, the performance of a tracheotomy early, before irreparable damage has occurred, frequently will be lifesaving.

Dr. Wilder Penfield, Montreal: I would like to ask Dr. Baker a question. A problem which neurosurgeons face often is the increasing consistency of the bronchial secretions when there is pulmonary involvement, either due to increase of intracranial pressure or to some other abnormality that affects the brain stem. The bronchial secretions are stringy, and add just one more difficulty to the cycle of difficulties which arise as a result of bulbar involvement. I would like to know his experience in regard to that type of case, with the use of this expectorant.

Dr. John H. Gibbon, Jr., Philadelphia: I think the authors of the paper and the presenter ought to be congratulated upon a very excellent clinical pharmacologic study. I would like to ask two questions. How frequently was nasal-tracheal aspiration with a catheter carried out in these cases? Also, with the use of the expectorants they advise, was the need for nasal-tracheal aspiration increased or decreasd?

Dr. Baker (closing): In answer to the question by Dr. Penfield, we have had no experience in the use of this drug in the treatment of bronchial secretions of increasing consistency in neurosurgical patients. We did not use intratracheal aspiration in conjunction with the sodium iodide. However, I think it would be a valuable addition to this method of prevention of atelectasis. The sodium iodide, however, should be given first on the evening following the surgery, after the patient has fully reacted, and is able to cough without difficulty.