THORACOPLASTY AND CONTRALATERAL PNEUMOTHORAX

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In certain instances of bilateral pulmonary tuberculosis it is a frequent practice to recommend induction of an artificial pneumothorax (A.P.) to control disease on one side when indications for thoracoplasty on the other side are clear cut. A number of writers over the last 16 years have pointed out the extra risk and added danger of operating with a contralateral A.P. present, but have not specified the nature of this increased hazard except to reaffirm the frequency of spontaneous pneumothorax on the contralateral side. The purpose of this paper is to report a series of patients in whom spontaneous pneumothorax did not occur, to make alternative suggestions to account for the changes which undoubtedly do occur on the side of the A.P., and to state reasons for believing that the A.P. is best dispensed with. The paper is based on observations made during and after operations.

This study in patients with contralateral A.P. undergoing thoracoplasty was provided by the alleged occurrence of spontaneous pneumothorax on the contralateral (A.P.) side in three consecutive patients during the first 24 hours after the first stage of the operation. It is my contention that there is no increased likelihood of spontaneous pneumothorax following thoracoplasty compared with its occurrence during the course of A.P. therapy alone, or at any rate of spontaneous pneumothorax as at present defined and recognized. However, before considering the somewhat complex nature of the changes that the contralateral lung may undergo, the composition of the clinical material must be described.

CASES UNDER CONSIDERATION

Out of 280 patients undergoing thoracoplasty at the Country Branch of the London Chest Hospital in 16 months up to December, 1950, 17 had contralateral pneumothoraces at the time of operation. There were no operative deaths in these 280 cases; that is to say, all patients were discharged from the hospital. The 17 patients with A.P. will be considered, but only in those aspects of their condition relevant to the present thesis. There is, therefore, no necessity to submit the indications for which the A.P. was induced or the thoracoplasty performed except in the case reports which follow. In general it can be said, as far as one can draw any conclusion from so small a series, that the nature of the disease was of less importance than certain other factors in producing the changes noted.

The thoracoplasties on these 17 patients were, with one exception, performed under local anaesthesia with premedication with omnopon; ¼ to ½ grains were administered, the amount depending on the patient's physique and tolerance of the operation. The extent of rib resected at the first stage included the whole of the upper two and the back end of the third rib, or the upper two and very short posterior lengths of the third and fourth ribs. Apicolyis was performed in all cases. The pleural cavity on the side of the operation was not injured. The patients were in the lateral position for operation, and received blood transfusion as a routine. In short, the operation was a standard procedure, modified as the circumstances of operating in the presence of a contralateral pneumothorax demanded.

To throw the changes in the A.P. into sharper relief, two patients on whom extrapleural pneumothorax was performed and with contralateral A.P. are also included. These operations again conformed to an accepted technique, and were performed under local anaesthesia.

THE NATURE OF THE CHANGES OBSERVED IN THE ARTIFICIAL PNEUMOTHORAX

It is difficult to offer a simple term to describe the changes noted in the A.P. during or immediately following operation. The essential nature of the change is a diminution of lung volume beneath the A.P. When mediastinal shift exists the lung is retracted; in other instances the lung appears to be compressed. Of the three possible descriptions—diminished lung volume, lung retraction, or increased lung compression—the last is put forward as the most accurate term available. "Lung compression," therefore, will be applied throughout to describe those changes seen in the
A.P. may be spontaneous cases A.P. in greater or lesser extent, and, a finding which may be of significance as far as aetiology is concerned, in two cases compression of the lung which was lobar in distribution rather than affecting the whole lung (Cases 1 and 2).

This puzzling and embarrassing occurrence has been noted in larger series of cases than the one being described, but there is little reference in the literature to details of its cause or effects. Many reasons exist for assuming that spontaneous pneumothorax, at the time of or shortly after operation, is not the cause. These reasons will be discussed later. Previous observers, however, are satisfied that contralateral spontaneous pneumothorax is common. Thus, Corylos and Ornstein (1936) reported superimposed spontaneous pneumothorax occurring 22 times in a series of 16 patients undergoing thoracoplasty in the presence of a contralateral pneumothorax. O'Brien, Tuttle, Day, and O'Connor (1938) reported the occurrence of spontaneous pneumothorax eight times in a series of 85 cases. The A.P. was lost or had to be abandoned after operation in 36 of the total of 85 cases. No reasons were given. More recently Price Thomas and Cleland (1943) reported spontaneous pneumothorax in four of a total series of 12, and Beard (1951) in a paper on thoracoplasty for advanced disease called attention to 11 cases with contralateral pneumothorax. Of these only five gave no cause for anxiety during operation. One death occurred in this group. Of exceptional interest are the findings of Finkelstein and Guggenheim (1940), who mentioned severe post-operative episodes in six of their 10 cases. So pertinent are their remarks that they are quoted:

"Among our own 10 cases, severe post-operative episodes resembling spontaneous pneumothorax occurred in six. In a number of cases such episodes followed each of several stages, and in one case the outcome was fatal. We are not completely satisfied that these episodes were in all instances actually superimposed spontaneous pneumothoraces, since the intrapleural readings were often bizarre and not characteristic of such an occurrence. We suspect that in some cases we were dealing with a different type of respiratory difficulty, possibly mediastinal flutter."

The use of the word bizarre applied to the intrapleural readings after operation, is apt; and certainly in this series the post-operative intrapleural readings could never be predicted with accuracy. No constant association was noted between the occurrence of lung compression and alterations in the reading to a more positive pressure. This, presumably, was the identical experience of the last two writers when they referred to severe post-operative episodes, resembling spontaneous pneumothorax but without confirmatory intrapleural readings of such an event. In the present series, increased lung compression was observed with readings the same as before operation, a more usual finding being a small rise in pressure compared with the pre-operative figure. (Care was taken that the patient's position before recording this figure was the same in each instance.)

Reference to the series of cases quoted, to the cases reported later, and to the radiographs shown, combined with the general and common knowledge that the contralateral A.P. behaves in a capricious manner during thoracoplasty, are the reasons for questioning the desirability of a contralateral A.P. at the time of operation. As to a precise cause producing this effect of increased compression, it has already been indicated that a diagnosis of spontaneous pneumothorax cannot be upheld. The reasons for this assertion are that each episode is related to the immediate post-operative period; no intrapleural pressure readings suggesting spontaneous pneumothorax have been observed in this series; none of the A.P.s were complicated by the development of fluid in the space; and no patient had any rise in temperature. The incidence of lung compression far exceeds in frequency that of spontaneous pneumothorax occurring in A.P. treatment alone: nine in 600 cases undergoing A.P. treatment reported by Paul (1950). Further, it is inconceivable that a patient undergoing three stages of thoracoplasty at fortnightly intervals can, on each occasion, within four hours of operation, develop a contralateral spontaneous pneumothorax. Two patients had incidents of increased lung compression after each of three stages, and observation of their progress in each instance led one to search for a cause other than spontaneous pneumothorax.

Possible Aetiological Factors

To some extent in the past the changes in the A.P. have been accepted as a fact without explanation or, in my view, over-simplified by attributing
the changes to spontaneous pneumothorax. The more that a single comprehensive cause is sought, the more difficult a real understanding of the mechanisms involved in the altered state of the A.P. becomes, and it is clear that a single cause will not account for the changes in all cases. Unequivocal diagnosis of spontaneous pneumothorax was not made in any case in this series, but the explanations offered for the phenomena observed do not necessarily cover every aspect of the problem.

The following factors, either singly or in combination, are considered as possibly causative: mediastinal shift with actual lung compression from reduction in the volume of the hemithorax while this is dependent in the lateral position on the operating table; excessive paradoxical movement leading to dyspnoea and drawing over of the mediastinum to the A.P. side on inspiration; sputum retention in the A.P. side with absorption atelectasis; spread of disease; postural, from prolonged lying on the A.P. under the left side; traumatic, from puncture of the opposite pleura at operation; artefact, from the x-ray films being taken in full expiration. These will now be considered in turn.

**Mediastinal Shift and Excessive Paradoxical Movement.**—There are good reasons for supposing that, singly or together, these are not the cause in every case. The condition may appear without radiological evidence of mediastinal shift, and, although paradoxical movement must result from any interference with the integrity of the chest wall, no gross movement of the mediastinum has been observed by the empirical method of screening in a number of patients who were screened immediately after that stage of the operation. Deliberate posturing of the patient on the side some time following operation when the Semb space has filled up with blood and produced mediastinal shift to the A.P. side does not result in radiological evidence of lung compression. This occurs essentially during the operative phase. Again, in the two cases of extrapleural pneumothorax in which the change was observed (Cases 3 and 4), the final pressures in the A.P. and extrapleural space were approximately equal. If the mediastinum were sufficiently mobile to move in pendulum fashion during operation, equalization of the air pressures on its two sides should centralize it. Neither of these patients had significant alterations in their A.P. pressure readings compared with the pre-operative figure, nor were they unduly dyspnoeic, and, because of the limited nature of the rib resection, paradoxical movement was absent.

An attempt has been made subsequently to investigate pressure changes in the A.P. space and its relationship to mediastinal movement by inserting a Monaldi tube through the first interspace and taking continuous pressure readings throughout the operation. As a source of information this manoeuvre is limited by the difficulty in keeping the tube patent. Turning the patient to the lateral side will increase pressure in the A.P. from about $-8 - 4$ to $-5 + 3$, as will pressure on the opposite chest wall (the side of the operation), depressing the scapula, holding the apex of the lung down during apicolyis; in fact any interference that would naturally be associated with a rise of pressure. An unexpected finding was that simple incision of the wall of the Semb space, before its evacuation, at the second stage of an operation, produced an appreciable rise. It is possible that dyspnoea precedes or precipitates the increased lung collapse under the A.P. The movement of the mediastinum is toward the A.P. side on inspiration (de Carvalho, de Sousa, and Vidal, 1948), and dyspnoea may accentuate this movement and produce lung collapse. It has already been indicated that the change may occur without dyspnoea, but as the severity and degree of dyspnoea remain difficult to assess at the bedside, it would not be justifiable to discard dyspnoea as a cause rather than an effect.

**Sputum Retention.**—Sputum retention with distal absorption atelectasis suggested itself as a cause by the occurrence of lobar collapse in two patients. A selective compression effect picking out only one lobe is difficult to imagine, and the more likely explanation in these two cases at least was bronchial obstruction. Two criticisms of this supposition exist: that the lobe involved under the A.P. was already diseased and therefore more prone to atelectasis, and that the change happens too rapidly for this to be the cause. However, Johnstone (1950) has described radiological changes in films taken in the operating theatre of patients after prolonged operation in the lateral position, and has demonstrated segmental and lobar atelectasis in the contralateral lung in these films, a result of secretions collecting in the under lung. He also described a small contralateral pleural effusion in his summary of the effects of operating in the lateral position. This has not been seen in this series. From the point of view of the subsequent management of the A.P. and the possibility of having to aspirate air from it and hence draw retained sputum more peripherally into the bronchial tree, this potential source of spread must be considered.
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Spread.—Spread of disease to the contralateral lung may occur at operation and be evident in the first post-operative film (Thompson, 1952). Initially the radiographic appearances are similar to those of a segmental lobar atelectasis, but the shadows are persistent and gradually the lung on the side of the A.P. contracts. Unless the possibility of immediate spread is considered the radiographic shadows may easily be put down to a more benign cause.

Posture.—It was considered desirable to make certain that the lateral posture alone would not account for the changes seen in the A.P. Another effect of the lateral position described by Johnstone in the paper already referred to is mediastinal displacement. This was confirmed by selecting a number of patients with bilateral disease and an A.P. on one side. These patients were given the routine premedication and postured with the A.P. side down for the length of time corresponding to the operation. This produces shift of the mediastinum, reduction in lung volume, and a rise in pressure in the A.P. while the patient remains on the side, with a return to the normal on restoring the patient to the vertical position. No permanent radiological change was observed and no lasting pressure change in the A.P. It was concluded that factors other than simple posture are responsible for the development of the changes in the A.P. already described by the term lung compression.

Trauma.—Tears in the opposite pleura were not encountered at operation in any of the patients in this series.

 Artefact.—Radiographs were taken in full inspiration and full expiration to preclude the possibility of the change being apparent rather than real. In most cases there was noticeably little difference in a comparison of these films, and certainly less than the difference between an inspiration and expiration film in patients with an A.P. alone. The reason for this may be either that dyspnoea prevents an adequate inspiratory movement or that the thoracoplasty allows paradoxical movement of the mediastinum, and inspiratory movement, instead of filling the lung on the A.P. side, will pull the mediastinum toward that side.

That from this brief résumé of possible aetiological factors no conclusion is drawn is immaterial to the hypothesis that, in a significant proportion of patients undergoing surgical collapse, a change in the contralateral A.P. occurs. From this change various undesirable sequelae follow.

Reasons for Discarding the Contralateral A.P.

To any principle there are exceptions, and it is agreed that to the general principle of advising against a contralateral A.P. exceptions exist. Such is the patient with bilateral cavitation on whom an extensive thoracoplasty is considered necessary on the one side and some form of non-surgical collapse therapy on the other. It is, clearly, essential to ascertain before operation that treatment of the contralateral side can be undertaken by a satisfactory A.P. Operating with the A.P. present will be the one and only means of making the attempt at obliterating the bilateral cavities. An example such as this, and others where a surgical programme cannot be begun until a satisfactory contralateral A.P. is established, or, in other words, patients on whom, because of age, general condition, or other factors, limited bilateral surgical collapse would not be permissible, are the only exceptions. It is against the A.P. that is induced for fear of progression of a small cavitated lesion, or to control the small infiltrate, or for no better reason than that the presence of scattered contralateral disease indicates an A.P., that attention is directed. Emphasis will be placed later on this so-called controlling influence of the contralateral A.P.

Criteria for establishing that, in general, a contralateral A.P. is undesirable are (1) that the operation of thoracoplasty was conducted with regard to the precautions usually observed with a contralateral A.P.; (2) that the sequelae of lung compression on the A.P. side which arise during the course of an operation so conducted are detrimental to the patient’s wellbeing; and (3) that operation can be performed and concluded with a better result as far as the contralateral side is concerned by operating without the mantling A.P. These will now be discussed point by point.

Precautions at Operation.—In a paper by Buxton and O’Rourke (1944) the conclusion was reached that the performance of thoracoplasty in the presence of a contralateral pneumothorax was desirable and that the results justified its more widespread use. They encountered pneumothorax complications in 13 of the 44 patients, and in 13 patients the planned programme was not completed because of complications, not necessarily due to the A.P. Considering that a large proportion of patients in their series were classified as having far advanced disease with bilateral cavitation, the results reported must be accepted as being good. They laid down, or inferred, certain precautions to be observed in the management of the
patient during the operation. These will be mentioned because similar precautions were observed in operating on the 17 patients in this series. These were that the A.P. was allowed to become shallow before operation; that the thoracoplasty was delayed until the disease under the A.P. gave evidence of improvement; that the A.P. should be at least of six months' duration (quoting Finkelstein and Guggenheim); and that the operation should be carefully staged. They performed 174 stages on their 44 patients. In the present series each patient had a three-stage operation. Thus, apart from what may be an important difference—that of the number of stages each patient had—the same attention to pre-operative management was afforded in both series. The method of anaesthesia was not described in Buxton and O'Rourke's paper.

Lung Compression on A.P. Side.—Of the 17 patients, 11 exhibited lung compression after one or each stage. The number of incidents of lung compression was therefore greater than 11. The most obvious sequel to this occurrence is that the patients' early convalescence is not the smooth and orderly progression that it should be. They may be unduly dyspnoeic, have difficulty in coughing up sputum, or suffer from a surfeit of attention, aimed equally at adequate supervision of the A.P. and the thoracoplasty.

The results in the 11 patients in a series of 17 were that in two the A.P. was abandoned immediately after operation as a life-saving measure, and a further three lost their pneumothoraces within three months of operation. Another two patients (cases 1 and 2) showed lobar collapse beneath the A.P. soon after operation which persisted in spite of attempts to expand the lobe, two showed an increase in contralateral disease within six weeks of operation, and in the remaining two the A.P. returned to its pre-operative state without harm. Five patients, therefore, from a series of 17 lost their pneumothoraces, for which good indications had existed before operation, during the early post-operative days, and in another two the risks of the A.P. were enhanced by the development of lobar collapse. These actual sequelae are sufficiently arresting to give serious consideration to the advisability of the contralateral A.P. The potential sequelae succeeding lung compression are equally serious. One has to accept lung compression in a proportion of cases as a fact, regardless of its cause, to justify this statement. It then becomes clear that, in fact, control of the contralateral side is lost by inducing an A.P., and those factors likely to prevent spread of disease are suppressed. As soon as lung compression occurs, the chances of the patient clearing infected sputum from the lung on the side of the A.P. are minimized. The existence of the A.P. itself reduces the effect of coughing very considerably, and the super-added compression adds to the difficulties of expectoration. The more compressed the lung is the more ineffectual the cough, and the onset of dyspnoea, by promoting paradoxical mediastinal movement, further aggravates this difficulty. The sputum retained in the bronchi will, if not promptly removed, produce atelectasis which rules out any possibility of the bronchi being cleared by the natural reflex of coughing. The remedies for this state of affairs are neither certain nor safe. Bronchoscopy will not prevent the onset of lung compression even if it readily allows the aspiration of retained sputum, and it has already been suggested that the lung cannot with certainty be brought further up and the A.P. made shallower by the simple expedient of taking air off the A.P. In four cases this was tried and found to be the case. It is, perhaps, because of this difficulty in achieving lung re-expansion that the condition has been considered to be due to spontaneous pneumothorax. In any event the risks of draining infected sputum more peripherally into the lung by taking air off the A.P. are obvious. This dilemma is not to be anticipated during the operation of every patient operated on with a contralateral A.P., nor will the situation be recognized unless frequent x-ray examinations are made, but it is on this account that the proposal is made that the best protection for the opposite lung during the operation and after is the existence of a normal, unimpeded cough mechanism. By the same reasons, confirmation is given to the added risk and extra dangers involved if one is compelled to operate with the contralateral A.P. present.

Better Operative Results Without A.P.—In assessing and comparing results between operating with and without a contralateral A.P. the difficulty is of comparisons in so diverse a disease as pulmonary tuberculosis. In the total of 280 cases from which the 17 cases with contralateral A.P. were taken, some 40 had bilateral disease at the time the thoracoplasty was undertaken. Various degrees of activity were encountered, and the chronic, stable, calcified focus was not included. After operation in these 40 cases undertaken with no contralateral A.P., in only two was contralateral disease exacerbated and progressive at three months after operation. Minor increases of disease in the early weeks were noted in three more, but response to streptomycin was satisfactory. The operation was followed by serious
consequences in the opposite lung, therefore, in only two out of 40 cases.

In addition to this practical manifestation of success, good reasons exist for operating without the contralateral A.P. as a regular practice. One is the known beneficial effect that collapsing one lung has on the other (Lundquist, 1941). By the thoracoplasty alone the contralateral side can be expected to improve if the operation is safely completed, and such factors as the patient’s resistance remain the same and no fortuitous or secondary complications arise. Another advantage now held over those who in the past perfwho to had to explore the possibilities of controlling contralateral disease by active measures is chemotherapy. Provided the organism is not drug-resistant, this consideration may be decisive in advising against an A.P. at the time a case is being assessed for surgery, and be sufficient to control contralateral disease until the operation is concluded and an A.P. can then be induced with safety and advantage to the patient.

In this respect it should be pointed out that this paper in no way seeks to belittle the value of thoracoplasty and pneumothorax as a long-term treatment. Its concern is in particular with the order of events in deciding on this long-term policy and the best time for the induction of the A.P., if such is considered desirable. This must clearly be after the satisfactory conclusion of any collapse operation, with the exceptions indicated.

The course of the disease under treatment may decide that the operation has to be done with the contralateral A.P. present. In this circumstance the possible advantages to be gained by operating with the patient in the prone or "face-down" position remain to be explored. Sellors (1952) has described the use of this position in thoracoplasty for patients with excessive sputum, and given technical reassurance on operative difficulty in removing the first rib and achieving a satisfactory apicolysis. The extent of encroachment on the under lung by mediastinal displacement with the patient in the lateral position at operation, and the effects of sputum collecting in the bronchi of the contralateral lung, are also discussed. Whether these two factors—posture and sputum retention—can wholly explain the dynamic problem of lung compression is uncertain.

ILLUSTRATIVE CASE REPORTS

CASE 1.—M. B., a man aged 22, weight 13 st. 2 lb., had a left A.P. induced in October, 1948, for left apical disease. There was a fibrotic lesion in the right apex with cavitation, and moderate displacement of the mediastinum to the right. A first-stage thoracoplasty was performed in November, 1949, under local anaesthesia. There was no significant dyspnoea after operation. The first post-operative film taken on the day of operation showed left upper lobe collapse. Pressures were the same as before operation. Bronchoscopic aspiration of secretion and withdrawal of air from the A.P. failed to re-expand the lobe. Incidents of lung compression at each of next two stages. The left A.P. was obliterated in May, 1950, and left-sided disease is increasing.

CASE 2.—G. D., a woman aged 28, weight 8 st. 2 lb., had a right A.P. induced in July, 1949, for right apical disease. There was a cavitated fibrotic lesion in the left apex with marked retraction of the mediastinum to the left. A first-stage left thoracoplasty was performed in February, 1950, under local anaesthesia. The first post-operative film taken on the day of operation showed right upper lobe collapse and pneumothorax on the side of operation. The subsequent course was the same as in Case 1. The right A.P. was obliterated in December, 1950.

CASE 3.—D. E., a woman aged 27, weight 8 st. 1 lb., had a right A.P. induced in May, 1945, for right apical disease with multiple small cavities. The A.P. was not altogether satisfactory because of atelectasis in the right upper lobe, but was left in view of left apical cavitation (Fig. 1a). Left extrapleural pneumothorax was induced in March, 1950, under local anaesthesia. The first post-operative film taken on the day of operation showed a considerable increase in lung compression in the right and a small left extrapleural strip (Fig. 1b). There was no dyspnoea post-operatively. There was some increase in pressure in the right A.P., but the mean pressure was below atmospheric. The right A.P. and extrapleural pressures were approximately equal. Convalescence was smooth.

In May, 1952, the patient was well and the right A.P. continues to be refilled.

CASE 4.—O. R., a woman aged 36, weight 8 st., had a left A.P. induced in November, 1947, for left apical disease with cavitation. Active right apical disease with cavitation was also present. Extrapleural pneumothorax was performed on the right side in May, 1948, under local anaesthesia. The first post-operative film, taken on the day of operation, showed considerable increase in lung compression on the left side and a small right-sided extrapleural pneumothorax.

In May, 1952, the patient was well, and the left A.P. is being maintained.

CASE 5.—J. P., a man aged 30, weight 9 st., had a right A.P. induced in January, 1948, for right apical disease. A tomogram confirmed the presence of a thin-walled cavity seen above the left clavicle in the plain film. Sputum was persistently positive. A first-stage left thoracoplasty was performed in February, 1950, under local anaesthesia. The upper two ribs and short posterior lengths of the third and fourth ribs were removed. The first film taken on the day after operation showed a central mediastinum, a patchy atelectasis of the right lung, and increased lung compression. This patient had considerable dyspnoea and difficulty in coughing.
FIG. 1 (Case 3).—(a) Before operation. Upper lobe under A.P. already atelectatic. (b) After operation showing considerable compression of contralateral lung.

FIG. 2 (Case 8).—(a) Before operation. (b) After operation. Position of mediastinum unchanged.
up sputum. Withdrawal of amounts of air up to 200 ml. during the first five days did not materially help his respiratory distress. The right A.P. reverted to its pre-operative equilibrium and there was no delay between the stages.

In May, 1952, the patient was well, working, and the right A.P. is being maintained.

Case 6.—A. C., a woman aged 26, weight 6 st. 2 lb., was frail and lightly built with far advanced disease. A left A.P. was induced in February, 1948. There was an extensive fibrotic lesion at the right apex with many small cavities, and marked retraction of the mediastinum to the right. A first-stage right thoracoplasty was performed in September, 1949, under local anaesthesia. The patient experienced considerable respiratory distress after operation. Pressures in the left A.P. were raised from the pre-operative figure, but were below atmospheric. Air was withdrawn immediately after operation with symptomatic relief. A post-operative film taken on the day after operation showed lung compression on the left. This was repeated at the second stage. The left A.P. was obliterated in June, 1951.

Case 7.—J. B., a man aged 23, weight 13 st. 10 lb., had a right A.P. induced for minimal right apical disease in August, 1949, as a preliminary to left thoracoplasty. A large left apical cavity with widespread infiltration in the left lung was seen on radiography. A first-stage left thoracoplasty was performed in January, 1950, under local anaesthesia. The upper two ribs and a short length of the third were resected. A film taken on the day after operation showed lung compression with increased infiltration in the right mid-zone. There was excessive bleeding into the Semb space and displacement of the mediastinum. Pressures in the right A.P. were increased after operation. The patient experienced considerable respiratory distress during the first few post-operative days. The disease in the right A.P. was progressive two months after operation.

The patient is not working two years after operation.

Case 8.—J. G., a woman aged 27, weight 7 st. 2 lb., had a right A.P. induced in January, 1950, for right apical disease preliminary to left thoracoplasty for a long-standing honeycombed lesion at the left apex (Fig 2a). A first-stage left thoracoplasty was performed in September, 1950, under general anaesthesia. The patient refused operation under local anaesthesia. There was some dyspnoea after operation. Pressures in the right A.P. were the same as before operation. A film on the day following operation shows increased lung compression and an atelectatic area in the right upper lobe (Fig 2b). The position of the mediastinum is unchanged. The right A.P. returned to its pre-operative condition at the end of the first week. Lung compression occurred again after the second stage immediately following operation. At the third stage two weeks after the second, when the sixth and seventh ribs were removed, the right lung became severely compressed, and a film taken in the theatre with the patient still anaesthetized showed very little aeration lung on the right. The patient remained unconscious, of poor colour, and with gross paradoxical movement until the right A.P. was sucked out. Convalescence from then on was uneventful, but the right A.P. could not be re-induced.

Summary

A series of 17 patients undergoing thoracoplasty with a contralateral A.P. present is described. Contralateral spontaneous pneumothorax has not been observed in this series.

Changes occurring in the contralateral lung are described, using "lung compression" as the descriptive term.

Possible causes are discussed without a conclusion being reached.

Actual and theoretical sequelae lead to the opinion that, with the exceptions described, the contralateral A.P. is undesirable.

The patients in this series were admitted to the Country Branch of the London Chest Hospital, under the care of either Mr. T. Holmes Sellors, Mr. Vernon C. Thompson, or Mr. Donald Barlow. I am grateful to them for their advice and interest in the preparation of this paper and for their permission to make these observations on their results.

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