THE EFFECT OF LIPIODOL ON ALVEOLAR GAS EXCHANGE [1, 2]

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Within the past 15 years the advances in intrathoracic surgery have made visualization of the bronchial tree with contrast media, particularly iodized poppyseed oil (lipiodol), a common practice. Despite the established safety of the procedure in the vast majority of instances, repeated questions have been raised as to the innocuousness of the oil in the pulmonary parenchyma. The feeling has been expressed by some authors that it is unwise to perform any intrathoracic procedure for several weeks to several months following the administration of lipiodol. Thus Churchill (1) has advised delaying operation for a minimum of six weeks after bronchography because of fear of postoperative atelectasis and pneumonitis due to the oil. Belsey (2) was of the same opinion and a more recent paper by Miscall (3) indicates a persistence of this feeling.

Pinkerton (4) has concluded from animal experiments that lipiodol in the absence of infection produces almost no reaction in the lungs. In clinical use, however, some evidence of lipoid granuloma has been observed (5).

It is also conceivable that lipiodol in the alveoli might impair transalveolar gas exchange and thus interfere with recovery from an intrathoracic operation. Whereas immediately after introduction the material is limited chiefly to the bronchial tree, soon thereafter its presence can be demonstrated diffusely filling the lung parenchyma where it may persist for several weeks or longer. In an attempt to evaluate the possibility of deficiencies of transalveolar diffusion following the administration of lipiodol the present study was undertaken.

MATERIAL AND METHODS

Ten patients who were to have lipiodol bronchograms were studied before, within several hours after the instillation of the oil and on the following day. Bilateral bronchograms were performed by the aspiration technique using 10 cc. of oil on each side. Since there is considerable puddling in the pyriform sinuses less than this actually enters the lungs. Nevertheless, with this method satisfactory mapping of the entire bronchial tree is regularly obtained. It should be stressed that a significant amount of residual lipiodol was seen in the alveoli of all patients on fluoroscopy at the time the last tests were performed.

The techniques employed to study the patients were: *1*) determination of the oxygen pressure (P_{O_2}) gradient between the alveolar air ($P_{A_{O_2}}$) and the arterial blood ($P_{a_{O_2}}$) [4]; *2*) measurement of the arterial oxygen saturation; *3*) determination of the time required to attain full saturation when the subject was switched from room air to 100% oxygen and *4*) determination of vital capacity and its subdivisions.

The patient was brought to the laboratory without medication, but not necessarily in fasting condition. After resting 10–15 minutes on a stretcher in the supine position a sample of arterial blood was withdrawn from the femoral artery in the course of one to two minutes, during which period the expired air was collected in a Douglas bag, which previously had been washed out twice with the same gas. The patient was provided with a mouthpiece and nose clip and a system of valves with small dead space was used for the air collection.

Precautions were taken to disturb the patient as little as possible. Thorough novocaine infiltration preceded the arterial puncture, and several minutes were allowed to elapse between the arterial puncture and the collection of blood and expired air. Twenty cc. of blood were collected in a lightly oiled syringe containing 0.5 cc. of a mixture of eight parts of commercial heparin solution and two parts of a saturated solution of sodium fluoride.

The direct determination of oxygen and carbon dioxide tensions of this blood sample was then immediately performed by a technique which has been described in detail elsewhere (7). In principle, it consists of the equilibra-

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[4] The symbols used throughout this publication have been made to conform with those recommended at the April, 1950, meeting of the American Physiological Society (6). Substitutions for formerly used symbols are as follows: 'P' is gas pressure in general; 'F' is fractional concentration in dry gas phase; 'I' is inspired gas; 'A' is alveolar gas; 'a' is arterial.

tion of 17 cc. of blood with an 80 cu. mm. gas bubble (initial composition of the bubble 12.15% O_2, 5.25% CO_2) for 10 minutes at 37° C. At the end of this time pressure equilibration of the respiratory gases in blood and gas phases is obtained with less than 1 mm. Hg change in gas pressure in the liquid phase. The bubble is then transferred to a Scholander micro gas analyzer, where its composition is determined. On the assumption that the total gas pressure of the bubble is equal to atmospheric, the partial pressures in the bubble, and therefore, in the blood, are thus found. This assumption is justified in the case of normal arterial blood. Though in some of the patients studied the total arterial gas pressure was probably somewhat less than atmospheric, this would not affect the partial pressures of CO_2 and O_2 to a significant degree. The error of this method, as found by performing the above determination on blood of known gaseous composition, amounts to $+0.1 \pm 3.3$ mm. Hg for O_2 and -5.3 ± 2.4 mm. Hg for CO_2. A correction of +3.0 mm. Hg has been made for the CO_2 pressure due to the diluting effect of the anticoagulant.

The "ideal" alveolar oxygen pressure was determined from analysis of the expired air, collected simultaneously with the blood, and from the directly determined arterial CO_2 pressure. As defined by Riley and associates (8, 9) and Rahn (10) the "ideal" or "effective" alveolar pressures are those which if present continuously and uniformly in all functioning alveoli, and if complete equilibrium were reached between the blood and gas phases in the lung, would permit CO_2 and O_2 exchange in amounts equal to that measured from analysis of inspired and expired air. As alveolar ventilation-perfusion relationships vary in different parts of the normal, and particularly of the pathologic lung, and as equilibrium of oxygen between blood and alveolar air in the abnormal lung often is not accomplished, "ideal" alveolar air must remain a theoretical quantity. Its practical usefulness lies in the fact that reproducible values can be assigned to alveolar oxygen and carbon dioxide pressures, whereas this would be impossible to accomplish through direct sampling in the many instances of non-homogeneous alveolar air. The calculation of the "ideal" alveolar P_{O_2} is based on the assumptions that the alveolar respiratory quotient equals that of expired air, and that the alveolar CO_2 tension equals that of arterial blood. Whereas the former assumption seems to be correct (10), the latter may not be strictly true in cases of severe pulmonary disease. However, the patients studied in this paper probably did not have a significant arterial-alveolar P_{CO_2} gradient.

The equation used to calculate "effective" alveolar P_{O_2} was the following (9, 11):

$$P_{A_{O_2}} = P_{I_{O_2}} + \frac{P_{a_{O_2}} \times F_{I_{O_2}}(1 - RQ)}{100 \times RQ} - \frac{P_{a_{CO_2}}}{RQ}.$$ [4]

As various techniques are in use for the direct determination of blood gas tensions and for the determination of alveolar air composition, the oxygen gradient in 10 healthy male subjects from 25–35 years of age was determined, using the technique described above. The results are summarized in Table I. The average gradient was 3.0 mm. Hg with a standard deviation of 2.2. Lilienthal and co-workers (12) using the same method for effective alveolar air and the Riley technique for direct measurement of the arterial gas tensions found an average gradient of 8.9 mm. Hg ± 2.9 in six normal subjects. Cournand and colleagues (13) with identical methods in 10 normal individuals found an average gradient of 10.0. Comroe and Dripps (14), employing a method for determining blood gas tensions comparable to ours and end-expiratory Haldane-Priestley alveolar air samples, found an average gradient of 0.7 ± 5.3. Barker, Lambertsen and associates (15), using effective alveolar air and an improved Riley method for arterial P_{O_2} found an average difference between the two of 3.7 mm. Hg which was statistically not significant.

TABLE I

Normal alveolar-arterial P_{O_2} gradient **

Case	Barometric pressure	Arterial P_{CO_2} (P_{aCO_2})	A Alveolar P_{O_2} (P_{AO_2})	B Arterial P_{O_2} (P_{aO_2})	A – B Gradient
	mm. Hg	*mm. Hg*	*mm. Hg*	*mm. Hg*	*mm. Hg*
1	740	41.2	98.8	93.8	5.0
2	741	37.2	96.0	90.2	5.8
3	750	35.4	102.2	99.8	2.4
4	750	37.1	102.4	99.8	2.6
5	750	38.2	99.2	95.0	4.2
6	742	40.0	99.5	100.0	−0.5
7	747	36.6	108.4	104.4	4.0
8	745	36.9	102.2	98.0	4.2
9	745	38.2	100.5	101.5	−1.0
10	747	41.4	101.5	98.0	3.5
					Av. 3.0 ± 2.2

* Normal males 25–35 years old.

Arterial oxygen saturation

Immediately following the collection of the blood sample for the direct determination of gas tensions a second syringe, the dead space of which was filled with heparin, was attached to the needle and 20 cc. withdrawn for the determination of oxygen saturation in duplicate. The manometric method of Van Slyke was used for oxygen content and the Sendroy technique for capacity (except in the first three patients in whom the usual tonometer method for capacity was employed). Duplicate determinations checked within 0.15 vol.%.

Vital capacity and subdivisions

The measurements of vital capacity, inspiratory capacity and expiratory reserve volume (6) were obtained with the patient supine. Three determinations of each volume were made and the largest value recorded. All volumes shown in Table III are corrected to 37° C., prevailing barometric pressure and saturated with water vapor (BTPS).

Saturation time from room air to 100% oxygen

The time in seconds required for the arterial oxygen saturation to reach a maximum value when the inspired gas was changed from room air to 100% oxygen was de-

TABLE II

Alveolar-arterial P_{O_2} gradient before and after lipiodol

Case Age Sex	Diagnosis	Time	O_2 Saturation	CO_2 Content	Arterial P_{CO_2} (P_{aCO_2})	A Alveolar P_{O_2} (P_{AO_2})	B Arterial P_{O_2} (P_{aO_2})	A−B P_{O_2} Gradient
			%	*mMol/L*	*mm. Hg*	*mm. Hg*	*mm. Hg*	*mm. Hg*
Ha	No pulmonary disease	Before	99.27*	19.29	43.0	88.8	90.3	−1.5
45		5 hours	94.67*	17.81	41.6	99.7	95.2	4.5
M		1 day	95.52*	20.74	43.7	93.9	94.2	−0.2
Co	Bronchitis and slight	Before	97.68*	22.16	40.5	95.6	93.8	1.8
52	emphysema	6 hours	94.05	21.74	38.5	96.6	92.9	3.7
M		1 day	96.62	21.43	38.8	90.6	90.4	0.2
Bo	Bronchiectasis	Before	99.78	20.74	36.8	103.3	102.6	0.7
24	LLL and lingula	4 hours	98.00	21.29	36.1	100.4	97.2	3.2
M								
Beh	No pulmonary disease	Before	97.06	18.95	39.9	92.4	96.0	−3.6
17		5 hours	94.12	19.56	46.4	81.2	80.5	0.7
F		1 day	92.68	21.88	47.7	91.7	89.0	2.7
Bu	Carcinoma obstructing	Before	85.06*	27.22	46.9	92.2	(48)†	44.2
48	R. bronchus	2 hours	86.38*	25.58	41.4	95.2	53.2	42.1
M		1 day	84.95*	27.75	45.6	94.9	56.1	38.7
Bet	Severe emphysema	Before	90.99*	22.48	36.9	98.7	62.3	36.4
41		3 hours	85.32*	25.73	38.5	98.8	57.4	41.4
M		1 day	91.13*	23.62	36.5	95.8	60.3	35.5
Ho	Bronchiectasis L. lung,	Before	93.57	24.20	40.6	91.9	81.2	10.7
56	fibrosis and severe	Before	91.03	24.40	42.0	92.3	81.6	10.7
M	emphysema	7 hours	86.87	24.84	42.1	95.1	52.8	42.3
		1 day	88.65	24.15	36.4	99.9	60.8	39.1
		2 days	92.95	23.30	42.5	89.8	89.9	−0.1
McC	Bronchiectasis	Before	94.20	21.71	41.5	94.9	83.1	11.8
20	LLL and lingula;	5 hours	94.59	21.90	37.3	100.6	82.7	17.9
M	minimal RLL	1 day			42.8	93.1	83.9	9.2
Da	Infected cysts LLL	Before	93.41	24.26	43.3	92.3	85.8	6.5
15		Before	—	—	44.3	98.5	85.7	12.8
F		5½ hours	92.40	22.26	44.3	89.2	82.2	7.0
		1 day	95.28	22.40	42.9	90.4	89.8	0.6
Mo	Bronchiectasis	Control	87.56	25.95	43.9	77.8	49.0	28.8
69	R. lung	Control	92.39	23.52	42.1	104.0	58.9	45.1
M		4 hours	81.60	25.00	42.9	91.1	52.5	38.6
		1 day	—	—	46.7	89.7	56.0	33.7

* Tonometer equilibration for capacity.
† Calculated from saturation as direct determination not satisfactory.

termined with a modified oximeter designed in this laboratory by Elam, Sleator and co-workers (16–18). The earpiece of this apparatus employs a tungsten filament light source and two selenium photocells from which the electrical responses are channeled separately. The first cell which receives transmitted light filtered through a Wratten 29 filter measures changes in the red spectral region and is sensitive to changes both in total hemoglobin content and in oxygen saturation. The infra-red light which reaches the other cell has passed through three thicknesses of Wratten 87 and two thicknesses of Wratten 88. The output of this cell reflects chiefly changes in the total amount of blood in the path and is independent of oxygen saturation above 60%. From the amplified and continuously recorded outputs of the two cells the oxygen saturation of the blood in the optical path can be determined by means of a calibration curve. This instrument is similar in principle to that described by Wood and Geraci (19). The lag of the recorder for full scale deflection is 0.45 seconds. The ear lobe to which the instrument was attached was "arterialized" by histamine iontophoresis using 5 milliamperes of current for one minute on both the anterior and posterior surfaces.

With the amplifications of the two channels set at an arbitrary level that would give an adequate differential between room air and 100% oxygen values the supine subject was allowed to breathe room air through a three-way stopcock and a system of one-way valves using a

TABLE III

Effect of lipiodol on lung volumes

Case	Time	Inspiratory capacity	Expiratory reserve volume	Vital capacity	Vital capacity change
		cc.	*cc.*	*cc.*	%
Ha	Before	3,385	1,059	4,345	
	6 hours	3,385	709	3,965	− 8.8
	1 day	3,385	952	3,810	−12.3
Co	Before	3,010	842	3,731	
	3½ hours	2,823	804	3,680	− 1.4
	1 day	2,910	784	3,758	+ 0.7
Bo	Before	3,560	922	4,300	
	5 hours	3,600	1,002	4,300	0.0
Beh	Before	2,820	697	3,100	
	5 hours	2,410	646	2,740	−11.6
	1 day	2,760	772	3,000	− 3.2
Bet	Before	1,460	635	1,810	
	3 hours	951	476	1,428	−21.5
	1 day	1,128	626	1,721	− 4.9
Ho	Before	1,240	816	1,830	
	6 hours	861	622	1,680	− 8.2
	1 day	880	828	1,811	− 1.0
	2 days	894	894	1,810	
McC	Control	2,605*	1,010	3,620	
	5 hours	2,710	1,003	3,770	+ 4.2
	1 day	3,020	1,069	3,838	+ 6.3
Da	Before	1,058	370	1,320	
	Before	1,430	529	2,118	
	5 hours	1,310	502	1,780	+ 3.6
	1 day	1,486	530	1,911	+11.2
Mo	Before	1,360	532	1,880	
	Before	1,400	635	1,800	
	4 hours	1,100	481	1,380	−25.0
	1 day	1,149	521	1,680	− 8.7

All volumes corrected to 37° C., prevailing barometric pressure and saturated with water vapor (BTPS).
* Control taken seven days after lipiodol.

mouthpiece and nose clip. When the oximeter baseline was found to be stable, turning of the stopcock at the end of a normal expiration allowed the subject to breathe from a reservoir of 100% oxygen and expire into the room. The instrumental dead space was 70 cc. Each period of oxygen breathing lasted from two to seven minutes and between determinations intervals of seven to ten minutes were allowed for the saturation to reach the room air baseline again. In each subject from three to nine measurements of saturation time were made and the results averaged.

The method of interpreting the records requires some comment. Fowler and Comroe (20) in measuring the saturation time used a single-channel Millikan oximeter which partially compensated for changes in total hemoglobin. The instrument was set at 96% saturation with the normal subject breathing room air. Because the moment of reaching maximum saturation when the subject breathed 100% oxygen was difficult to appraise accurately, Fowler and Comroe used as endpoint 0.5% saturation below the highest value. The instrumental lag was 0.07 seconds to reach 95% of the final value. This method of measurement requires the assumptions that identical deflections of the instrument in different individuals represent identical changes in saturation and also that all subjects were 96% saturated while breathing room air.

In our studies we measured the total deflection from room air to 100% oxygen and subtracted 10% of this deflection to obtain an arbitrary endpoint. The time in seconds between the first inspiration of 100% oxygen and this point is called T_{90} or the saturation time.[5] The oximeter was thus used as a qualitative instrument only.

As a corollary of the above measurement, the lung-to-ear circulation time was also obtained from the records by measuring the interval from the first inspiration of 100% oxygen to the first upswing of the saturation curve recorded at the ear.

RESULTS

Alveolar-arterial Po_2 *gradient*

From our control studies on normal individuals an alveolar-arterial Po_2 gradient of less than 7.4 mm. Hg can be expected in 95% of normal individuals (mean plus 2 x standard deviation). Considering this value as the upper limit of normal it was found that out of 10 patients studied all but four had abnormally large gradients, ranging from 9.7 to 44.2 mm. Hg at rest before lipiodol. This was associated in all instances with a normal "effective" alveolar Po_2. This increase in gradient can be considered as the result of an abnormally large contribution of mixed venous blood to the systemic arterial blood. This large "effective venous admixture" might result either from the presence of a significant number of perfused but nonventilated alveoli or from impaired diffusion across a pathologically thickened alveolar membrane with or without adequate ventilation. Arterial oxygen saturation less than normal (93%) was found in the four cases with the greatest gradients. These data are summarized in Table II.

After the administration of lipiodol the Po_2 gradient increased by 6 mm. Hg or less in all but two instances. These changes and that occurring

[5] Our data are nevertheless roughly comparable to those of Fowler and Comroe (20) for the following reasons. These authors noted a mean increase of 3.72% saturation. From this 0.5% was subtracted for the endpoint which thus occurred 3.22% above the room air level. This is 3.22/3.72 or 86% of the total deflection which compares quite closely with our endpoint (90% of total deflection).

in Da (a *decrease* of 2.7 mm. Hg) are of questionable significance since they are close to the limit of experimental error. In only one patient (Ho) was a significant increase in P_{O_2} gradient found (31.6) and this persisted until 48 hours after the oil was given. This 56 year old man had advanced bronchiectasis of the left lower lobe and lingula associated with considerable emphysema and fibrosis. The change in his arterial oxygen saturation (5.4%), although not the largest in the series, reduced it to a definitely anoxic level from a control value close to normal. This individual was the only one of the six in this series who eventually underwent pulmonary resection who did not survive the postoperative period. He died on his fourth postoperative day of respiratory insufficiency. The remaining patients survived operation without unusual respiratory symptoms. Mo, who appeared to be just as poor an operative risk, had only a minimal change in alveolar-arterial gradient after lipiodol although the reduction in his vital capacity was the largest in the series (460 cc.). Statistical analysis of the data as a whole, using Student's t-test indicates no significant change in alveolar-arterial P_{O_2} gradient nor in absolute value of arterial P_{O_2} after lipiodol administration.

From the above findings the conclusion can be drawn that the presence of deficient alveolar function *per se* does not necessarily predispose to further impairment by lipiodol, but when this does occur it may be an ominous sign.

As might be expected from the greater diffusibility of carbon dioxide, the data reveal no significant increase in arterial P_{CO_2} following lipiodol except in one case (Beh). As this patient had no demonstrable lung pathology the finding is probably due to experimental error.

Arterial oxygen saturation

Although some cases had a fall in saturation as large as 5% after lipiodol most values remained within the range of normal (*i.e.*, above 93%). Three patients (Bet, Mo and Ho) did develop a significant degree of desaturation after bronchography, but in the former two this was not associated with a significant change of arterial P_{O_2} or of alveolar-arterial P_{O_2} gradient. Although the change in oxygen saturation occurring within several hours after bronchography was found to be statistically significant, it should be pointed out that duplicate determinations may differ by as much as 2% saturation.[6]

Vital capacity

Statistical analysis of the vital capacity data (Table III) indicates a significant reduction ($P < .05$) from the control value on the day the bronchograms were made (average reduction 7.6%) but not on the following day (average difference 1.5%). Individually two cases showed a slight increase in vital capacity after lipiodol, two showed minimal or no change and four had from 8–25% reduction within several hours with a return to within 9% or less of the control value by the following day. Only one case (Ha) showed a progressive decrease in vital capacity amounting to a maximum loss of 12.3%. This man, however, had no demonstrable pulmonary pathology and his cooperation was open to question.

Oximeter data

The results of the studies of saturation time are shown in Table IV. In the four normal cases

[6] For this reason and because of the nearly horizontal course of the oxygen dissociation curve in the high range of oxygen saturation, derivation of oxygen tension from oxygen saturation in this range is not permissible and the direct measurement of tension becomes a more sensitive index of changes.

TABLE IV

Saturation time in normal subjects and patients before and after lipiodol

Normal subject	Saturation time	Patient	Saturation time			B – A
			A Before	B After 2–7 hrs.	C After 24 hrs.	
	seconds		*seconds*	*seconds*	*seconds*	*seconds*
GB	31.5	Bu	86.6	53.9	47.1*	−32.7
GB	26.4	Ha	22.1	19.7	18.0	− 2.4
AR	22.5	Bet	31.0	32.3	51.5	1.3
AR	22.4	Co	44.5	44.6	38.9	0.1
AR	29.4	Ho	82.4	101.9	132.2 81.9†	19.5
AR	28.4	Bo	23.8	19.2	—	− 4.6
RP	24.6	Da	24.6	—	—	—
WE	24.0					
Mean	26.2		45.0	45.3	57.5	− 3.2
S.D.	3.4		28.0	30.3	43.7	

Each value in this table represents the average of from two to five readings taken consecutively on the same day.
* Two days after lipiodol.
† Four days after lipiodol.

considerable variability was found, the greatest difference between readings at a single session in the same subject being 17.0 seconds. The mean normal saturation time was 26.2 (±3.4 seconds). This compares closely with the average of 15–30 seconds found by Elam and colleagues (17) with the same equipment and a mean of 25.0 found by Wood, Taylor and Knutsen (21) with their instrument. Using the Millikan oximeter Fowler and Comroe (20) found a mean of 52.0 seconds ± 18.6 and Douglas and Edholm (22) 58.4 ± 6.1.

In the six patients studied a mean saturation time before lipiodol of 45.0 seconds ± 28.0 was found. The reproducibility of the data was comparable to that in the normal subjects except in one case in which a difference of as much as 47.0 seconds was found between successive readings. The administration of lipiodol did not result in a consistent change in saturation time, the mean change amounting to − 3.2 seconds.

It should be emphasized that the saturation time is influenced by several mechanical and physiologic factors other than the transport of O_2 across the alveolar membrane. Among these are proper timing of the beginning of inspiration of O_2, efficiency of intrapulmonary mixing, circulation time from lung to ear and instrumental factors including changing ear lobe volume as the edema from histamine subsides. Inability to control these rigidly undoubtedly accounts for the poor reproducibility.

Measurements of the lung-to-ear circulation time was also obtained from the records. In normals a mean of 5.4 seconds ± 0.7 was found. The mean for the patients (6.6 seconds ± 2.0) was not significantly different. No prolongation of circulation time after lipiodol was observed. (Average several hours after bronchography 6.5 ± 1.1; one day afterwards 6.2 ± 1.7.)

DISCUSSION

From our studies of the alveolar-arterial P_{O_2} gradient the conclusion can be reached that in most instances lipiodol in the amounts commonly used does not increase the proportion of mixed venous blood reaching the arterial circulation ("effective venous admixture"). It, therefore, neither decreases alveolar ventilation nor does its presence on the alveolar surface form a significant barrier to diffusion of oxygen. Even in those cases with an abnormally high control gradient, lipiodol does not generally appear to add further to the abnormality. This applies both to patients with diffuse pulmonary disease (emphysema) as well as to those with more localized processes such as parenchymal infiltration or atelectasis. The pulmonary pathology of the one case in our series with a considerable increase in gradient following lipiodol was not fundamentally different from that found in the other patients, except perhaps in extent. For the purposes of evaluating the advisability of delaying operation after lipiodol administration, however, it should be noted that even in this individual the gradient returned to the control level within 48 hours. In addition, all abnormal values found on any of the tests in the remaining patients had returned to the pre-lipiodol level within 24 hours.

It must be realized that varying amounts of bronchial secretion may result in spontaneous fluctuations in the oxygen gradient. Such fluctuations have actually been demonstrated in some of the patients in whom more than one control study was done. Any comparison between studies before and after lipiodol should be considered in the light of this varying baseline.

In evaluating our data on vital capacity and subdivisions comparison with the work of Zavod (23) can be made. This author found in 50 patients an average loss of 15% of vital capacity within one hour of the administration of lipiodol. This loss decreased to 10% on the following day and had disappeared by the fourth day. It was more at the expense of expiratory reserve volume than of inspiratory capacity. A similar trend is found in our data although the losses of vital capacity are not as marked and not as consistent. We are reluctant to attach any importance to the immediate reduction in vital capacity since it may be due to inadequate cooperation of the patient in whom the presence of residual lipiodol often precipitates cough on maximal expiratory effort.

In summary, we may conclude that the fear of further impairment of alveolar function need not delay an intrapleural operation more than a few days after bronchography.

CONCLUSIONS

1. A study has been made of the pulmonary function of 10 patients before and after bronchography by means of lipiodol.

2. In only one patient was the alveolar-arterial Po_2 gradient found to be definitely increased following the procedure, although five cases in addition to this one had an abnormally high gradient in the control study.

3. Arterial carbon dioxide tension was essentially uninfluenced by lipiodol.

4. The vital capacity was reduced by an average of 7.6% several hours after bronchography.

5. Measurement of the saturation time on changing the inspired gas from room air to 100% oxygen revealed no significant effect after lipiodol. Lung-to-ear circulation time was similarly unchanged.

6. All abnormal values returned to the control level within 24 hours except in one case in whom the pre-lipiodol values were not reached until 48 hours later.

7. It is concluded that the presence of lipiodol has no significant influence on the respiratory gas exchange except in occasional patients with advanced pulmonary pathology.

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