

## THE RESERVE AIR AS AN AID IN THE DIAGNOSIS OF EMPHYSEMA

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The main signs of severe emphysema, deformation of the chest, hyper-resonant percussion note, and harsh breath sounds with numerous rhonchi caused by the accompanying bronchitis, are well known and permit a diagnosis without difficulty. However, there are many cases of moderate emphysema in which none of these signs, or only some, are present. Their diagnosis causes great difficulty. Christie (1944) has therefore said in a review that he prefers to rely for his diagnosis at least as much on the history and the typical symptom of increasing breathlessness on exertion as on the signs. In these circumstances it is desirable to improve the accuracy of this diagnosis.

It is generally recognized that the mid-capacity of the lungs in emphysema is increased. The mid-capacity is the sum of the residual air, reserve air, and half the tidal air. It is increased because the lungs become over-inflated. This is "emphysema" in its widest sense. It occurs as a transient change in muscular exercise, excitement, and CO<sub>2</sub> breathing, and any expiratory resistance may provoke it. Verzár (1933) has called this transient increase of the mid-capacity the third form of regulation of the respiration, since the increase of the mid-capacity increases the respiratory surface. These transient changes can be observed in many healthy subjects and cannot be regarded as abnormal. If they become permanent pathological emphysema has developed. It is uncertain at which stage of this development anatomical changes—rarefaction of the respiratory epithelium, atrophy of blood vessels, and, later, bullae—appear. They may be present at its beginning, or in other cases may appear only as a result of continuous over-inflation. As it is not possible to estimate the degree of anatomical changes by clinical methods, I shall use the term "emphysema," without regard to the anatomical change, to denote a continuous state of over-inflation of the lungs.

An increased mid-capacity must be shown before the presence of emphysema can be assumed. This means, if one disregards the unimportant half tidal

air as a constituent of the mid-capacity, the proof of an increased functional residual air, using Christie's (1933) terminology. This consists of the residual air proper which cannot be removed from the lungs by any exertion, and of the reserve air which can be exhaled by voluntary expiratory effort. The determination of the residual air is complicated, and the methods at our disposal are of doubtful value in emphysema. The reserve air, however, is easily determined by a recording spirometer.

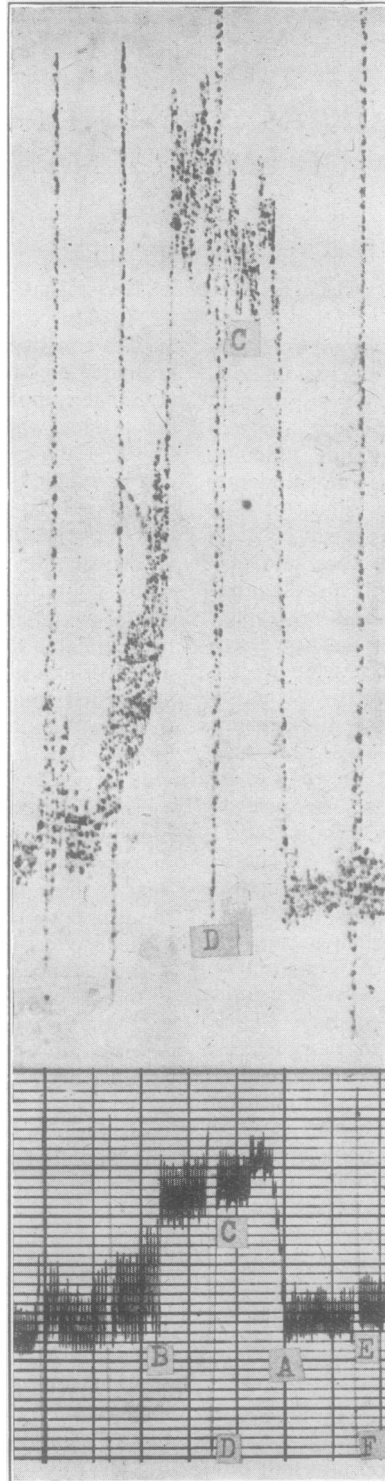
If the tidal air movement is carried out by lungs more inflated than normal, it means that during an ordinary expiration the lungs do not recoil as fully as normal lungs. It must not be taken to imply that they cannot be compressed by external forces, or that the reserve air could not be emptied by voluntary effort. Therefore we should expect an increased reserve air in emphysema as long as the muscular forces of the body are capable of compressing the chest. This increase will not be evident if these forces are diminished by atrophy of the muscles or other causes, or if they are insufficient to overcome obstacles hindering compression—for instance, bronchial spasm or skeletal changes. If these obstacles prevent full compression of the chest by the muscles we cannot expect an enlarged reserve air. That part of it which cannot be exhaled because of obstruction or muscular inefficiency becomes part of the residual air. In such cases the reserve air will appear normal or even small, and the vital capacity will be reduced accordingly.

### EXPERIMENTAL STUDIES

*Experimental Expiratory Stenosis: Artificial Emphysema.*—If a normal subject is connected with a recording spirometer and expiration obstructed by a screw clip on the expiratory tube, it can be seen that at once the amount of air in the chest increases. The tidal air level rises steeply and finally establishes itself on a higher plane. If now the maximum inspiration and expiration are taken

(the routine method of recording the vital capacity and its subdivisions), it will be seen that the reserve air has increased from about 30% of the vital capacity, a normal percentage, to, for instance, 50%, which is abnormally high. If a thoracometer tracing is made it will be seen that, at the same time as the tidal air level on the spirometer drum rises, the circumference of the chest increases correspondingly. This confirms that the chest volume increases. The movements of the diaphragm have also been followed up radiologically in five experiments. In every case the diaphragm assumed a lower level during the expiratory stenosis. As soon as the screw clip is removed, the tidal air resumes its former level. Fig. 1 gives an example from the experiment, with spirometer and thoracometer tracings synchronized; Fig. 2 gives another without the thoracometer tracing. If the tracing of a typical patient with moderate emphysema\* with good costal and diaphragmatic movement (Fig. 3) is compared with the tracing of a normal subject during experimental stenosis (Fig. 1, lower part, and Fig. 2), hardly any difference can be found. In the first case we have an artificial, transient emphysema, in the second a permanent one. In both the muscular strength of ribs and diaphragm is sufficient, but without an expiratory effort the emphysema persists and the reserve air remains enlarged. As the vital capacity is normal in both cases, it seems probable that the residual air

\*This subject had the typical history of emphysema; the vital capacity was very little reduced; the degree of asthmatic spasm was small and easily controlled; the diaphragmatic level was radiologically lower than normal.



may also be normal. An increased mid-capacity on stenosis breathing has been found before by Siebeck (1909) and Bass (1924).

*Diaphragmatic Action and Reserve Air.*—It is well known that in emphysematous persons the diaphragm is found at an abnormally low level. Grossmann and Herxheimer (1948) have described a radiological method of measuring this level. They found a lowered diaphragm not only in severe but also in moderate emphysema. If the diaphragm is fully mobile and the lungs can be compressed, little or no bronchial spasm being present, the expiratory movement of the diaphragm must be much greater from a low than from a normal level. Therefore, the reserve air must be larger in an emphysematous subject, if the upward movement of the diaphragm is the main factor in the movement of the reserve air. This is indeed the case, as I have been able to confirm by comparing the thoracometer and spirometric tracings (Herxheimer, 1949). Whereas in the movement of the complementary air the ribs take a great part, the reserve air is mainly moved by the abdominal muscles causing the upward movement of the diaphragm. If, therefore, the diaphragm in emphysema is found low, the reserve air must be expected

FIG. 1.—EFFECT OF EXPIRATORY OBSTRUCTION.—(Upper tracing, thoracogram; lower tracing, spirogram.) From A to B expiration was partially obstructed by a screw clip. Note that the reserve air is increased during this period (the reserve air CD is 76% of the vital capacity against the normal reserve air EF = 36%).

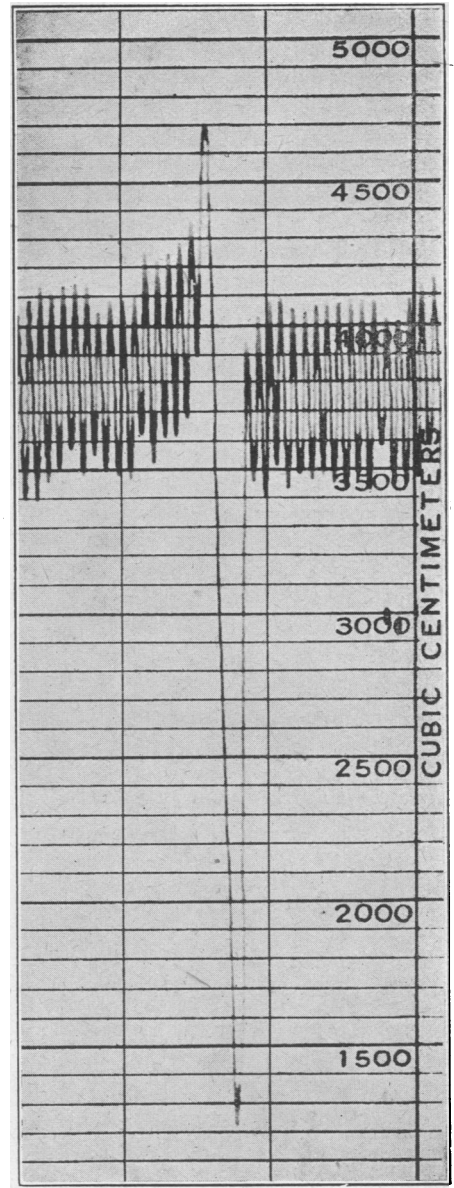
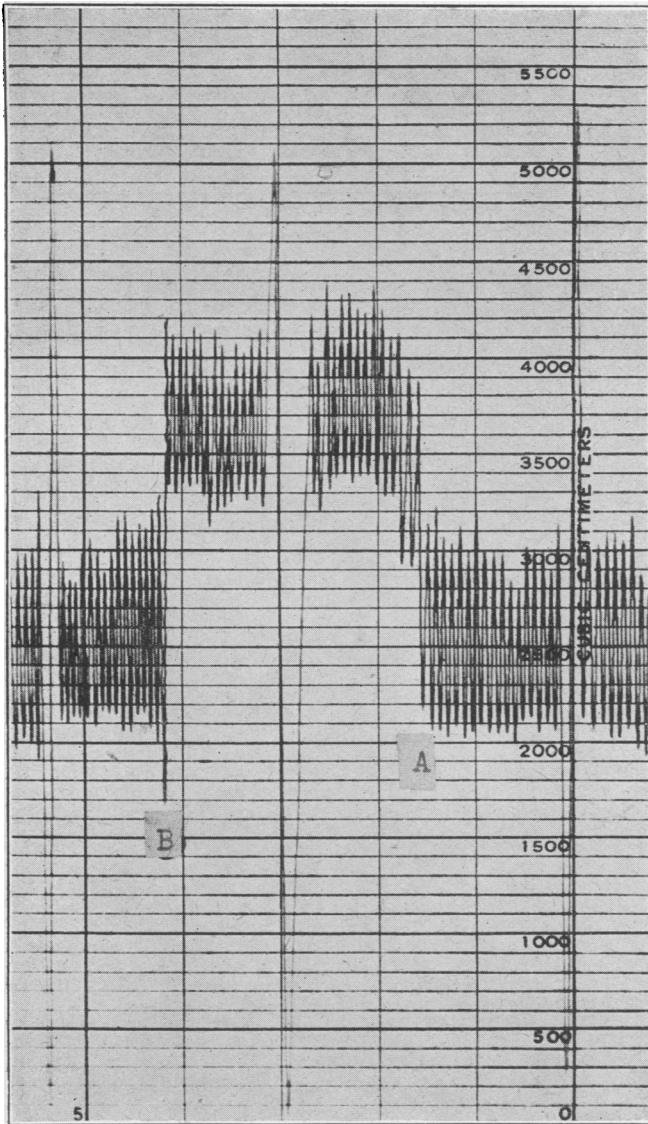


FIG. 2.—NORMAL SUBJECT. Experimental stenosis between A and B. FIG. 3.—EMPHYSEMA. Note the similarity between this tracing and the part from A to B on Fig. 2.

to be large, except in cases in which the chest cannot be emptied normally by muscular effort—e.g., in advanced emphysema with severe bronchospasm.

*Measurement of the Reserve Air.*—In all patients with emphysema seen during the last four years the reserve air has been recorded. Emphysema was assumed to be present if the patient had been

increasingly breathless on exertion for some time, if he complained of cough, and if congestive heart failure and localized pulmonary disease had been excluded. Typical clinical signs were present in many, but often there was only one, and often they were not pronounced. The signs frequently absent were deformity of the chest and hyperresonance. In all cases the diaphragm was found

to be low radiologically, and in many its level had been determined according to the method described by Grossmann and Herxheimer (1948). Asthmatic patients were included either if there was

This history seemed to justify the assumption that some degree of emphysema was present even if its signs were not pronounced. All other asthmatics were excluded, even if they suffered from breathlessness on exertion.

TABLE I  
RESERVE AIR IN NORMAL SUBJECTS

Case No.	Age	Sex	Vital Capacity (ml.)	Reserve Air (Expressed as Percentage of Vital Capacity)
1	15	M	3,905	39
2	15	M	3,500	39
3	15	M	3,990	31
4	15	M	4,505	34
5	16	F	2,890	39
6	39	F	2,380	37
7	17	F	3,335	33
8	16	M	4,280	33
9	16	M	4,480	36
10	23	F	3,230	37
11	23	F	3,740	32
12	16	M	2,960	36
13	17	M	4,580	33
14	21	M	5,020	41
15	21	M	4,800	34
16	21	M	5,290	40
17	21	M	5,565	38
18	12	F	2,180	40
19	12	F	2,040	28
20	11	M	2,055	31
21	9	M	1,945	36
22	12	M	1,965	28
23	12	M	1,700	31
24	12	M	1,870	23
25	12	M	1,870	23
26	17	M	3,170	34
27	12	M	2,400	24
28	22	M	4,665	33
29	21	M	5,480	28
30	21	M	4,410	29
31	22	F	3,270	38
32	21	M	4,475	35
33	17	M	4,740	32
34	22	F	3,535	28
35	21	M	4,120	35
36	21	M	4,370	26
37	21	M	5,470	31
38	21	M	4,400	24
39	22	F	3,430	37
40	21	M	3,800	38
41	26	F	3,870	29
42	58	M	3,230	34
43	53	M	3,880	28
44	16	M	3,775	32
45	22	F	3,100	29
46	25	F	4,640	28
47	25	F	2,800	36
48	22	M	3,410	34

Average reserve air: 33% of vital capacity.

deformity or if they were over 30 years old and had suffered from severe bronchial asthma over long and continuous periods for at least 15 years.

TABLE II  
RESERVE AIR IN EMPHYSEMATOUS AND ASTHMATIC SUBJECTS WITH "NORMAL" AND "HIGH" VITAL CAPACITY

Case No.	Age	Sex	Vital Capacity (ml.)		Reserve Air (Expressed as Percentage of Vital Capacity)
			*Expected	Measured	
1	52	F	2,350	2,415	61
2	34	F	2,460	3,055	45
3	26	M	3,160	3,375	52
4	55	M	2,800	2,955	54
5	?	M	3,130	4,120	45
6	54	M	4,140	3,200	61
7	51	M	3,980	4,380	49
8	40	M	3,300	4,320	43
9	60	M	2,970	3,030	52
10	42	M	3,265	3,465	71
11	23	M	3,190	4,455	47
12	44	F	2,540	2,955	43
13	41	F	2,510	3,045	43
14	34	F	2,810	3,050	58
15	31	F	3,090	3,430	51
16	41	F	2,480	2,755	44
17	37	M	3,180	3,215	38
18	57	M	2,950	3,425	53
19	45	F	2,320	2,625	32
20	46	M	2,950	3,700	50
21	59	M	3,390	4,205	31
22	54	M	3,480	4,470	53
23	38	M	3,550	5,105	50
24	42	F	2,470	2,480	47
25	25	F	2,070	2,440	45
26	42	M	3,080	4,020	50
27	16	M	2,520	2,920	40
28	?	M	3,030	3,510	49
29	31	F	2,460	2,570	41
30	52	F	2,570	3,175	40
31	23	F	2,290	2,870	45
32	36	M	3,540	3,580	48
33	45	F	2,550	2,630	44
34	45	M	3,160	3,340	50
35	65	M	3,515	3,500	55
36	48	M	3,180	3,385	65
37	35	M	4,070	4,070	71
38	31	M	3,740	5,220	64
39	46	M	2,825	3,030	41
40	29	M	3,610	4,840	48
41	35	M	3,625	4,370	32
42	16	M	2,200	2,900	59
43	36	M	3,010	4,600	50
44	16	F	2,105	2,135	46
45	20	F	2,510	2,880	51
46	45	M	3,480	3,500	47

Average reserve air: 49% of vital capacity.

\* Dreyer's tables were used to give this figure.

The vital capacity was used to divide these patients into two groups—those with a normal or high vital capacity and those in whom it was reduced. The vital capacity shows great variability even in normal subjects, and it is hardly possible to predict it accurately by any of the usual formulae based on weight or body surface. It had been noticed that there was a correspondence between the reduction in vital capacity and the disability of the patient. It was, therefore, assumed that those with a reduced vital capacity suffered from a more severe degree of the disease. Dreyer's (1920) standards according to weight were taken as an arbitrary limit of the normal. These values are low when compared with the other standards, and therefore it is reasonably certain that values below this limit represent a reduced vital capacity. All other patients were included in the second group. This group thus consists of patients with "normal" vital capacity (some of whom might range as slightly reduced according to other standards) and with vital capacities higher than that. It must be stressed that all these patients had a typical history of increasing breathlessness on exertion and cough or a long history of asthma. Most of them had some clinical sign of emphysema and all of them had radiologically a low-level diaphragm. In all of them signs of bronchitis were present. I believe that these signs and symptoms are sufficient to make the presence of clinical emphysema probable even if the vital capacity is not reduced. Such cases might reasonably be classed as moderate emphysema; it is possible that they occur much more frequently than is generally assumed. Severe emphysema is known to develop gradually during long periods, and its earlier (moderate) stages are likely to produce less pronounced symptoms and signs than the later ones. The first stages may even cause no symptoms at all and easily escape detection. It seems desirable to detect them, since treatment of

them may offer more hope than that of the severe cases.

A number of normal subjects were also investigated to gain material for comparison. These were mostly students and nurses, but a few school children and some out-patients without gross disability and without chest disease were also included. Table I shows the figures obtained in these "normal" subjects. The reserve air, recorded in the sitting position, lies between 20% and 41% of the vital capacity. This is well in accordance with the figures obtained by Hurtado and others (1934) and Kaltreider and others (1938). Their averages in different groups of normal subjects lie between 17% and 23%, and their individual figures vary between 4% and 38%. As these values were obtained in the supine position, about 12% must be added, according to Kaltreider's and my own

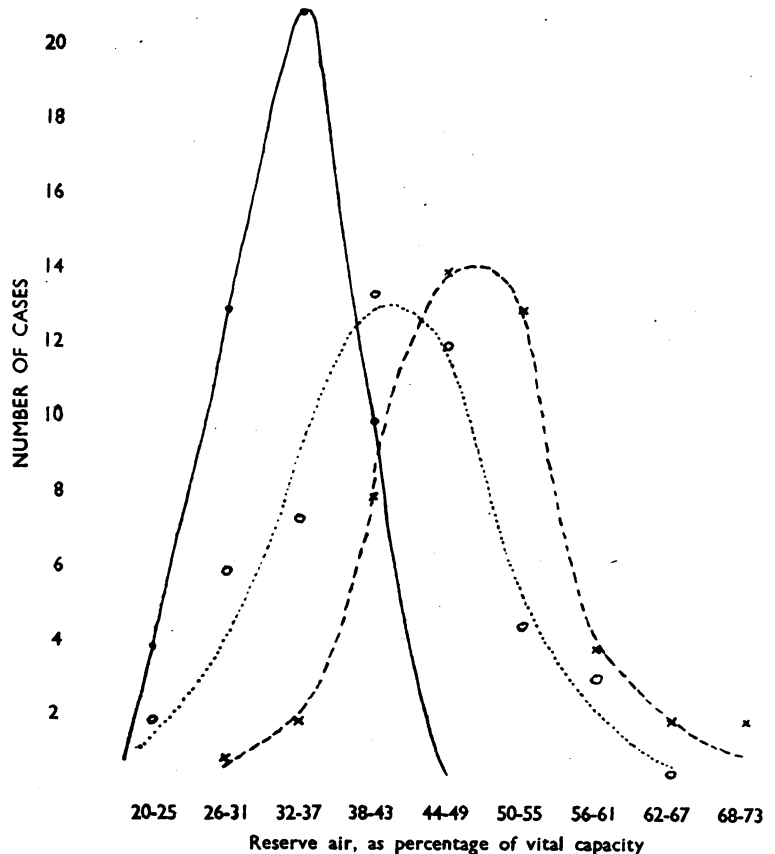


FIG. 4.—Frequency distribution of reserve air percentage in normal and emphysematous subjects. — normal subjects. - - - - emphysematous subjects with normal vital capacity. . . . . emphysematous subjects with reduced vital capacity.

(Herxheimer, 1948) experience, in order to compare them with results obtained in the sitting position. Our average of 33% therefore accords well with the averages of these authors.

Table II shows the results in the patients with "normal" and "high" vital capacity. As mentioned before, these patients were less handicapped by their illness than those whose vital capacity was reduced. Their emphysema was therefore regarded as moderate. Their average reserve air is 49% against 33% in the normal subjects,

as can be seen from the frequency distribution (Fig. 4). The difference seems significant, as almost all the figures lie above the upper limit of the normal values. Only three patients had a reserve air clearly below that limit. Two of them (20 and 41) had an emphysematous-looking chest with an increased antero-posterior diameter. They were asthmatics with comparatively short histories of one and a half and two years, and the short history may account for the absence of emphysema and of an increased reserve air. The third

TABLE III  
RESERVE AIR IN EMPHYSEMATOUS AND ASTHMATIC SUBJECTS WITH "REDUCED" VITAL CAPACITY

Case No.	Age	Sex	Vital Capacity (ml.)		Reserve Air (Expressed as Percentage of Vital Capacity)	Case No.	Age	Sex	Vital Capacity (ml.)		Reserve Air (Expressed as Percentage of Vital Capacity)
			*Expected	Measured					*Expected	Measured	
1	58	F	2,250	1,895	41	40	42	F	3,215	2,850	31
2	49	F	2,845	2,575	33	41	55	M	2,770	1,920	48
3	40	F	3,030	2,770	58	42	57	M	2,990	2,050	58
4	63	F	2,700	1,455	20	43	58	F	2,880	2,160	25
5	52	M	2,825	2,175	30	44	52	M	3,520	1,765	32
6	52	M	3,660	2,635	34	45	57	F	3,000	2,505	41
7	50	M	2,940	2,480	45	46	57	M	3,670	3,280	44
8	45	M	3,330	2,395	50	47	35	M	3,420	3,140	52
9	54	M	3,080	2,860	59	48	43	M	2,810	1,090	43
10	50	F	3,060	2,170	32	49	66	M	3,900	3,150	34
11	25	F	2,550	2,130	48	50	54	M	3,860	3,350	46
12	52	F	2,800	1,945	39	51	26	F	2,940	2,100	28
13	14	M	2,650	2,435	40	52	41	M	3,200	2,675	26
14	24	F	1,990	1,855	47	53	57	M	4,260	3,630	41
15	65	M	3,210	3,075	49	54	52	M	3,770	3,140	40
16	62	M	3,030	2,950	49	55	37	F	2,570	2,560	40
17	56	M	3,030	2,025	56	56	34	F	2,730	2,580	37
18	60	M	3,370	2,540	49	57	53	M	3,480	2,390	54
19	58	M	4,210	2,730	44	58	45	M	3,160	2,490	48
20	34	F	2,780	2,405	40	59	37	F	2,860	1,900	28
21	53	F	2,530	1,940	44	60	23	F	2,090	1,970	46
22	58	M	4,480	3,764	34	61	63	M	3,500	1,770	50
23	55	F	3,575	1,875	26	62	52	M	3,405	2,650	59
24	53	F	2,570	1,930	63	63	34	F	2,140	1,480	51
25	46	M	3,550	2,780	41	64	51	M	2,560	2,310	55
26	46	F	3,540	3,125	29	65	54	M	3,660	3,440	44
27	36	F	2,550	2,465	39	66	33	F	2,230	1,915	37
28	32	M	3,400	2,955	28	67	44	M	3,025	2,730	41
29	55	M	3,500	2,105	44	68	54	M	2,810	2,365	38
30	55	F	1,910	1,450	35	69	49	M	3,790	3,070	21
31	46	M	2,960	2,030	45	70	61	F	1,945	1,435	52
32	42	M	3,025	2,630	30	71	51	M	3,080	1,960	48
33	50	M	3,180	2,950	39	72	56	M	4,400	3,550	38
34	57	M	2,770	1,930	36	73	52	M	4,210	3,510	43
35	51	M	3,150	2,260	38	74	49	F	2,140	1,700	42
36	28	F	3,230	2,550	43	75	38	F	2,450	2,010	34
37	36	F	2,325	1,610	44	76	53	F	2,370	1,300	41
38	46	F	2,615	2,460	38	77	35	F	2,440	1,730	39
39	38	M	3,100	3,065	38						

Average reserve air: 41% of vital capacity.

\* Dreyer's tables were used to give this figure.

case (22) had a long and typical history; radiologically the diaphragm was moderately low, nevertheless the reserve air was normal. I cannot explain this exception from the rule.

Table III shows the results in the patients with reduced vital capacity. It includes 35 cases with a reserve air of over 41%, the upper limit of the normal, and 42 out of the 77 cases lie at or below 41%. The mean of the total is 41% against 33%

in the normal subjects. The patients with a normal reserve air in this table probably have an abnormally large residual air. If they are given an antispasmodic drug and the bronchial spasm nearly always present in emphysema is relieved, vital capacity is found to increase. At the same time there is far more than a mere proportional increase in the reserve air which, in many cases, becomes abnormally large. An example of this is given in Fig. 5.

#### DISCUSSION

It appears from this material that the reserve air has diagnostic importance. If the emphysematous condition (an enlarged mid-capacity) is created artificially, the reserve air increases. It must also be expected to increase as soon as the diaphragm is permanently kept at an abnormally low level. Finally, the expected increase is actually present in all "emphysematous" cases with a normal or high vital capacity. It may seem to be absent in those who have a reduced vital capacity, but in these it is often possible to show that an increased reserve air had been hidden or "masked" by the reduction in vital capacity. If the latter is brought back to normal, the reserve air becomes abnormally large.

This high percentage of reserve air in emphysema is no new discovery. The figures of Hurtado and others (1934) show, in the supine position, an average reserve air of 29.2% of the vital capacity in 26 cases of emphysema against a normal average of 17%–23% in different groups of subjects. Jimenez Diaz and others (1942) also found the reserve air increased. These authors drew no conclusions from this part of their findings. This may be because they did not discriminate between severe and moderate cases, and therefore found many with apparently normal, but in fact "masked" high reserve air.

Our findings have been so regular that they may well improve our chances of recognizing moderate emphysema. We have found it most useful in practice, especially if used in conjunction with the radiological determination of the diaphragmatic level. If this is below normal and the reserve air increased, the diagnosis can be regarded as reasonably certain. It would be desirable to correlate these findings with the results of autopsy, but our material is not yet sufficient for this purpose.

Methodically, there are two difficulties. One is the "unmasking" of apparently normal reserve air. It may not be easy to find the optimum dose of a spasmolytic which increases the vital capacity. Apart from that, the bronchial obstruction

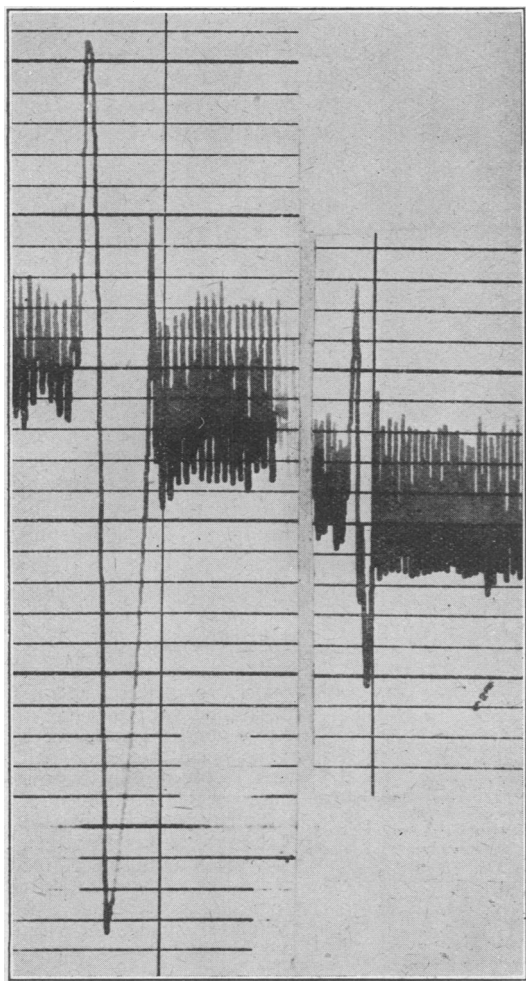
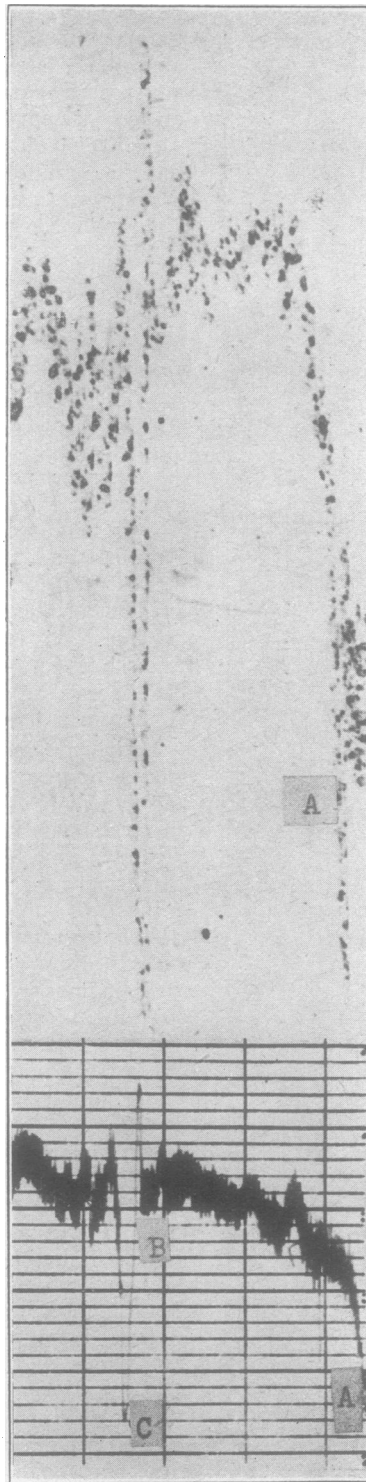


FIG. 5.—EFFECT OF BRONCHIAL SPASM IN "MASKING" A HIGH RESERVE AIR.—The subject, M., showed an asthmatic state and emphysema. The tracing on the right was taken during an acute increase of the bronchospasm: vital capacity 1,340 ml., reserve air 27%. Immediately afterwards 3% isopropyl-adrenaline was inhaled for two minutes and one minute later the tracing on the left was taken. The vital capacity is now 2,920 ml., the reserve air 50%.

may not be the only reason for the failure of the diaphragm to rise; the diaphragm itself may be atrophied and fibrosed (Fromme, 1916; Hitzengerger, 1927) and not capable of relaxing. But in these cases of severe emphysema the diagnostic difficulty is usually not great. If the reduced vital capacity is not caused by emphysema but by congestive failure, the spasmolytic drug used for "unmasking" is usually badly tolerated. In addition, the circulation time, which is prolonged in congestive failure but normal in emphysema, can be used for the differential diagnosis.

Another difficulty is that in a few sensitive patients an increased reserve air may be produced by the small resistance offered by the respiratory apparatus. Although the spirometer used in these investigations has never offered a higher resistance than 1.5 ml. of water, it could be observed by means of the thoracometer that some patients increased their chest circumference during the first minute of breathing and thus artificially produced a large reserve air. The slight resistance in the system acts in these subjects in the same way as a stenosis acts in normal persons (Fig. 1). In Fig. 6 a thoracometer tracing of such a subject sensitive to slight resistance is shown. At first, the patient breathed without the mouthpiece and without connexion with the spirometer. As soon as the mouthpiece was fitted and the connexion established, the chest circumference increased; this indicated that some additional air had been retained in the chest. In the subsequent recording of the subdivisions of the vital capacity, this would



be expected to lead to an increase in the reserve air.

As a rule this phenomenon disappears at the second estimation of the vital capacity. For this reason one should not rely on one set of recordings if there is any suspicion that an increased reserve air might be caused by the resistance of the spirometer.

If the reserve air is significantly increased in emphysema the complementary air would be expected to become correspondingly small. This is indeed the case, and the example in Fig. 3 shows the amount of its reduction, especially if the always variable tidal air be excluded. It might also be argued that the finding of an increased residual air is of a better diagnostic value than the reserve air. But the determination of the residual air in emphysema is more difficult than that of the reserve air; and its results must become more doubtful when bronchial spasm shuts off large lung areas.

FIG. 6.—EFFECT OF RESISTANCE OF SPIROMETER ON RESERVE AIR (upper tracing, thoracogram; lower tracing, spirogram).—Before point A was reached, the subject's breathing had been recorded only by the thoracograph, which does not influence respiration. At A the subject was connected with the mouthpiece of the spirometer. It can be seen from the thoracogram that the chest expands at once, and that the spirogram shows after two minutes the enlarged reserve air typical for emphysema (BC). In this case the transient emphysema has been caused artificially by the resistance of the spirometer. (The line below A in the thoracogram was caused by a movement of the arm during the fitting of the mouthpiece.)



## SUMMARY

The presence of a low level diaphragm in emphysema and the fact, shown in previous investigations, that the reserve air is mainly moved by the diaphragm, suggests that patients with emphysema in whom the respiratory muscles are unimpaired and able to overcome expiratory obstacles such as bronchial spasm must be expected to have an increased reserve air.

In artificial emphysema in normal subjects produced by means of an expiratory stenosis, the spirogram shows an increased reserve air and is in every respect similar to that of genuine emphysema.

A total of 123 emphysematous patients were examined. Of one group of 46 patients with a normal or high vital capacity all but three had an increased reserve air (49% of the vital capacity against 33% in 48 normal subjects). In a second group of 77 patients with reduced vital capacity the reserve air was often found normal.

It was thought that a potentially large reserve air was being masked by inability to overcome bronchial spasm in these cases; the administration

of spasmolytics "unmasked" a large reserve air.

An increased reserve air may be a useful diagnostic sign in emphysema, especially in conjunction with a radiologically determined low level diaphragm.

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