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A Contribution to the Study of the Association of Pulmonary Emphysema and Peptic Ulcer

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The association of peptic ulcer with pulmonary emphysema was first noticed by Green and Dundree (1952). In a series of 700 consecutive autopsies in males, they found that ulcer was approximately three times as frequent in those with emphysema (19%) as in the group as a whole (6.4%).

Their report also included 72 living patients with «chronic pulmonary disease» of whom 14 (19%) had ulcer. Later, in 1955 Weber and Gregg published a well documented report upon the coincidence of benign gastric ulcer and chronic pulmonary disease. In their series of 70 patients with gastric ulcer there were 40 cases of chronic lung disease, mainly chronic obstructive emphysema.

In 1956 Lowell & al. in a special report noted the association of emphysema, peptic ulcer and smoking. Recently Latts and his associates (1956) examined the hospital records of 686 men in whom hypertrophic pulmonary emphysema had been diagnosed, and they discovered that peptic ulceration had also been demonstrated in approximately 1 in 5, and that signs and symptoms suggestive of peptic ulceration had been noted in the same member of cases. The findings were based on clinical evidence in 479 out of 586 cases, and on postmortem evidence in the remaining 107. More patients

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in the postmortem group than in the clinical group had severe emphysema (86°/o compared with 5o°/o) and more of them had peptic ulceration (27°/o compared with 15°/o). 80°/o of the ulcers were in the duodenum. During the period covered by the investigation, peptic ulceration and emphysema were found together 3.6 times as often as could be expected from coincidence.

Recently Plotkin (1957) from the Veteran Administration Hospital, Whipple, Arizona has discussed the association of chronic cor pulmonale and gastroduodenal disease.

«There were 65 cases of pulmonary emphysema and chronic cor pulmonale in 414 necropsies performed in the period 1948-55, and in 27 of these there was gastroduodenal disease, gastric ulcer, mostly on the lesser curvature in the prepyloric region, in 10; duodenal ulcer in 11; and hypertropic gastritis in 6. Death from massive haemorrhage occurred in 7 of the 27 cases and from haemorrhage and perforation in 2, only in 3 cases was the diagnosis established before death. For purposes of comparison the author studied the findings on X-ray examination of the gastroduodenal tract in 65 patients in hospital suffering from pulmonary emphysema and cor pulmonale. It was found that 7 had gastric ulcer, 10 duodenal ulcer, 5 both gastric and duodenal ulceration, and 28 had hypertrophic gastritis. In only a small minority was the presence of gastroduodenal disease suggested by the clinical picture».

Stimulated by these reports, we began to study this problem at the Department of Medicine in Sorraya University Hospital, and in our private clinic. This report deals with all cases of pulmonary emphysema, especially «hypertrophic obstructive type» encountered during a period of 13 months of medical practice. Besides we reviewed our cases of pulmonary emphysema and peptic ulcer in patients hospitalized and seen separately at the Department of Medicine for a period of 12 months. This is a preliminary report and we hope to be able to publish a more detailed report later.

DEFINITION

It is appropriate to give here a brief account of our criteria on the definition and diagnosis of pulmonary emphysema and peptic ulcer.

A) **Pulmonary Emphysema** according to Richards (1955), is a condition in which pulmonary alveoli are abnormally dilated with distension frequently with attenuation and rupture of the alveolar walls. It is classified as acute and chronic; the latter being divided as: 1) Diffused form, 2) Bullous, 3) Localized. The diffused form is of two types: a) Obstructive or hypertrophic, b) Non-obstructive, atrophic or senile type.

Pulmonary Emphysema here we mean the chronic hypertrophic obstructive type. Our criteria in the clinical and radiological diagnosis besides the characteristic dyspnea are the classic data accepted by most textbooks of medicine as follows:

Deformation of the chest wall and diaphragmatic attachement in inspection; absence or decrease of vocal fremitus in palpation especially at the base of both lungs; hyperresonance of the lungs at percussion and decrease of cardiac dullness, decrease or absence of breath sounds at the base with prolonged expiration and faintness of cardiac sounds at the base and apex, maximum of which is best heard at the xiphoid process. The vital capacity and other ventilatory measurement being very much diminished and signs of pulmonary insufficiency present. In X-ray examination widening of intercostal spaces and horizontalization of the ribs, low position of diaphragm, hyperclarity of lung fields and existence of vertical shadow of the heart.

B) **Peptic Ulcer**

By peptic ulcer we mean both gastric and duodenal ulcers with their classical signs, symptoms and radiological appearance.

MATERIAL

We divided our cases into three groups: A) patients having both pulmonary emphysema and peptic ulcer, B) patients having pulmonary emphysema alone and C) patients with proved peptic ulcer alone without any signs of pulmonary emphysema. As we were much interested in the relation, if any, of smoking both to pulmonary emphysema and peptic ulcer on one hand and to the habits of peptic ulcer patients on the other, we included in our study, whenever possible, the amount and kind of smoking used by patients, the special constitution and the appearance of those having peptic ulcer.

RESULTS

Here we summarise the results of our studies in the three following tables.

The first table concerns with patients hospitalized in the Department of Medicine, Sorraya University Hospital, Isfahan, whose files have been studied retrospectively.

All the patients hospitalized in the Department of Medicine of Sorraya University Hospital during one year (from September 23, 1954 until September 23, 1957) amounted to 690, of whom 31 patients had peptic ulcer only (group C), 41 patients with pulmonary emphysema (group B) and 26 patients with both pulmonary emphysema and peptic ulcer (group A).

In brief 9.85°/o of patients had had pulmonary emphysema, 4.55°/o peptic ulcer and 38.8°/o of the above mentioned emphysemateous patients developed peptic ulcer that is to say the chances of acquiring peptic ulcer in emphysemateous patients are 8.6 times more than normal people.

The 2nd table concerns the 1218 outpatients seen at our private clinic who were particularly studied for search of any association

Table I

No.	Name	Age	Sex	Deg. of Smok·Hab.	Const.	Pulmonary Emphys.		Peptic Ulcer	
						Clin.Signs and Symp.	X-Ray Signs	Clin. Signs and Symp.	X-Ray Signs
1	M. H.	60	M	*	D.C.Ect.	+	not perf.	+	not perf.
2	H. S.	60	M	* *	D.C.Ect.	+	» »	−	» »
3	Y. M.	60	M	* *	D.C.Ect.	+	+	−	» »
4	T. G.	62	M	* *	D.C.Ect.	+	+	+	» »
5	G. G.	60	M	−	D.C.Ect.	+	+	−	» »
6	B. N.	65	M	* *	D.C.Ect.	+	not perf.	−	» »
7	M. H.	65	M	−	not det.	−	» »	+	+
8	M. H.	70	M	−	D.C.H.Ect.	+	+	−	not perf.
9	S. N.	65	M	* *	D.C.Ect.	+	not perf.	−	» »
10	N. B.	50	F	−	D.C.Ect.	+	+	−	» »
11	Y. J.	40	M	* * *	D.C.Ect.	+	not perf.	+	+
12	A. A.	56	M	*	D.C.Ect.	+	» »	−	not perf.
13	H. H.	98	M	−	D.C.Ect.	+	» »	−	» »
14	R. S	50	M	* *	D.C.Ect.	+	» »	−	» »
15	R. M.	41	M	* *	D.C Ect.	+	+	−	» »
16	B. A.	51	F	* *	D.C.Ect.	+	+	+	» »
17	K. N	40	F	* * *	D.C.Ect.	+	+	−	+
18	M A.	55	M	* *	D.C.Ect.	+	+	−	+
19	H. F.	50	M	* *	D.C.Ect.	+	+	−	+
20	S. M.	30	F	−	D.C.Ect.	+	+	−	not perf.
21	S. A.	45	M	* * *	D.C.Ect.	+	not perf.	+	+
22	A. R.	52	M	* *	D.C.Ect.	+	» »	−	not perf.
23	M. A.	40	F	−	D.C.Ect.	+	+	−	−
24	N. A.	55	M	* *	D.C.Ect.	+	+	−	not perf.
25	R. A.	60	M	* *	D.C.Ect.	+	not perf.	−	» »
26	H. S.	25	M	* *	D.C.H.Ect.	+	+	−	» »
27	H. K.	40	M	* * *	D.C.Ect.	+	not perf.	+	+
28	G. G.	50	M	* *	D.C.Ect.	+	+	+	+
29	I. K.	50	M	* *	D.C.Ect.	+	+	−	not perf.
30	A. R.	50	M	* *	D.C.Ect.	+	+	+	+
31	J. M.	42	M	−	D.C.Ect.	+	not perf.	−	+
32	I. A.	50	M	* *	D.C.Ect.	+	» »	−	not perf.
33	R. N.	50	M	* * *	D.C.Ect.	+	» »	−	» »
34	H. M.	40	M	* *	D.C.H.Ect.	+	+	+	+

Table I (Continued)

No.	Name	Age	Sex	Deg. of Smok. Hab.	Const.	Pulmonary Emphys.		Peptic Ulcer	
						Clin. Signs and Symp.	X-Ray Signs	Clin. Signs and Symp.	X-Ray Signs
35	H. I.	63	M	* *	D.C.Ect.	+	not perf.	+	not per
36	T. D.	60	M	* *	D.C.H.Ect.	+	+	—	»
37	B. D.	70	F	—	D.C.Ect.	+	not perf.	—	»
38	H. S.	50	F	—	D.C.H.Ect.	+	» »	+	»
39	H. K.	60	M	* *	D.C Ect.	+	» »	—	»
40	K. S.	50	F	—	D.C.Ect.	+	» »	—	»
41	H. G.	60	M	* * *	D.C.Ect.	+	+	+	+
42	M. K.	50	F	—	D.C.	+	not perf.	—	not per
43	A. K.	65	M	* *	D.C.Ect.	+	+	—	»
44	R. C.	55	M	* * *	D.C.H.Ect.	+	+	+	+
45	G. H.	60	M	* *	D.C.H.Ect.	+	not perf.	—	not per
46	A. H.	65	M	* *	D.C.Ect.	+	» »	—	»
47	S. N.	55	M	* *	D.C.	+	» »	—	»
48	N. A.	50	M	* * *	D.C.Ect.	+	+	—	»
49	M. S.	60	M	* *	D.C.	+	+	—	»
50	Y. M.	60	M	* *	D.C.Ect.	+	not perf.	—	»
51	M K.	40	M	* *	D.C.Ect.	—	» »	+	»
52	S. G.	50	M	* *	D.C.Ect.	+	+	—	»
53	H. S.	50	M	* *	D.C.Ect.	+	+	+	»
54	R. H.	47	M	* *	D.C.	+	not perf.	—	»
55	A. S.	30	M	—	D.C.Ect.	—	—	+	+
56	S. Z.	38	M	*	D.C.Ect.	+	+	+	+
57	N. S.	50	M	* *	D.C.Ect.	+	+	—	not por
58	J. A.	50	M	—	D.C.	+	+	—	»
59	Y. M.	40	M	* *	D.C.Ect.	+	not perf.	+	»
60	M. S.	27	F	—	D.C.Ect.	—	» »	+	+
61	A. A.	45	M	—	D.C.	+	» »	—	—
62	H. A.	30	M	* *	D.C.	+	+	—	—
63	A. J.	55	M	—	D.C.	+	not perf.	—	not per
64	M. M.	40	M	* *	D.C.H.Ect.	+	+	—	—
65	A. N.	45	M	* *	D.C.Ect.	+	+	+	not per
66	M. V.	52	F	* * *	D.C.Ect.	+	+	+	+
67	R. A.	60	F	* *	D.C.Ect.	+	+	+	+
68	H. A.	86	F	*	D.C.Ect.	+	+	+	+
69	A. S.	40	M	* * *	D.C.H.Ect.	+	+	+	+
70	A. R.	35	M	* *	D.C.H.Ect.	+	+	+	+

Table II

No.	Name	Age	Sex	Deg. of Smok. Habit	Const.	Pulmonary Emphys.		Peptic Ulcer	
						Clin. Signs and Symp.	X-Ray Signs	Clin. Signs and Symp.	X-Ray Signs
1	H. N.	80	F	not det.	not det.	+	+	+	+
2	A. R.	55	M	« «	« «	+	+	+	+
3	M. K.	50	F	« «	D.C.H.	+	+	+	+
4	G. H.	50	M	* *	D.C.H.	+	+	+	+
5	B. M.	50	F	* *	not det.	+	+	+	+
6	M. A.	75	M	not det.	D.C.H.Ect.	+	+	+	+
7	A. A.	56	M	* *	D.C.H.Ect.	+	+	+	+
8	M. M.	57	F	not det.	D.C.H.Ect.	+	+	+	+
9	J. N.	60	M	* * *	D.C.H.Ect.	+	+	+	+
0	G. G.	56	M	* *	not det.	+	+	+	+
1	H. S.	65	M	* * *	« «	+	+	+	+
2	R. N.	46	M	not det.	D.C.H.	+	+	+	+
3	M. B.	63	M	* *	D.C.H.	+	+	+	+
4	M. Z.	54	M	* * *	D.C.H.Ect.	+	+	+	+
5	H. S.	45	F	not det.	D.C.	+	+	+	+
6	A. S.	55	M	* *	D.C.Ect.	+	+	+	+
7	S. T.	46	F	* *	D.C.	+	+	+	+
8	H. S.	64	M	* *	D.C.	+	+	+	+
9	Z. S	45	F	* *	not det.	+	+	+	+
0	H. R.	80	M	* *	« «	+	+	+	+
1	Y. B.	70	M	* *	D.C.H.Ect.	+	+	+	+
2	H. T.	65	F	not det.	not det.	+	not perf.	+	+
3	H. V.	75	M	* *	D.C.H.	+	+	+	+
4	J. N.	65	M	* * *	not det.	+	+	+	+
5	H. N.	75	M	* * *	« «	+	+	+	+
6	H. G.	65	F	* * *	D.C.H.Ect.	+	+	+	+
7	H. S.	65	M	* * *	not det.	+	+	+	+
8	R. Z.	50	M	* *	« «	+	not perf.	+	+
9	E. J.	28	F	not det.	D.C.H.Ect.	—	« «	+	+
0	R. H.	40	F	—	D.C.H.Ect.	+	+	+	+
1	H. G.	60	F	* * *	D.C.Ect.	+	not perf.	+	+
2	H. R.	60	M	* * *	not det.	+	+	+	+
3	C. M.	68	M	* * *	« «	+	+	+	+
4	H. A.	46	F	not det.	« «	+	+	+	+
5	S. A.	27	M	* *	D.C.H.Ect.	+	+	+	+
6	.. N.	68	M	* *	not det.	+	+	+	+
7	M. A.	57	M	* * *	D.C.H.Ect.	+	+	+	+
8	M. B.	82	M	* * *	D.C.H.Ect.	+	+	+	+
9	A. S.	38	M	* * *	D.C.H.Ect.	+	not perf.	+	+
0	M. N.	21	M	not det.	D.C.H.	—	not perf.	+	+

Table II (Continued)

No.	Name	Age	Sex	Deg. of Smok. Habit	Const.	Pulmonary Emphyse.		Peptic Ulce	
						Clin. Signs and Symp.	X-Ray Signs	Clin. Signs and Symp.	X-Ray
41	I. A.	65	M	* * *	L.C.Ect	+	+	+	+
42	R. N.	50	M	* * *	D.C.H.Ect.	+	+	—	—
43	M. R.	60	F	—	not det.	+	not perf.	+	+
44	K. B.	47	M	* * *	D.C.H.Ect.	+	+	+	+
45	G. K.	36	M	* * *	D.C.H.Ect.	+	+	+	+
46	A. A.	40	M	not det.	not det.	+	+	+	+
47	F. N.	54	M	* * *	D.C.H.Ect.	+	+	+	+
48	H. Z.	75	M	not det.	D.C.H.Ect.	+	+	+	+
49	L. A.	45	M	* * *	D.C H. Ect.	+	—	+	+
50	S. B.	45	F	not det.	not det.	+			

of pulmonary emphysema and peptic ulcer during a period of thirteen months, of whom 50 patients had peptic ulcer (groupC), 48 patients had pulmonary emphysema (group B) and 47 patients had both pulmonary emphysema and peptic ulcer.

The 3rd table concerns the 400 outpatients of our private clinic whose files were retrospectively studied, of whom 15 patients had peptic ulcer only (group C), 16 patients had pulmonary emphysema only (group B), and 16 patients had peptic ulcer and pulmonary emphysema (group A).

The comparison of the two afore said tables indicates that the incidence of peptic ulcer in outpatients is more than in those hospitalized, and the incidence of pulmonary emphysema in hospitalized patients is more than outpatients.

Such discrepancy exists for the simple reason that ulcer patients are not usually willing to be hospitalized unless they develop some complications, and emphysemateous patients especially in advanced stages are unable to walk and thus cannot be treated as ambulatory patients.

Table III

No.	name	Age	Sex	Deg. of Smok. Hab.	Pulmonary Emphys.		Peptic Ulcer	
					Clin. Signs and Symp.	X-Ray Signs	Clin. Signs and Symp.	X-Ray Signs
1	F. M.	63	F	not det.	+	+	—	—
2	A. A	46	M	» »	+	+	—	—
3	M. R.	55	M	» »	+	not perf.	—	—
4	M. G.	40	M	* * *	—	—	+	+
5	S. S.	28	F	not det.	—	—	+	+
6	A. P.	50	M	* * *	+	+	—	—
7	A. V.	36	M	not det.	+	+	—	—
8	F. R.	50	M	* * *	+	+	—	—
9	N. H.	48	F	* *	—	—	+	+
10	A. M.	50	M	* * *	+	+	—	—
11	A. K.	40	M	* * *	+	+	—	—
12	K. A	60	F	* *	+	+	—	—
13	A. K.	52	M	* * *	+	not perf.	—	—
14	S. Z.	35	F	not det.	—	—	+	+
15	R. S.	46	F	* *	—	—	+	+
16	A. F.	28	M	* *	—	—	+	+
17	M. H.	54	M	* * *	+	not perf.	—	—
18	T. H.	25	F	not det.	—	—	+	+
19	H. B	48	M	* *	—	—	+	+
20	J. P.	38	M	* *	—	—	+	+
21	H. M.	56	F	* * *	—	—	+	+
22	G. H.	25	M	* * *	—	—	+	+
23	I. K.	40	F	not det.	—	—	+	+
24	H. A.	35	M	» »	+	+	—	—
25	B. S.	50	F	» »	+	+	—	—
26	K. S.	37	F	» »	+	not perf.	—	—
27	A. H.	39	M	* *	—	—	+	+
28	H. B.	45	M	* *	—	—	+	+
29	K. M.	40	F	not det.	—	—	+	+
30	A. F.	48	F	* * *	+	not perf.	—	—
31	M. P.	39	M	* * *	+	+	—	—
32	K. K.	35	M	not det.	+	not perf.	+	not perf.
33	N. M.	55	M	* * *	+	» »	+	+
34	B. R.	50	M	* * *	+	» »	+	+
35	K. B.	44	F	not det.	+	+	+	+
36	P. Z.	48	F	* * *	+	+	+	+
37	H. H.	45	M	* * *	+	+	+	+
38	A. T.	51	M	* * *	+	+	+	+
39	A. L.	28	M	* * *	+	+	+	+
40	A. K.	47	M	* *	+	+	+	+

Table III (Continued)

No.	Name	Age	Sex	Deg. of Smok. Habit	Pulmonary Emphyse·		Peptic Ulcer	
					Clin. Signs and Symp.	X-Ray Signs	Clin. Signs and Symp.	X-Ray Signs
41	A. S.	80	M	* * *	+	+	+	+
42	A. A.	55	M	* *	+	+	+	+
43	A. C.	40	M	* * *	+	+	+	+
44	M A.	51	M	* * *	+	+	+	+
45	R. S.	50	M	* *	+	not perf.	+	+
46	M. Z.	50	M	* *	+	+	+	+
47	M. S.	50	M	* *	+	+	+	+

Deg. = *degree*
Smok. = *smoking*
Const. = *constitution*
Clin. = *clinical*
F = *female*
M = *male*
det. = *determined*
\+ = *present*
— = *absent*
D. C. = *dark complexion*
L. C. = *light complexion*
H. = *hairy*
Ect. = *ectomorphic*
Perf. = *performed*
G. = *gastric*
* = *the number of asterisk indicates the degree of smoking.*

On the basis of all this we can conclude that:

1) The incidence of peptic ulcer in emphysemateous patients is more than in the general population.

2) 38.8 per cent of the hospitalized patients suffering from pulmonary emphysema had peptic ulcer as well, while this ratio amounted to 99o/o in the patien s attending the most of our private clinic.

3) Ulcer patients, from the point of view of physical constitution and temperament were of a particular type as follows:

They were of asthenic physique, with dark complex on with much body hair (Hirsute) and a state of nervousness and overconscientiousness.

4) A heavy or moderate addiction to smoking (cigarettes, pipes or water-pipe) has been present in the history of most of our patients whether with peptic ulcer or pulmonary emphysema.

4) Of special interest is the fact that the high percentage of group C obtained in table 2 can be explained by the fact that they were especially studied for this purpose, and by the relative insensitivity of end organs of pulmonary emphysema patients to pain. Poor circulation, venous stasis or metabolic changes induced by emphysema may have some bearing on this phenomenon.

DISCUSSION

There appears that a relation exists between pulmonary emphysema and peptic ulcer on one hand and tobacco smoking on the other. The high percentage of emphysemateous patients having peptic ulcer could not be a mere coincidence and there must be some relation between these two conditions.

For a reasonable approach to this problem we can consider two possibilities. They may have some aethiological factors in common or one of the two conditions may predisposes the patient to the other.

The aetiological factors are considered and compared in the table (table 4).

Table IV

Etiology	Occurence	Age	Sex	Heredity	Occupation	Smoking
ulmonary mphysema	5o/o of all autopsies	middle and later life >50	more common in males	nonprobable	chronic pulmonary infection, toxic dusts, etc.	probable
peptic ulcer	10 o/o	all ages	M/F=4/1	probable	no special occupation predisposes to peptic ulcer	probable

It appears therefore that two common factors may exist between the aetiology of pulmonary emphysema and peptic ulcer namely: the sex factor (the preponderance of male to female) and probably smoking. The importance of sex as a factor in production of disease is not quite clear, its effect may be due to hormonal, nervous, psychic, social, etc. changes. It is probable that nervous tension plays a much more dominant role in the male than in female. The relation between peptic ulcer and nervous tension is beyond doubt, although the exact mode of its action is not still clear. But as far as we know there had been no mention in the relation of pulmonary emphysema and nervous system. Perhaps besides the mechanical factors (including occupational and infective causes) that produce pulmonary emphysema in man there exists some nervous factors that cause a state of autonomic nervous system instability and produce over-distention of pulmonary alveoli or aggravate the over-inflation that was already present.

The other factor that may play a role in the production of both pulmonary emphysema and peptic ulcer is excessive smoking. Every one knows the action of tobacco smokes in the respiratory system and on the stimulation of gastric juice and perhaps production of hyperacidity or gastroduodenal irritation, and it would be logical if we presume that the difference of smoking in the two sexes might be the key to the preponderance of both diseases in the male.

Here in Iran smoking is not very popular among females and most of the female smokers are those who use water-pipe only. Most of our patients have been heavy smoker for a relatively long period of their history. But although smoking may play a role in the predisposition of male to pulmonary emphysema and peptic ulcer it does not explain why most of the emphysemateous patients become victims of peptic ulcer. Besides we know several other diseases that are produced or aggravated by tobacco smokes like bronchogenic carcinoma that are not associated with peptic ulcer.

For these reasons we believe that the secret of this association must be somewhat in the special pathology induced by emphysema.

If we consider the aetiology and pathogenesis of peptic ulcer in man we find three pertinent factors, namely:

1) Gastric acid and pepsin:

Peptic ulcer may be produced experimentally by stimulating gastric acidity with parenteral histamin in bees-wax or by the continuous instillation of HCl into the stomach. The average duodenal ulcer patients secrete acid at more than three times the normal rate during the basal fasting state, the 12-hour nocturnal period, after meals or following stimulation by histamin, insulin or caffeine.

In gastric ulcer the free acid is not significantly elevated but is always present in sufficient amount to permit peptic digestion.

2) The decreased tissue resistance:

The factors responsible for the tissue resistance to ulceration are not known. The prospective action of gastric mucusa, the lysozyme (a mucolytic enzyme), the vascular factors; all had been implicated but no single one is accepted by all workers in this field. Perhaps the last factor, in the form of local ischemia or congestion, œdema or venous stasis may play a definite role in the genesis of ulcer and these vascular changes are seen in peptic ulcer induced by steroid hormones.

Other contributing factors altering tissue resistance or increasing gastric secretion are: exposure to cold, anemia, fatigue, infections, malnutrition, dietary indiscretion, excessive smoking, faulty denture and alcoholism.

3(Physical and emotional, stress:

There is considerable evidence that sustained emotional stress (anxiety, fear, worry and frustration) plays a significant role in the pathogenesis and recurrence of peptic ulcer. Although there may be a characteristic personality or emotional pattern, patients with peptic ulcer have been described as overconscientious, compulsive,

perfectionist, aggressive, tense, stubborn, critical, competitive, ambitious, hard working and self-assertive; and this type of personality have been found in most if not all our ulcer patients. Hypersecretion of acid and pepsin, accompanied by vascular changes within this gastric mucosa, has been demonstrated during emotional disturbance (quiet, hostility, fear, anger, resentement, and rage).

The hormonal pathway by which stress may be mediated to the stomach by way of the hypothalamus and pituitary and adrenal glands, resulting in an increase of gastric acid and pepsin and an increase excretion of urinary uropepsin is well illustrated in the following schema quoted from Thomas E. Machella in Harrison principles of internal medicine.

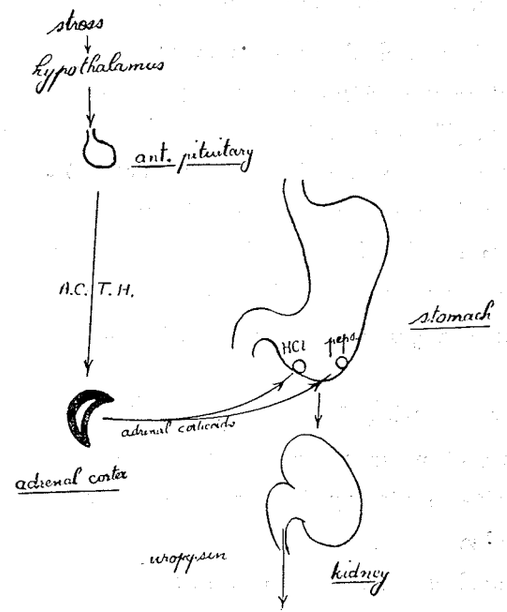

Diagram I.- This hormonal path-way is of course different from the vagus nerve.

Now if we study the effects that peptic ulcer may produce on the body, we can easily deduce that pulmonary emphysema may

enhance the production of peptic ulcer in three ways:

1) Increase of gastric acidity.

2) Decrease of resistance of the gastero-duodenal mucosa.

3) Probably physical and emotional stress induced by pulmonary emphysema.

a) We know beyond any doubt that the chief cause of peptic ulcer is hyperacidity of gastric juice. We know also that there exists a direct relation between the CO2 of the blood and the secretion of acid in the gastric juice; thus, hyperventilation reduces, and breathing C02 rich mixture increases the acid secretion.

Now if we consider changes that occur in obstructive type of emphysema in the blood chemistry, we see that arterial oxyigene saturation is diminished and in advanced cases, elimination of carbon dioxide may be compromised and blood CO2 increased; this engenders a vicious cycle: high CO2 diminishes the sensitivity of the respiratory center, and CO2 retention is thus further increased. The sequence of events thus produced are summarized as follows:

Pulmonary emphysema increase blood CO2 increase HCl in gastric juice predisposition to peptic ulcer.

b) The dyspnea of emphysema and the invalidity and disability produced by such a chronic incapacitating disease may cause powerful stress upon the patient's mind and by way of over stimulation of adrenal cortex and autonomic nervous system, may cause further increase of gastric juice, and probably enhance the production of peptic ulcer.

In brief the association of peptic ulcer to pulmonary emphysema may be explained by the action of three factors concurrently or separately:

1) The increase of blood CO2 in pulmonary emphysema and consequently increase of HCl in gastric juice and predisposition of patient to peptic ulcer.

2) The stress put upon the psyche of emphysemateous patient

by known and unknown pathways increases the gastric HCl further, and make the formation of ulcer more feasible.

3) Smoking may play some role in the production of both pulmonary emphysema and peptic ulcer.

4) Other factors like circulatory changes induced by emphysema, anoxia, hyper-irritability of patients and so on, may have some part in the occurrence of peptic ulcer in emphysemateous patients (see diagram).

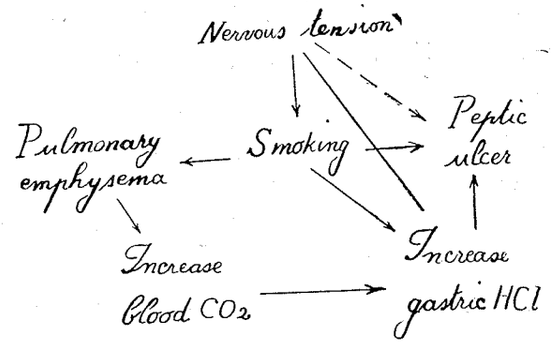

Diagram II.- The dotted line is probabe pathway.

SUMMARY

This is a short survey on association of pulmonary emphysema and peptic ulcer in 680 hospitalized and 1218 private patients. The incidence of peptic ulcer in emphysemateous patient has been approximately evaluated as 38,8o/o in hospital and 99o/o in private patients especially studied for this purpose. The probable cause or causes that may enhance the production of peptic ulcer in emphysemateous persons is discussed. A special constitution has been found in most cases of peptic ulcer; the result of which will be published later on.

In view of the fact that some of the emphysemateous ulcer patients had bronchial asthma in their history, and as such patients may develop no pathognomonic symptoms or signs of peptic ulcer,

steroid hormones are likely to be unduly prescribed for them which may result in the appearance of some of the complications of peptic ulcer such as hemorrhage or perforation. So we recommend that before making quite certain of the absence of the clinical and radiological signs or symptoms of peptic ulcer, any prescription of steroid hormones should strictly be avoided.

CONCLUSION

L' auteur a décrit ses études préliminaires sur l'association de l'emphysème pulmonaire, type hypertrophique et obstructive, avec ulcère peptique gastro-duodénal. Une brève discussion a été faite sur l'explication probable de la pathogénie de l' ulcus chez les emphysémateux.

Il a proposé d'examiner tous les cas d' emphysème au point de vue de l'existence possible de symptômes et signes cliniques et radiologiques de l' ulcus, surtout chez ceux qui ont eu un antécédent asthmatique et un traitement stéroïde.

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Notice :

These observations have been made in the city of Isfahan situated in the central part of Iran at an altitude of 1430 meters above sea level. Most of the patients studied were from Isfahan or its suburbs.

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Uvéite Massive á Pseudo-Cristallin *

G. CHAMS[1] *et G. SADOUGHI*[2]

Voici une maladie très rare dont nous n'avons rencontré qu'un seul cas dans notre service. En 1957, G. Offret et M. Massin nous ont fait un exposé des travaux de P. Michaud et J. Forestier sur ce sujet. Ces deux auteurs, ainsi que quelques autres, classent cette forme d'uvéite parmi les iridocyclites exsudatives, «l'exsudat lentiforme de la chambre antérieure» et la considèrent comme assez caractéristique de l'iritis gonococcique (P. Bonnet). Cette uvéite est rarement observée sous sa forme totale, massive à pseudo-cristallin.

P. Michaud et J. Forestier nous ont rapporté deux observations avec spondylarthrite ankylosante. Ils ont conclu que cette forme d'uvéite appartenait bien à l'uvéite dite «exsudative» appelée encore uvéite diffuse aiguë, particulièrement typique de l'uvéite rhumatismale. Elle éclate comme un coup de tonnerre ce qui montre bien qu'il s'agit d'une manifestation allergique intense de la prise en masse de l'humeur aqueuse, et non pas d'exsudat.

OBSERVATION

J. Sâi, 55 ans, vient nous consulter le 30 octobre 1957 pour une baisse

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