On some forms of sudden death, and sudden death in general / by Alex. Ogston, M.D.

ON

SOME FORMS OF SUDDEN DEATH,

AND

SUDDEN DEATH IN GENERAL.

By Alex. Ogston, M.D., Aberdeen.

TABLE I.—*Of* 580 *cases of sudden death.*

Cause of death.	Number.	Percentage.	Remarks.
Drowning	98	16·89	
Wounds and injuries	87	14·99	
Suffocation	50	8·62	16 of these in new-born children.
Pneumonia	49	8·44	
Apoplexy	43	7·41	3 of these in new-born children.
Still-birth	33	5·68	
Cause undiscoverable	27	4·65	8 of these in adults, chiefly from decomposition, and 19 in new-born children.
Pulmonary apoplexy	23	3·96	
Poisoning	23	3·96	1 of these in a new-born child.
Hanging	17	2·93	
Thrombosis	14	2·41	
Convulsions	12	2·06	
Aneurysm	12	2·06	
Exposure to cold	12	2·06	
Œdema of the lungs	10	1·72	
Encephalitis and brain disease	9	1·55	
Fatty heart	9	1·55	
Mechanical heart-disease	7	1·20	
Peritonitis	5	·86	
Rupture of the heart	4	·68	
Meningitis	3	·51	
Strangulation	3	·51	
Childbed diseases	3	·51	
Bronchitis	3	·51	
Hydrophobia	2	·34	
Burns	2	·34	
Hydrocephalus	2	·34	
Hæmoptysis	2	·34	
Starvation	2	·34	
Erysipelas	2	·34	
Scarlatina	2	·34	
Cellulitis	1	·17	

TABLE I (continued).—*Of* 580 *cases of sudden death*

Cause of death.	Number.	Percentage.	Remarks.
Phthisis	1	·17	
Cholera	1	·17	
Liver disease	1	·17	
Pericarditis	1	·17	
Abortion	1	·17	
Ulceration of intestines	1	·17	
Hæmatemesis	1	·17	
Enteric fever	1	·17	
Old Age	1	·17	
	580	99·89	

TABLE II.—*Of* 326 *cases of sudden death occurring under ordinary circumstances.*

Cause of death.	Number.	Percentage.	Remarks.
Suffocation	50	15·337	16 of these in new-born children.
Pneumonia	49	15·030	
Apoplexy	43	13·190	3 of these in new-born children.
Still-birth	33	10·122	
Cause undiscoverable	27	8·282	8 of these in children and adults, chiefly from decomposition, and 19 in new-born children.
Pulmonary apoplexy	23	7·055	
Thrombosis of heart and main blood-vessels	14	4·294	
Exposure to cold	12	3·681	
Aneurysm	12	3·681	
Œdema of the lungs	10	3·067	
Encephalitis and brain disease	9	2·760	
Fatty heart	9	2·760	
Mechanical heart disease	7	2·147	
Peritonitis	5	1·533	
Rupture of the heart	4	1·227	
Arachnitis and meningitis	3	·920	
Bronchitis	3	·920	
Hydrocephalus	2	·613	
Starvation	2	·613	
Hæmoptysis	2	·613	
Phthisis	1	·307	
Cholera	1	·307	
Liver disease	1	·307	
Pericarditis	1	·307	
Ulceration of intestines	1	·307	
Hæmatemesis	1	·307	
Old age	1	·307	
	326	99·994	

TABLE III.—*Of cases of Sudden Death*

No.	Age.	Sex.	Cause of Death.	Stage and Character of Pneumonia.	Seat and extent of Pneumonia.
1	Adult	M.	Pneumonia	Not given	Not given
2	Do.	F.	Alcoholic poisoning	Red hepatisation	Double, partial
3	Boy	M.	Pneumonia	Gray hepatisation	Right, total
4	6 weeks	M.	Do.	Red hepatisation	Double, partial
5	Adult	F.	Do.	Do.	Left, total; right, partial
6	25	F.	Drowning	Do.	Double, total
7	Adult	M.	Pneumonia	Do.	Left, partial
8	Not given	F.	Do.	Do.	Do.
9	Adult	M.	Do.	Gray hepatisation	Right, total; left, partial
10	12	M.	Thrombosis	Red hepatisation	Double, partial
11	82	M.	Pneumonia	Do.	Left, total
12	70	M.	Do.	Do.	Right, total
13	38	F.	Do.	Do.	Do.
14	Adult	F.	Do.	Do.	Double, partial
15	Do.	M.	Do.	Do.	Double, total
16	Do.	F.	Do.	Do.	Right, total; left, partial
17	Do.	M.	Suffocation in smoke	Do.	Right, partial
18	Do.	M.	Pneumonia	Do.	Left, partial
19	Not given	M.	Do.	Tubercular hepatisation	Double, total
20	Adult	F.	Do.	Red hepatisation	Right, partial
21	Do.	F.	Apoplexy	Do.	Left, partial
22	3 months	F.	Pneumonia	Do.	Left, total
23	Not given	F.	Do.	Do.	Right, partial
24	Do.	F.	Do.	Do.	Double, partial
25	Do.	M.	Meningitis	Do.	Right, partial
26	Adult	F.	Œdema of the lungs	Do.	Left, partial
27	Do.	F.	Hypertrophy of heart	Do.	Double, partial
28	Do.	M.	Pneumonia	Do.	Do.
29	Do.	F.	Exposure to cold	Do.	Right, total
30	Do.	F.	Pneumonia	Gray hepatisation	Do.
31	Do.	M.	Do.	Red and gray hepatisation	Right, total; left, partial
32	Not given	M.	Do.	Red hepatisation	Right, total
33	Adult	M.	Do.	Do.	Left, partial

[1] In this and the following Tables, old adhesions of the pleuræ

where Pneumonia is present.

Pleuræ[1] and Pericardium.	State of Heart and Valves.	Distribution and Character of Blood in the Heart.	Remarks.
Not given	Not given	Not given	
Healthy	Healthy	Clotted, equal on both sides	
Right pleurisy	Do.	Do.	
Healthy	Do.	Only on the right side	
Right pleurisy	Valvular disease	Not given	Mitral and tricuspid vegetations.
Healthy	Healthy	Only on the right side	
Right pleurisy	Do.	Equal on both sides	
Left pleurisy	Do.	Chiefly on the right side	
Double pleurisy	Do.	Do.	
Healthy	Do.	Thrombosis, chiefly on right side	
Left pleurisy	Do.	Fluid, equal on both sides	
Healthy	Do.	Only on right side	Thrombosis of right heart.
Right pleurisy	Do.	Not given	
Double pleurisy	Do.	Chiefly on the right side	Do.
Pericarditis	Hypertrophy of heart	Do.	Tricuspid valve fenestrated.
Healthy	Healthy	Do.	
Pleuræ and pericardium red	Do.	Do.	
Healthy	Do.	Do.	
Do.	Do.	Only on the right side	
Double pleurisy	Do.	Not given	Pericarditis also present.
Healthy	Do.	Do.	
Do.	Do.	Fluid, chiefly on right side	
Do.	Do.	Equal on both sides	Blood clotted in left, fluid in right heart.
Do.	Do.	Chiefly on the right side	Thrombus in pulmonary artery.
Do.	Do.	Do.	Thrombus in the right heart.
Fluid in left pleura	Fatty heart, and valv. dis.	Clotted, chiefly on right side	Tubercular mass in lower lobe of left lung.
Healthy	Heart large; valves healthy	Fluid, chiefly on right side	
Do.	Heart fatty	Fluid, equal on both sides	
Do.	Healthy	Thrombosis on both sides	Tubercles in apex of right lung.
Right pleurisy	Do.	Thrombosis on right side	
Healthy	Do.	Fluid, equal on both sides	
Right pleurisy	Do.	Fluid, chiefly on right side	
Healthy	Heart fatty	Empty of blood	Extensive pulmonary apoplexy of right lung.

are disregarded, and such pleuræ are noted as healthy.

No.	Age.	Sex.	Cause of Death.	Stage and Character of Pneumonia.	Seat and Extent of Pneumonia.
34	Adult	M.	Pneumonia	Red hepatisation	Double, partial
35	Child	M.	Do.	Do.	Do.
36	Do.	M.	Do.	Do.	Do.
37	Adult	M.	Exposure to cold	Do.	Left, partial
38	Infant	M.	Pneumonia	Do.	Double, partial
39	Adult	F.	Do.	Do.	Right, total; left, partial
40	Do.	M.	Do.	Do.	Left, partial
41	Do.	F.	Do.	Gray hepatisation	Right, total; left, partial
42	Do.	F.	Do.	Red hepatisation	Left, partial
43	Do.	M.	Do.	Do.	Double, partial
44	Do.	F.	Do.	Gray hepatisation	Left, total; right, partial
45	Not given	F.	Do.	Red hepatisation	Double, total
46	Adult	M.	Suffocation	Do.	Double, partial
47	Not given	M.	Softening of the brain	Do.	Left, partial
48	Adult	M.	Pneumonia	Do.	Double, total
49	Do.	F.	Do.	Do.	Left, total
50	Do.	F.	Do.	Do.	Left, total; right, partial
51	Do.	F.	Do.	Do.	Double, partial
52	Do.	M.	Do.	Do.	Double, total
53	65	M.	Aneurism	Gray hepatisation	Right, total
54	Adult	M.	Apoplexy	Red hepatisation	Right, total
55	10 weeks	F.	Pneumonia	Do.	Double, total
56	62	M.	Do.	Do.	Left, partial
57	Adult	F.	Do.	Do.	Double, total
58	2	M.	Do.	Do.	Left, total; right, partial
59	Adult	F.	Do.	Do.	Do.
60	40	M.	Pulmonary apoplexy	Do.	Right, partial
61	58	M.	Apoplexy	Do.	Double, partial
62	75	M.	Pneumonia	Gray hepatisation	Right, partial
63	58	F.	Exposure to cold	Red hepatisation	Double, partial
64	45	F.	Pneumonia	Do.	Double, total
65	47	F.	Do.	Red and gray hepatisation	Double, partial
66	65	M.	Do.	Red hepatisation	Do.
67	20	F.	Œdema of the lungs	Do.	Left, partial

Pleuræ[1] and Pericardium.	State of Heart and Valves.	Distribution and Character of Blood in the Heart.	Remarks.
Healthy	Healthy	Fluid, chiefly on right side	
Do.	Do.	Thrombosis of right side	
Do.	Do.	Fluid, chiefly on right side	Thrombus in right heart.
Do.	Do.	Equal and excessive on both sides	Thrombi on both sides of heart.
Do.	Do.	Chiefly on the right side	Thrombus in right heart.
Do.	Do.	Fluid, chiefly on right side	
Double pleurisy	Mit. and aort. disease	Do.	
Healthy	Healthy	Chiefly on the right side	Blood clotted in right, fluid in left heart.
Do.	Heart pale and flabby	Fluid, chiefly on right side	
Do.	Heart fatty	Do.	
Do.	Do.	Do.	
Double pleurisy	Healthy	Semi-fluid, chiefly on right side	
Healthy	Do.	Fluid, chiefly on right side	
Do.	Do.	Do.	
Do.	Do.	Do.	
Do.	Do.	Equal on both sides	Pulmonary apoplexy of lower two lobes right lung. Thrombosis of heart.
Do.	Do.	Semi-fluid, chiefly on right side	
Do.	Do.	Fluid, equal on both sides	
Do.	Do.	Fluid, entirely on right side	
Do.	Do.	Fluid, scanty on both sides	Rupture of iliac aneurism behind peritoneum.
Do.	Do.	Fluid, chiefly on right side	
Do.	Do.	Fluid, equal on both sides	
Do.	Aort. and mit. disease	Clotted, equal on both sides	Heart fatty.
Do.	Heart fatty	Semi-fluid, chiefly on right side	
Do.	Healthy	Clotted, chiefly on right side	
Do.	Heart fatty	Do.	Aortic and mitral disease.
Do.	Healthy	Fluid, equal on both sides	
Do.	Heart fatty	Only on the right side	Thrombosis of right heart.
Do.	Do.	Chiefly on the right side	Thrombosis of the heart.
Do.	Healthy	Abundant on both sides	
Do.	Heart fatty	Fluid, chiefly on right side	
Do.	Healthy	Clotted, chiefly on right side	
Do.	Heart fatty	Semi-fluid, equal on both sides	Do.
Do.	Do.	Fluid, equal on both sides	

TABLE IV.—*Pulmonary Apoplexy.*

No.	Sex.	Age.	Cause of Death.	Seat and Extent of Pulmonary Apoplexy.	Complications of Lungs, Pleuræ, and Pericardium.	Appearances in Heart and Blood-vessels.
1	M.	56	Apoplexy	Left, total	None	Blood chiefly on right side.
2	M.	39	Injuries of chest	Left, partial	Do.	Blood equal on both sides.
3	M.	Adult	Pulmonary apoplexy	Double, partial	Do.	Blood chiefly on right side.
4	M.	10	Stabbing	Right, partial	Pleurisy, hæmothorax	Thrombus in right heart.
5	F.	Adult	Fracture of skull	Left, partial	None	Blood fluid, chiefly on right side.
6	M.	Do.	Hæmoptysis	Double, partial	Pleurisy, double	Do.
7	F.	Do.	Pulmonary apoplexy	Left, partial	Adherent pericardium	Enlarged heart.
8	M.	Unknown	Do.	Do.	None	Fatty heart, blood chiefly on right side.
9	M.	Do.	Do.	Right, total	Do.	Fatty heart, blood clotted, chiefly on right side.
10	M.	Adult	Peritonitis	Left, total	Do.	Heart empty of blood.
11	F.	40	Thrombosis	Left, partial	Œdema of lungs	Thrombosis of right heart and pulmonary artery.
12	M.	Adult	Pulmonary apoplexy	Double, total	None	Fatty heart.
13	M.	Do.	Do.	Right, total; left, partial	Do.	Fatty heart. Thrombosis of right side.
14	M.	45	Do.	Double, partial	Do.	Blood chiefly on right side.
15	F.	16	Do.	Do.	Œdema of lungs	Blood fluid, chiefly on right side.
16	M.	Adult	Pneumonia	Right, partial	Œdema and hep. of lungs	Heart fatty, with little blood.
17	F.	Do.	Pulmonary apoplexy	Right, total	None	Heart empty.
18	F.	Do.	Do.	Double, partial	Do.	Blood fluid, chiefly on right side.
19	M.	Do.	Fracture of skull	Do.	Do.	Blood fluid, equal on both sides.
20	M.	Do.	Pulmonary apoplexy	Do.	Œdema of lungs	Blood fluid, only on right side.
21	F.	Do.	Do.	Right, total; left, partial	None	Heart fatty. Blood fluid, chiefly on right side.
22	F.	Do.	Pneumonia	Right, partial	Red hepatisation	Thrombosis on both sides of the heart.
23	M.	Do.	Pulmonary apoplexy	Double, total	None	Blood fluid, only on right side.
24	M.	24	Do.	Left, total; right, partial	Do.	Heart fatty. Blood chiefly on right side.
25	M.	75	Do.	Right, total; left, partial	Do.	Blood fluid, chiefly on right side.
26	M.	Adult	Do.	Do.	Œdema of lungs	Blood clotted, only on right side.
27	M.	40	Do.	Double, partial	Red hepatisation	Blood fluid, equal on both sides.
28	M.	50	Do.	Left, total; right, partial	None	Blood fluid, chiefly on right side.
29	M.	39	Fracture of skull	Left, partial	Œdema and hep. of lungs	Thrombosis of right heart.

30	M.	67	Pulmonary apoplexy	Double, partial	None	Blood fluid, scanty on both sides.
31	M.	25	Do.	Do.	Do.	Blood fluid, equal on both sides.
32	M.	56	Do.	Do.	Do.	Thrombosis of right heart.
33	M.	37	Fracture of skull	Double, total	Do.	Blood fluid, chiefly on right side.
34	M.	63	Pulmonary apoplexy	Left, total ; right, partial	Do.	Heart empty of blood.
35	M.	73	Fatty heart	Left, partial	Do.	Heart fatty. Blood fluid, equal on both sides.
36	F.	Adult	Pulmonary apoplexy	Left, total	Do.	No blood in the heart.

TABLE V.—*Thrombosis of the Heart and Main Blood-vessels.*

No.	Age.	Sex.	Cause of Death.	Situation of Thrombus.	State of Heart.	State of Cardiac Valves.	Condition of Lungs.
1	12	M.	Thrombosis	Both sides and their vessels	Healthy	Normal	Congested and hepatised posteriorly.
2	2½	F.	Rupture of liver	Right cavities	Do.	Do.	Anæmic.
3	73	F.	Sores after injuries	Right ventricle	Do.	Do.	Normal.
4	70	M.	Pneumonia	Right cavities	Do.	Do.	Right lung hepatised and congested. Left lung congested.
5	28	M.	Thrombosis	Cavities on both sides	Do.	Do.	Normal.
6	Adult	F.	Pneumonia	Right cavities	Do.	Do.	Lower lobes hepatised. Remainder œdematous.
7	Do.	M.	Scarlatina after injuries	Do.	Do.	Do.	Normal.
8	38	M.	Hanging	Do.	Do.	Do.	Do.
9	Adult	F.	Injuries of larynx	Do.	Do.	Do.	Œdematous.
10	Do.	F.	Thrombosis	Do.	Do.	Mitral and tricusp. thickened	Congested. Left lung œdematous.
11	10	M.	Stab in chest and liver	Do.	Do.	Normal	Pulmonary apoplexy of right lower lobe.
12	Not given	M.	Pericarditis	Do.	Do.	Do.	Tubercular.
13	Adult	M.	Erysipelas after injuries	Do.	Do.	Do.	Normal.
14	Do.	F.	Hæmorrhage from wounds	Left auricle	Do.	Do.	Do.
15	Not given	F.	Pneumonia	Pulmonary artery	Do.	Do.	Lower lobes hepatised. Remainder emphysematous.
16	Adult	F.	Arsenical poisoning	Right vessels and cavities	Do.	Do.	Congested and emphysematous.

No.	Age.	Sex.	Cause of Death.	Situation of Thrombus.	State of Heart.	State of Cardiac Valves.	Condition of Lungs.
17	17	M.	Exposure to cold	Right cavities	Healthy	Normal	Anæmic.
18	70	F.	Do.	Do.	Right side thinned	Aortic and tricusp. thickened	Normal.
19	83	F.	Do.	Both sides and their vessels	Fatty	Mitral and tricusp. thickened	Anæmic.
20	Adult	F.	Rupture of uterus	Right cavities	Healthy	Normal	Normal.
21	Do.	M.	Suffocation when in drink	Cavities on both sides	Do.	Do.	Do.
22	Do.	F.	Fatty heart	Left cavities	Fatty	Do.	Do.
23	Do.	F.	Liver disease	Right vessels and cavities	Healthy	Do.	Do.
24	Child	M.	Starvation	Right ventricle	Do.	Do.	Do.
25	14	F.	Phosphorus poisoning	Right cavities	Do.	Do.	Do.
26	3	M.	Thrombosis	Right vessels and cavities	Do.	Do.	Congested and œdematous.
27	Adult	M.	Do.	Right cavities	Fatty	Do.	Congested. Left lung œdematous.
28	Not given	M.	Meningitis	Do.	Healthy	Do.	Lower and middle lobes of right lung hepatised.
29	Do.	F.	Hypertrophy of the brain	Do.	Do.	Do.	Congested.
30	4½ months	M.	Starvation	Do.	Do.	Do.	Normal.
31	Adult	F.	Exposure to cold	Cavities on both sides	Do.	Do.	Left lung emphysematous. Right lung hepatised.
32	40	F.	Thrombosis	Right vessels and cavities	Do.	Do.	Right lung œdematous. Pulmonary apoplexy of left lower lobe.
33	Adult	F.	Pneumonia	Do.	Do.	Do.	Left lung congested. Right lung hepatised and œdematous.
34	60	M.	Thrombosis	Both sides and their vessels	Fatty	Aortic bony and insufficient	Much œdematous and congested.
35	Adult	M.	Do.	Right cavities	Do.	Normal	Do.
36	Do.	M.	Pulmonary apoplexy	Right vessels and cavities	Do.	Do.	Pulmonary apoplexy of right, and back of left lung.
37	Do.	F.	Aortic aneurism	Left ventricle	Healthy	Do.	Normal.
38	Not given	F.	Apoplexy	Right cavities	Do.	Do.	Do.
39	Child	M.	Pneumonia	Do.	Do.	Do.	Middle right, and upper left lobe hepatised. Remainder œdematous.

40	Child	M.	Pneumonia	Right cavities	Healthy	Normal	Lower lobes hepatised. Remainder œdematous.
41	Adult	M.	Exposure to cold	Both sides and their vessels	Do.	Do.	Lower left lobe hepatised.
42	Do.	M.	Encephalitis	Cavities on both sides	Do.	Do.	Emphysematous.
43	Infant	M.	Pneumonia	Right cavities	Do.	Do.	Hepatisation of both lungs, except upper lobes.
44	Adult	M.	Cut throat	Do.	Do.	Do.	Normal.
45	Do.	F.	Thrombosis	Do.	Do.	Do.	Tubercular and œdematous.
46	35	F.	Fracture of the skull	Do.	Do.	Do.	Congested and œdematous.
47	Adult	F.	Lung disease. Pneumonia	Cavities on both sides	Do.	Do.	Left lung hepatised. Pulmonary apoplexy of right upper lobes.
48	4½	F.	Poisoning with laurel	Do.	Do.	Do.	Normal.
49	25	F.	Arachnitis	Right cavities	Do.	Do.	Do.
50	58	M.	Apoplexy	Do.	Fatty	Do.	Back parts hepatised.
51	Adult	F.	Thrombosis	Do.	Healthy	Do.	Excessively œdematous.
52	75	M.	Pneumonia	Cavities on both sides	Fatty	Thickened	Gray hepatisation of right upper lobe. Remainder œdematous.
53	39	M.	Fracture of the skull	Right cavities	Healthy	Normal	Œdematous, congested, and hepatised behind. 2. Apoplectic depôts.
54	58	F.	Exposure to cold	Do.	Do.	Do.	Hepatised behind.
55	84	M.	Thrombosis	Both auricles and the vessels	Fatty	Do.	Œdematous.
56	65	M.	Pneumonia	Both sides and their vessels	Do.	Mitral and aortic thickened	Lower lobes hepatised. Upper lobes œdematous.
57	55	M.	Fracture of the skull	Pulmonary artery	Healthy	Normal	Œdematous.
58	70	M.	Drowning	Right auricle	Do.	Do.	Do.
59	49	M.	Thrombosis	Both sides and their vessels	Fatty	Do.	Do.
60	65	M.	Exposure to cold	Left auricle	Healthy	Do.	Normal.
61	56	M.	Pulmonary apoplexy	Right cavities	Do.	Do.	Pulmonary apoplexy of lower lobes and right middle lobe.
62	73	F.	Thrombosis	Both sides and their vessels	Fatty	Do.	Congested.
63	68	M.	Do.	Do.	Healthy	Do.	Œdematous.

TABLE VI.—

No.	Age.	Sex.	Cause of Death.	Situation of Œdema.	Lung Complications.	Pleural Complications.
1	6 weeks	M.	Pneumonia	Double	Hepatisation	None
2	Adult	F.	Do.	Do.	Do.	Pleurisy. Containing fluid [1]
3	Do.	M.	Do.	Right	Do.	Right pleurisy
4	Not given	F.	Do.	Double	Do.	Left pleurisy
5	Adult	M.	Do.	Left	Do.	Double pleurisy
6	76	M.	Fatty heart	Double	None	Serum in right pleura
7	70	M.	Pneumonia	Right	Hepatisation	None
8	Adult	F.	Do.	Double	Do.	Double pleurisy
9	Do.	M.	Do.	Do.	Do.	None
10	Do.	F.	Injuries	Do.	None	Do.
11	Do.	F.	Thrombosis	Left	Do.	Do.
12	Not given	F.	Brain disease	Double	Do.	Do.
13	Adult	F.	Pneumonia	Right	Hepatisation	Pleurisy. Containing fluid
14	3 months	F.	Do.	Double	Do.	None
15	Adult	M.	Fractured ribs	Right	None	Hæmothorax
16	Do.	F.	Œdema of lungs	Double	Do.	None
17	Do.	F.	Suffocation	Do.	Do.	Do.
18	Do.	M.	Fracture of skull	Do.	Do.	Do.
19	Do.	M.	Apoplexy	Left	Do.	Do.
20	Do.	M.	Œdema of lungs	Double	Do.	Do.
21	Child	F.	Do.	Do.	Do.	Do.
22	Adult	M.	Do.	Do.	Do.	Do.
23	3	M.	Thrombosis	Do.	Do.	Do.
24	Adult	M.	Do.	Left	Do.	Do.
25	Do.	M.	Poisoning by morphia	Double	Do.	Do.
26	Do.	F.	Œdema of lungs	Do.	Hepatisation	Fluid in pleuræ
27	Do.	F.	Pericarditis	Do.	Tubercular consolidation	None
28	Do.	M.	Pneumonia	Do.	Hepatisation	Do.
29	40	F.	Thrombosis	Right	Pulm. apoplexy	Do.
30	Adult	F.	Pneumonia	Do.	Hepatisation	Do.
31	Do.	M.	Hypertrophy of heart	Do.	None	Do.
32	60	M.	Thrombosis	Double	Do.	Do.
33	Adult	M.	Do.	Do.	Do.	Do.
34	Do.	M.	Pneumonia	Right	Hepatisation	Do.
35	Infant	F.	Bronchitis	Double	None	Do.
36	Not given	M.	Pneumonia	Do.	Hepatisation	Pleurisy, containing fluid
37	Adult	M.	Fatty heart	Do.	None	None
38	Do.	M.	Pneumonia	Left	Hepat. and pulm. apoplexy	Do.
39	Do.	M.	Do.	Double	Hepatisation	Do.

[1] Serum in the pleuræ or pericardium

Œdema of the Lungs.

State of Heart and Pericardium.	State of Blood in the Heart.	Distribution of Blood in Heart.	State of Cardiac Valves.
Healthy.	Not given	Only on right side	Healthy.
Dilatation of heart	Do.	Not given	Thickened mitral and tricuspid.
Healthy	Fluid	Equal on both sides	Healthy.
Do.	Clotted and fluid	Chiefly on left side	Do.
Do.	Clotted	Chiefly on right side	Do.
Heart large & fatty	None	Both sides empty	Do.
Serum in pericard.	Fluid and thrombosis	Only on right side	Bony points in mitral.
Healthy	Thrombosis	Equal on both sides	Healthy.
Serum in pericard.	Clotted	Chiefly on right side	Do.
Healthy	Thrombosis	Only on right side	Do.
Do.	Fluid and thrombosis	Chiefly on right side	Thickened mitral and tricuspid.
Do.	Fluid	Do.	Healthy.
Pericarditis	Not given	Not given	Do.
Serum in pericard.	Fluid	Chiefly on right side	Do.
Healthy	Clotted	Equal on both sides	Do.
Do.	Fluid	Chiefly on right side	Do.
Do.	Do.	Do.	Do.
Do.	Do.	Only on right side	Do.
Do.	Do.	Chiefly on right side	Do.
Do.	Do.	Only on right side	Insufficient aortic valves.
Do.	Do.	Do.	Healthy.
Heart fatty	Do.	Chiefly on right side	Do.
Healthy	Thrombosis	Only on right side	Do.
Heart fatty	Fluid and thrombosis	Equal on both sides	Do.
Do.	Clotted	Only on right side	Do.
Do.	Do.	Chiefly on right side	Thickened aortic and tricuspid.
Pericarditis	Fluid	Do.	Insufficient aortic valves.
Heart fatty	Do.	Equal on both sides	Healthy.
Healthy	Fluid and thrombosis	Both sides full	Do.
Do.	Do.	Chiefly on right side	Do.
Adherent pericard.	Fluid	Equal on both sides	Thickened mitral and aortic.
Heart fatty	Clot and thrombosis	Chiefly on right side	Insufficient aortic.
Do.	Fluid and thrombosis	Do.	Healthy.
Healthy	Fluid	Equal on both sides	Do.
Do.	Clotted and fluid	Do.	Do.
Do.	Fluid	Chiefly on right side	Do.
Heart fatty	Do.	Equal on both sides	Do.
Do.	Do.	Both sides empty	Calcareous aortic
Healthy	Do.	Chiefly on right side	Healthy.

is mentioned only when considerable.

No.	Age.	Sex.	Cause of Death.	Situation of Œdema.	Lung Complications.	Pleural Complications.
40	Child	M.	Pneumonia	Double	Hepatsiation	None
41	Do.	M.	Do.	Do.	Do.	Do.
42	Adult	M.	Œdema of lungs	Do.	None	Do.
43	Do.	M.	Pulmonary apoplexy	Right	Pulm. apoplexy	Do.
44	Do.	F.	Pneumonia	Double	Hepatisation	Do.
45	Do.	F.	Do.	Do.	Do.	Do.
46	Do.	M.	Do.	Do.	Do.	Do.
47	Do.	F.	Do.	Do.	Do.	Do.
48	Do.	F.	Thrombosis	Left	Tubercle	Do.
49	35	F.	Fracture of skull	Right	None	Do.
50	Adult	F.	Peritonitis	Double	Do.	Do.
51	Do.	F.	Pneumonia	Right	Hepatisation	Do.
52	Do.	F.	Do.	Double	Do.	Do.
53	Do.	M.	Do.	Do.	Do.	Do.
54	Do.	M.	Pulmonary apoplexy	Left	Pulm. apoplexy	Do.
55	65	M.	Rup. of iliac aneurism	Right	Hepatisation	Do.
56	Adult	M.	Apoplexy	Do.	Do.	Do.
57	10 weeks	F.	Pneumonia	Double	Do.	Do.
58	5 weeks	M.	Smothering	Do.	None	Do.
59	62	M.	Pneumonia	Do.	Hepatisation	Do.
60	Adult	F.	Do.	Do.	Do.	Do.
61	49	F.	Œdema of lungs	Do.	None	Do.
62	Adult	F.	Thrombosis	Do.	Do.	Fluid in pleuræ
63	75	M.	Pneumonia	Do.	Hepatisation	None
64	39	M.	Fracture of skull	Do.	Hepat. and pulm. apoplexy	Do.
65	60	F.	Fatty heart	Do.	None	Do.
66	65	F.	Old age	Right	Do.	Do.
67	84	M.	Thrombosis	Double	Do.	Do.
68	47	F.	Pneumonia	Left	Hepatisation	Do.
69	65	M.	Do.	Double	Do.	Do.
70	64	F.	Œdema of lungs	Do.	None	Fluid in pleuræ
71	55	M.	Fracture of skull	Do.	Do.	None
72	44	M.	Œdema of lungs	Do.	Do.	Fluid in pleuræ
73	49	M.	Thrombosis	Do.	Do.	None
74	68	M.	Do.	Do.	Do.	Do.
75	20	F.	Œdema of lungs	Do.	Hepatisation	Do.
76	42	M.	Apoplexy	Do.	None	Do.

State of Heart and Pericardium.	State of Blood in the Heart.	Distribution of Blood in Heart.	State of Cardiac Valves.
Healthy	Fluid and thrombosis	Chiefly on right side	Healthy.
Do.	Do.	Do.	Do.
Heart fatty	Clotted and fluid	Do.	Do.
Healthy	Fluid	Only on right side	Do.
Do.	Clotted and fluid	Chiefly on right side	Do.
Do.	Fluid	Do.	Do.
Heart fatty	Do.	Do.	Do.
Do.	Do.	Equal on both sides	Do.
Healthy	Clot, fluid, and thromb.	Chiefly on right side	Do.
Do.	Thrombosis	Do.	Do.
Do.	Clotted and fluid	Do.	Do.
Do.	Do.	Do.	Do.
Do.	Fluid	Equal on both sides	Do.
Do.	Do.	Only on right side	Do.
Heart fatty	Clotted	Chiefly on right side	Thickened mitral.
Healthy	Fluid	Equal on both sides	Healthy.
Heart fatty	Fluid and clotted	Chiefly on right side	Thickened mitral and tricuspid.
Healthy	Fluid	Equal on both sides	Healthy.
Do.	Do.	Chiefly on right side	Do.
Heart fatty	Clotted	Equal on both sides	Thickened aortic and mitral.
Do.	Do.	Chiefly on right side	Do.
Healthy	Fluid	Equal on both sides	Thickened mitral.
Do.	Fluid and thrombosis	Chiefly on right side	Ossified aortic valve.
Heart fatty	Clotted and thrombosis	Do.	Thickening of all valves.
Healthy	Clotted, fluid, and thrombosis	Do.	Healthy.
Heart fatty	Fluid	Equal on both sides	Do.
Healthy	No blood	Both sides empty	Thickened mitral and aortic.
Heart fatty	Clotted and thrombosis	Equal on both sides	Healthy.
Healthy	Clotted	Chiefly on right side	Thickened mitral and tricuspid.
Heart fatty	Clot, fluid, and throm.	Equal on both sides	Thickened mitral and aortic.
Fluid in pericard.	Fluid	Do.	Do.
Healthy	Thrombosis	Only on right side	Thickened mitral and tricuspid.
Fluid in pericard.	Fluid	Equal on both sides	Thickened mitral.
Heart fatty	Thrombosis	Do.	Thickened mitral and tricuspid.
Healthy	Do.	Do.	Healthy.
Heart fatty	Fluid	Do.	Thickened mitral and aortic.
Do.	Do.	Chiefly on right side	Healthy.

The causation of sudden death, although a subject to which a good deal of interest attaches, has hardly received from pathologists the attention it deserves; and consequently, while our knowledge of morbid anatomy is extending at a rapid rate, this particular province has still much unexplained and mysterious about it. That this should be so is the result of several circumstances. In the first place, the inquiry into the causes and modes of death is at no time an easy task; and, in the second place, the pathologist, to whom the medical world naturally turns for assistance, is not in a position to advance or to acquire much information bearing on this question. In our hospitals, institutions to which pathology owes its very existence as a science, the study of sudden and unexpected death is out of the question; such deaths occur in them only exceptionally, and the main aim of hospital autopsies is therefore almost exclusively the study of pathological lesions as connected with and suggested by the history of the individual obtained during life.

The position of the private practitioner contrasts favorably with that of the morbid anatomist in respect of opportunities for acquiring a practical knowledge bearing on this subject, and did the general practitioner avail himself of even a limited number of these opportunities, we would not have to complain of deficiency of information on this topic. But, unfortunately, in almost every instance, these are allowed to slip away, and to the lips of the physician called to the corpse of one who has suddenly and unexpectedly expired, the stereotyped verdict—heart disease or apoplexy—instinctively rises,—a verdict which has been, in some form or other, employed from time immemorial by the judicious practitioner, glad of a broad cloak for his ignorance, and an escape from the awkward questioning of the friends. In early times, it is well known, most sudden deaths were set down to the effects of poison, while in later years, the city of refuge for those who have not the courage to say, "I do not know," has unfortunately been rendered nearly impregnable by the discovery of the fatty heart; so that it is not overstrained to say that, among the richer classes at least, it must seem to the uninitiated laity as if no one whose demise has been sudden and unlooked for could enter into his future state otherwise than in one of a very few ways.

All the blame, however, should hardly be thrown on the general practitioner, for indeed he would have his difficulties to contend with did he adopt any other course. To refuse a death certificate until an autopsy was allowed, in the face of the opposition of the relatives, and of the fear of some more unscrupulous rival, would be an act which few indeed would commend; and their scanty applause, even with the addition of an approving conscience, would be but small consolation for the empty pockets such conduct would entail.

It is hopeless, then, to expect enlightenment on this head from the general practitioner; and the sole other source of information consists in the judicial inquests on cases of sudden death, where the results of these are obtainable. It has therefore been the object, in putting together the above tables and the following remarks upon them, to compare the results furnished by such sources, with the notions, or, at least, the utterances of many who, holding the position of fathers and heads in the profession, are yet most active agents in perverting our registrar's returns to a monstrous extent. Indeed, when regard is had of the multitude of people who are returned, after sudden death, as having succumbed to heart disease and apoplexy, and of children who are rashly supposed to have been "overlain," not to mention other favourite but erroneous hypotheses, it must be confessed that very many columns of these returns are worse than worthless.

In relation to Table I, where the results of 580 cases of sudden death, as disclosed by post-mortem examination, are brought together, some few explanations are necessary. The table is compiled from a series of full records of complete post-mortem examinations in cases of sudden and suspicious death, made with the view of reporting thereon to the Crown authorities. Those cases embrace many deaths which, under usual circumstances, might not have come under the denomination of sudden and suspicious, but it has been deemed better to allow them for the present to retain their position, as they illustrate very well the general run of disease, and prove that many maladies, in themselves almost incapable of remaining unrecognised, under peculiar external circumstances, or possessing an unusually latent character, contribute their mite towards filling up the catalogue of sudden deaths. In glancing over the list, for instance, it cannot but excite surprise to see such maladies as enteric fever, scarlatina, erysipelas, starvation, &c., figuring among the causes of sudden death, and it would require an account of the peculiarities of each particular case, an undertaking which space forbids us entering upon, to vindicate for such diseases the position they are made to occupy. But the fact remains, that such diseases do, under exceptional circumstances, destroy life in such a manner as to deserve being classed among the others in the above table, hydrophobia being, perhaps, the only one which ought properly to have been omitted from it.

Besides these more striking diseases, there are a number of others which could not have been expected to occupy so prominent a place as they hold; diseases which, though generally running their course under the observation of the physician, do sometimes progress in so latent and insidious a manner that their existence remains unknown and unsuspected by the individual himself, and the autopsy gives the first hint of their having been present. Among these may be

mentioned pneumonia, cellulitis, meningitis, encephalitis, cholera, liver disease, hydrocephalus, peritonitis, pericarditis, and bronchitis.

To complete the table, deaths from wounds and injuries, drowning, poisoning, hanging, strangulation, burns, &c., have been retained, although, strictly speaking, they do not belong to cases of death occurring suddenly under ordinary circumstances.

In Table II, an attempt has been made to reduce the first table of cases more to the level of ordinary experience of sudden death, and to place together merely such instances as would, by most inquirers into the subject, be comprehended under this designation. At the same time it must be remembered that such a thing as an absolute definition of what constitutes an ordinary sudden death is, in the very nature of things, out of the question, and that, in proportion as the subject is regarded from the practical rather than from the theoretical side, the difficulties of drawing a line of demarcation multiply themselves. Hence cases of suffocation have been retained, since this accident, although rare and tolerably obvious to an eyewitness where occuring in adult life, forms a large proportion of the diseases of infancy where the termination of life is sudden and inexplicable. In truth, although the signs during the autopsy are perfectly diagnostic of this occurrence in many of the deaths of young children and even of adults, little or no idea can be formed from the circumstances or the accounts of the friends, as to the mode in which it had originated.

In the second table, those cases where the symptoms during life, or the marks and appearances on the body after death, or the history of the case, where obtainable, would, with approximate certainty, indicate the agency at work, have all been omitted. Cases of drowning, where the wet clothes, &c., would tell their tale; cases of wounds and injuries, cellulitis, poisoning, hanging and strangulation—where the mark of the cord would remain,—hydrophobia, burns, convulsions, childbed diseases, erysipelas, scarlatina, abortion, and enteric fever, have all been omitted; and if any still retained may seem liable to challenge, it may be urged that numbers of those omitted should by right have been retained, while it should further be borne in mind that the cases must be estimated as they occurred in practice, and not as our notions of probability would suggest to us.

In the second table, purged and curtailed as it is, we find, still playing a very important part, many diseases which could hardly have been supposed likely to be represented in it, and these are all the more striking, as they preponderate over, or at least rival in importance, other maladies recognised as causes of sudden death. In fact, the two generally recognised causes, heart-disease and apoplexy, sink into the shade when compared with the numbers of pneumonias, brain diseases, &c. Cases of apoplexy stand as 43, or

13 per cent., while pneumonia numbers 49, or 15 per cent. Three cases of apoplexy, too, occurred in new-born children, reducing apoplexy in more advanced years to 40, or 12·270 per cent.; none of the cases of pneumonia occurring in children so young as this. Fatty heart numbers only 9 cases, or 2 per cent., and mechanical morbus cordis only 7 cases, the two forms of heart disease making together 16 cases, or 4·907 per cent., and reaching, if rupture of the heart is included, 20 cases in all, or 6·134 per cent.

It will be observed that some of the headings embrace diseases as yet hardly recognised by the profession as existing in the form of independent maladies, and which are certainly seldom present to the mind of the physician when reflecting on the mechanism of death,—diseased states, for example, like pulmonary apoplexy, thrombosis, and œdema of the lungs, and which, nevertheless, indicate their frequent occurrence by the magnitude of the figures appended to them, being respectively 23, 14, and 10 in number; but as some of these will be referred to at greater length subsequently, a minute inquiry into their conditions of existence is needless here.

These results show how little in a given case the practitioner can guess what has been the agency at work, and although the cases on which they are based are derived, for the most part, from the poorer classes, the conclusions apply to a very great extent to all ranks of society. While among the wealthy, probably, some few of the latent diseases might have been discovered during life, those which remain are cases of maladies common to all grades and conditions of men, and which I have found in private post-mortem inspections in sudden death among the middle classes, to be very frequent indeed. In fact, the autopsies of private patients of my own and other medical men lead me to believe that in them the causes of sudden death are very much the same as among the lower classes.

Having observed how commonly pneumonia is present in the bodies of those who have died suddenly, and having been led to investigate as closely as possible all such cases which occurred, I was brought face to face with a series of pathological appearances within the chest, which I had frequently before observed, but of which, till then, I had failed to appreciate the importance, viz. the frequent occurrence, either separately or conjointly with each other and pneumonia, of such conditions as œdema of the lungs, thrombosis within the heart and pulmonary vessels, and pulmonary apoplexy, and the relations they bear to other diseases of the body.

On careful observation of even a few such cases, it becomes plain that some or all of these diseased conditions or their combinations, constitute, in certain diseased or debilitated states of the body, the regular mechanism of death, and the main difficulty lies in assigning to each its true position and importance in this respect. On purpose to facilitate, as far as possible, a searching study of the conditions

where each is present, it has been thought better to notice these diseases separately, and to draw up for each a brief table of the cases in which it was found.

Pneumonia.

To commence with pneumonia, which has, since the time of Laennec, been known as a frequent pathological occurrence, in the form of what he termed "peri-pneumonie des agonisans," pneumonia of the dying, and also in the form of true inflammatory pneumonia.

In regard to the different forms of pneumonia to be found in the dead body, and especially in regard to the causes and characters of such, great differences of opinion prevail. Most authorities agree in saying that to the naked or assisted eye of the pathologist the different forms of pneumonia present themselves with characters so identical, that from the appearance of the lung alone it cannot be determined what form of pneumonia was present, and even those who pretend to draw a distinction do it in such a loose and indefinite way, that their definitions may be thrown aside, and it may be assumed that the appearance of the lung is no guide to the nature of the process of inflammation going on in it. Every one familiar with autopsies will agree in this, that the different stages of congestion, red and grey hepatisation, being capable of being assumed by pneumonias of different forms, as from inflammation, from obstruction, or from hypostasis, the altered appearances of the lung tissue are identical in each. And yet the appearances indicate that a difference of cause must have been present, for in one case a single lobe of a lung is found, in what may be called inflammatory pneumonia, with its tissue solidified from exudation of lymph, and vascular and congested in addition, while the pleura or pericardium in its vicinity bears traces of recent and acute inflammation; in a second case a lobe is also found solidified from lymph (with perhaps less evidence of vascularity), and converted into a solid mass up to the very pleura covering it, and the pleura in this case will not so much as be reddened, although its cavity may contain a few drops of serum; while in a third case, a case of hypostatic pneumonia, the consolidation, not limiting itself to a lobe, but occupying more or less the back parts of both lungs, could with difficulty have its locality accurately defined, and shades off gradually and without distinct demarcation into the neighbouring healthy lung. It seems, in other words, as if the pneumonia of the second class were simply a concentrated form of the third class, or hypostatic pneumonia, and as if the inflammatory were merely this pneumonia of the second class, plus the peculiar inflammatory virus, which we do not know save by its effects on the pleura and pericardium, but which makes diseases elsewhere so different, which causes, for example, that the

serum in a case of acute peritonitis will give rise to a virulent poisoned wound when accidentally inoculated, while the fluid in ascites is utterly harmless.

The term "pneumonia from obstruction" has been applied to some forms of lung inflammation by a recent writer on pneumonia, Dr. Octavius Sturges, who in an admirable paper in 'St. George's Hospital Reports' for 1867, has discussed this subject fully and with great ability.

Dr. Sturges divides pneumonia into four classes.

1st. Pneumonia in debilitating diseases.

2nd. Pneumonia in specific fever, or where some secreting organ is interfered with, *i. e.* in blood poisoning.

3rd. Pneumonia owing to mechanical causes.

4th. Pneumonia from idiopathic lung inflammation.

In his paper on this subject, Dr. Sturges shows that we are, by mechanical means, in a position to imitate the state of hyperæmia and congestion preceding inflammation ('St. George's Hospital Reports,' 1867, p. 219). "Venous obstruction," he says, and he cites "Simon's Lectures" in support of his statements, "however produced will give rise to an exudation which will be serous, or albuminous, or spontaneously coagulable, according as the pressure is less or greater." Quoting also a paper by Dr. Robinson in the 26th volume of the 'Medico-Chirurgical Transactions,' Dr. Sturges shows satisfactorily that in the stage of fibrinous effusion which has been mechanically produced in the kidneys by obstructing the flow of blood through the renal vein, the difference between the appearances found and those present in inflammation "is more of degree than of kind," and applying the observations and arguments to the lungs, he proves (l. c. pp. 220—221) that a pneumonic infiltration from obstruction is a highly probable phenomenon.

But it seems to me that while quite justified in assuming that there is an obstructive, as distinguished from an inflammatory pneumonia, Dr. Sturges has not made out the same claim to separate the obstructive inflammations, under which head are included, not only those where obstructive heart disease is present, but also those where alterations of the blood, or loss of power of the heart exist, from hypostatic pneumonia; and further, that his fourth class of idiopathic pneumonia ought by rights to include his second class of inflammation occurring during specific fever and interrupted secretion, a class in which, he says, "the lung affection occurs with marked local symptoms, resembling idiopathic pneumonia, with which, indeed, it may be pathologically identical."

It would seem, on the contrary, more philosophical to class together all pneumonias where the true inflammatory element is present, and which would all be characterised by the sympathy of the system generally with the local disease, as expressed in the

accompanying fever, and usually by the local signs, and after death by the element of inflammatory infection of the neighbouring parts, pleura, pericardium, &c.; and this, while freely admitting that the same inflammatory pneumonia, occurring in extremely adynamic subjects, may completely alter its usual appearances and give rise to the typhous or adynamic form so well described by Trousseau ('Clin. Med.,' 1862, Tome I).

In regard to the other class of pneumonia, of which specific inflammation does not seem to form an element, it is only a forced distinction that can be drawn between the mechanical and hypostatic forms. There is nothing in the appearances or situation of the disease, or in its symptoms, which would justify their separation, unless perhaps that hypostatic pneumonia usually affects both lungs, and especially their lower and back parts; while mechanical pneumonia, on the contrary, is oftener seated in one or more lobes confined to these, and occupying their entire extent.

Dr. Sturges' views of the production of this form of pneumonia, while decidedly a great advance on what had previously been brought forward, have still something to be added to them; and it is this something which gives a similar meaning to the two last-mentioned classes of cases, and resolves pneumonia of this nature, not so much into a cause of death as into the position of a method or mode of death, as it appears really to be.

No doubt the weight of the experiments in regard to obstructed circulation producing fibrinous effusion, similar to that occurring in inflammation, is great, but it is not necessary that obstruction be called into account to explain the results. The chief effect of obstruction of circulation is to lessen the rapidity with which the blood flows through the vessels. Beyond effecting this, and a slight amount of increased pressure within the vessels, obstruction can have no effect, and it would seem more reconcileable with facts to attribute the phenomena produced to the diminished rapidity of circulation which ensues. This idea gains strength if it is borne in mind what is one of the chief phenomena of the inflammatory process as observable in the capillaries of a frog's foot. In it the lessened rapidity of circulation soon comes on, plays a very important part, and is quickly followed by exudation of liquor sanguinis, and other changes similar to those which supervene in the kidney, whose rapidity of circulation is lessened by obstruction applied to its vein. Diminished rate of circulation is known to produce œdema, and it seems no more than fair to attribute the appearances produced by venous obstruction to the diminished speed of the circulation which is its consequence. That this is true many facts indicate. Serous effusion is usually producible by venous obstruction, as seen in phlegmasia dolens, and is also producible by simply diminished rate of circulation, a fact so well known as to need no proof. It exists

also as the consequence of obstruction to the entrance of arterial blood, as in embolism of a part, where the diminished rate of circulation consequent on the plugging of the artery, produces the œdema characteristic of this occurrence. I have no doubt that in venous obstruction going on to the effusion of lymph, the retarded circulation is the most important factor, and that, although the pressure may also have its influence in producing the exudation, such exudation may occur without pressure and simply from retarded circulation, favoured in some cases by alterations in the composition of the blood. The doctrine of Virchow, that the fibrin of the exudation is the product of the tissues, not of the blood ('Cell. Pathologie,' pp. 154, 367; 'Spec. Path. und Therap.,' Bd. I, p. 75) may also be allowed some weight in the question of the production of these forms of pneumonia.

On these grounds we would attribute all non-inflammatory pneumonias to diminished circulation; and where the disease appears to have selected one particular lobe or locality, to the existence of something diminishing the circulation in that situation more than in the rest of the lungs.

Pneumonia of this sort occurs in individuals in whom an altered condition of the blood is usually distinctly present as the result of old disease of the kidneys, liver, or heart, or of habits of intoxication, bad nourishment, or of marasmus, old age, &c. In those individuals the lessening of the vital powers is coincident with a diminished rate of circulation and with altered properties of the blood, which reaching a certain stage produce the non-inflammatory pneumonia of the lungs, unattended with any symptoms, and, although the individual seems to die as the result of general debility, pneumonia reveals itself at the autopsy, vindicating its claim to be regarded rather as a mode than as a cause of death. Similarly does the disease originate in individuals the subjects of no enfeebling agency, but who, prostrated by fever, apoplexy, or some other severe malady, have the circulation gradually failing as their vital force becomes exhausted, until, in their last few days on this side of the grave, the circulation has diminished to such an extent as to produce pneumonic alterations in the lungs, alterations to be recognised, in all probability, for the first time on the post-mortem table. The occurrence of pneumonia in combination with other similarly produced modes of death, as œdema of the lungs and thrombosis, as will afterwards have to be explained, strengthens greatly this view of the subject, and I have little doubt that many of our diseases, occurring in connection with the tubercular diathesis, and yet showing no tubercular indications in the pathological appearances produced, as the central softening of the brain found in what is falsely termed acute hydrocephalus, are simply similar non-inflam-

matory tissue alterations occurring in states of retarded circulation, the locality of the disease being determined by some local cause.

The table which has been drawn up of cases of pneumonia embraces all those where pneumonia occurred in the 580 cases of sudden death, and contains, besides the 49 where pneumonia was returned as the cause of death, as being the most important post-mortem appearance, 18 others where it was present though subordinate. No attempt has been made to separate the forms of pneumonia further than concerns the stage in which they existed; and to avoid rendering the table too bulky for use, it has been thought necessary to omit mention of its connection with troubles of the kidneys and liver, and to confine the particulars noted to the more interesting combinations with pleurisy, pericarditis, fatty and other heart disease, fluidity and distribution of the blood, &c.

Pulmonary Apoplexy.

The cause of death, next in numerical importance to pneumonia, of which special mention has to be made, is pulmonary apoplexy.

Presenting itself, in its diffuse form, in frequent combination with pneumonia, thrombosis of the heart and œdema of the lungs, its importance is sufficiently demonstrated by the Table IV, where it is shown to have occurred, in the 580 cases, 23 times as the most marked appearance and therefore returned as the cause of death, and 13 times as a subordinate appearance; 36 times in all.

It is not proposed to discuss the subject of pulmonary apoplexy here; I would refer instead to a paper by my father on this subject, published in the 'Brit. and Foreign Med.-Chir. Review' for April, 1866, and to the table I have drawn up; but the question of how pulmonary apoplexy is produced is one on which a few words may with advantage be added.

The way in which pulmonary apoplexy presents itself in many cases is a frequent puzzle to the pathologist, and it—more perhaps than any morbid alteration—is to be found complicating cases where there already exists an evident and sufficient cause of death, or even it may be giving the appearance as if two causes of death were co-existing, and it is sometimes hard to determine which claims to be the most important cause. To take only one instance of this from the table, there are there noted four cases of fracture of the skull in which pulmonary apoplexy was present, and in each of these its extent varied; in one it existed merely as two apoplectic depôts in one lung; in another the whole of the lower lobe of the left lung was consolidated from this cause; in a third the back parts of both lungs were affected; while in the fourth the total extent of both lungs was the seat of the disease, there not existing a single portion of either lung, however small, which was not black, consolidated,

and nearly airless, from this infiltration of blood. This last case, occurring as it did in a man whose assailant was afterwards tried for culpable homicide, left the medical men engaged in the case in a very unenviable fix. The injuries in the head were confined to fractures of the basis cranii, to injuries of the brain, and to effusion of blood among the membranes of the brain, but not abundantly enough to have caused the slightest compression. The man was known to have been in perfect health the moment before receiving the injury. I saw him dead half an hour later, and yet here were two causes of death, each in itself sufficient, and none of which could have existed before the injury was received. The explanation which suggested itself to me was the following, and it may be applicable, to a certain extent, to more of the cases in the table than those where fracture of the skull caused death. In the case just cited the natural mechanism of death after the injury to the brain would be by coma, and hence collection of blood on the right side of the heart and in the lungs, causing congestion of these organs. Now, pulmonary apoplexy is merely an advanced state of congestion, where the blood is infiltrated into the substance of the lung tissue instead of being retained within the vessels. What was the cause which changed congestion into extravasation is not clear; it may have been excessive heart's action, or some local cause seated in the lungs; but even this imperfect explanation removes the difficulties of the case, by converting the pulmonary apoplexy from a primary to a secondary cause of death.

In the case in the table it occurred twenty-seven times in the male sex, and only nine times in females, or three fourths of the cases being among males, and only one fourth among females.

Thrombosis of the Heart and large Vessels,

Although constituting the cause of death in only fourteen cases, occurs in a much larger number than this, being present in all in 63 cases, or in 10·8 per cent. of the total number.

The phenomena of thrombosis, as studied and elucidated by Virchow and his followers, consist in the loss of fluidity of the blood or of some of its constituents within their proper channels or receptacles, and embrace the results (where any exist) of this coagulation. Thus, so long as the coagulation within the vessel gives rise to no obstruction to the circulation, symptoms of its existence may be entirely wanting, excepting in the case of a portion or the whole of the clot being washed away with the current of the blood and becoming fixed in the next portion of the vascular system whose diameter is too small to allow its passage. In these circumstances the phenomena of plugging of the vessel have been designated by the name of embolism, and the impacted embolus occasions frequently the formation on it of further depositions of coagulum.

The act of the washing away of a portion of the thrombus is commonly attended with rigors, and this symptom forms a good diagnostic mark in distinguishing embolism from absorption of septic fluid, which is seldom (if ever) attended with the production of rigors.

Where the original thrombus increases to such an extent as to offer a barrier to the circulation, or where the process presents itself as embolism, œdema of the part supplied by the vessels generally occurs.

In the heart and main vessels this process of thrombus formation constitutes an extremely common method of death, the thrombus being sometimes the main agent in the cessation of life, and sometimes only a subordinate appearance.

In chronic maladies attended with diminished rapidity of circulation, or with an altered composition of the blood, it is but natural to suppose, and the analogy existing in the process of spontaneous cure of aneurism confirms the supposition, that while the constituents of the blood, as already shown under pneumonia, occasionally deposit themselves in the tissues under the forms of serous and fibrinous exudation, such deposition should sometimes take place within the mass of the blood itself. And such deposition of fibrine in the mass of the blood I believe to be the only explanation which our knowledge admits of, of the production of thrombosis of the heart and large vessels in the process of slow and gradual death. The thrombi are usually found, when small, in some part of the organs of circulation where the blood current can be fairly supposed to be slowest, where there exists something like a side eddy in the stream of the circulation. Their deposition is more common and more copious on the venous than on the arterial side of the heart and vessels, and they seem to select, in preference to all other localities, the auricular appendices of the auricles for their first formation. In many cases the thrombus remains limited to the right, or to both auricular appendices, and is there observed as a soft pale-yellow gelatinous clot, composed of fibrine pervaded and soaked through with serum, and exhibiting, where cut into, a meshwork of pale-yellow, slender fibres, with a large quantity of yellowish straw-coloured fluid, mechanically held in their interspaces. The more central or oldest parts of the thrombus are paler and a little firmer than the more superficial or last formed parts, yet without anything like an attempt at arrangement into layers, such as is found in the brittle and more opaque lymph lining an aneurism. The thrombus is, in addition, usually firmly connected and interlaced with the unevennesses of the inner surface of the heart, so that a considerable amount of force is requisite to separate and isolate it. The next stage of the disease, where the circulatory disturbances favour the increase of the thrombus, seems to be the formation of other thrombi

along the course of the vessels connected with the heart, and in preference along the course of the superior vena cava and its branches, and it is possible to pull out from the interior of this vessel a long yellow clot, whose ramifications indicate that it has been formed in the superior cava, the innominate, jugular, and subclavian veins. The thrombus, however, seems seldom to fill the veins, and merely to exist as a continuous cord lying in their calibre, appearing, from its greater yellowness at one side, to have lain along one of the walls of the set of vessels. Within the head the sinuses are also occupied in part by such a line of yellow fibrinous thrombus. This thrombus appears to retain its position, and to avoid being washed down into the heart by the extent of its ramifications, the tenacity with which its several parts adhere together, and by the lessened force of the circulation to which it owes its deposition. *I do not recollect ever having met with an analogous formation in the inferior vena cava.* Along the curvature of the arch of the aorta, a similar small thread of thrombus is sometimes found, extending however neither into the arteries of the head and upper extremities nor into the descending aorta, to any distance. Exceptional forms of thrombi are occasionally met with, such as pale and little consistent clots, of the size of a bean, or thereby, which appear to have been floating free in the auricles, and which seem, from their complete and rolled aspect, not to have been anywhere attached. It may be doubted, however, whether their attachments have not been interfered with in opening the heart. Thrombi, such as above described, do not apparently become readily washed into the pulmonary artery, the clots found there being *usually* continuous with those in the heart, as a consequence of a continuance of the growth of the thrombus about to be described. In the more pronounced forms of the disease, the already existing thrombi, those particularly in the auricular appendices, and especially on the right side of the heart, suffer an increase of bulk from continued deposition of fibrine, and still firmly attached to the heart walls, they grow larger and larger until they, in many instances, occupy the whole of the heart's cavities, being firmly interlaced in the auriculo-ventricular valves, and extending into the pulmonary artery so far that, on drawing them out from this vessel, and floating the portion in water, they can be seen as a cast of the minutest ramifications of this artery. The extension of the thrombus into the pulmonary artery appears *for the most part* to take place by direct continuation of the cardiac thrombus into the vessel, and seldom by the development there of an independent thrombus, since in some cases the thrombus is found to pass from the ventricle only into the main stem of the artery, and ends suddenly in a blood clot; in other and more frequent cases the thrombus fills the main stem and a part of its two primary branches, while in a third series of cases, the casts of all, down to the finest ramifications, are yellow and

fibrinous. These thrombi in the heart and pulmonary artery do not distend the cavities in which they lie; even in the most advanced cases, they merely fill them. In a few of the autopsies the pulmonary veins were also filled with thrombi, which were continuous with that in the left auricle; but, as a rule, the development of thrombus on the arterial side of the lungs is very limited indeed, and confined almost entirely to the auricle and ventricle.

The extensive development of thrombosis is attended with a markedly œdematous condition of the lungs, a state usually present to a more or less marked extent in even slight cases, although sometimes it has been found wanting. In this œdematous state the lungs are bulky, pitting on pressure, rather heavy and solidified, and from their cut surfaces air and serum can with ease be expressed. In the more marked cases the serum is clear and yellowish, and the whole pours out in abundance, frothing like champagne; where less œdema is present, or where the lungs are congested, the serum is pinkish, tinged with blood.

Such a process as this naturally requires a little time, a few hours or so, for its production; and hence it is much more frequent in gradual deaths, however unexpected they may have been, or however sudden they may seem to the friends and neighbours, than in those where the very mode of death indicates that it must of necessity have been rapid. Out of 98 cases of drowning, it was met with but once, and in an individual drowned in this wise. He was a farm servant, and had been in bad health for some time. Walking near a pond one day he was taken ill, and falling into it was drowned before he could be rescued. In him the thrombosis may have been forming before he fell into the pond.

The table of cases of thrombosis shows that where it occurred the death was caused—

14	times by	Thrombosis itself.
9	,,	Injuries.
10	,,	Pneumonia.
1	,,	Pericarditis.
3	,,	Poisoning.
7	,,	Exposure to cold.
1	,,	Childbed disease.
1	,,	Liver disease.
2	,,	Starvation.
2	,,	Brain diseases.
2	,,	Meningitis.
1	,,	Aneurism.
2	,,	Apoplexy.
2	,,	Pulmonary apoplexy.
1	,,	Erysipelas.
And 1	,,	Scarlatina.

In all which diseases the death would probably have been gradual, while it was found—once in death from suffocation, once in hanging, once in drowning (as mentioned above), and once in fatty heart; and in this case the thrombosis itself was more probably the true cause of death, whilst in the case of suffocation the individual was insensible from drink at the time, and was choked in his own vomit. Thus, out of 63 cases, it occurred only once in death which must of necessity have been sudden.

An altered and impoverished state of the blood appears to be an almost universal and very necessary condition in the formation of thrombosis. In young children, where the infrequent occurrence of chronic diseases leads to infrequent occurrence of impoverished states of the blood, thrombosis as above described is a rare phenomenon indeed. I have seen it once in a child three months of age, and in a case occurring since these tables were drawn up, once in a new-born child, where it existed as a rounded, complete, very soft, little consistent, and apparently unattached clot in the cavity of the right auricle. In all the other cases where it occurred the individual was above the age of three months.

The analogy of thrombosis as above described with that occurring elsewhere is seen from the existence of an œdematous state of the lungs, which was noted as present in 23 out of the 63 cases, while 27 times the lungs were tolerably healthy.

Finally, viewed in the light of being merely a method of death, thrombosis of the heart and large vessels is, as would be expected, frequently complicated with diseased states of the lungs other than œdema. In the table it will be found to have been coincident—

With pneumonia in	. . . .	17 cases.
,, œdema of lungs in	. .	23 ,,
,, pulmonary apoplexy in	. .	6 ,,
,, tolerably healthy lungs in	.	27 ,,
,, fatty heart	. . . .	12 ,,

It would be interesting to know, in those who recover after such fibrinous thrombi have been deposited, what becomes of them, and whether they are always reabsorbed. It is possible, in some cases at least, that they might during recovery give rise to sudden death from being washed away and impacted in the pulmonary artery or elsewhere, producing in this manner the phenomena of embolism.

Œdema of the Lungs.

In Table VI (of œdema of the lungs) all cases where this is noted as having been present are included, with the exception of the cases of drowning in which it occurred, these having been omitted as irrelevant.

It will be perceived from this table that it usually exists along with some other important pathological appearance, but that some-

times it is itself the most important change observable after death, and it would be refusing credit to the evidence of our senses to deny that it occasionally exists in the dead body as the only morbid phenomenon, and is, therefore, in itself an idiopathic cause—or rather mode—of death. It is connected, like pneumonia and thrombosis, with debilitated states of the system, and in the remarks upon pneumonia its independent existence will be seen to be capable of being accounted for by diminished circulation existing in these debilitated individuals.

Reprinted from the British and Foreign Medico-Chirurgical Review, Oct., 1869.