were found, but at necropsy the upper lobe of the left lung presented several haemorrhages in its substance, the largest being the size of a walnut.

Combined Type

The third type is a combination of the foregoing conditions much exaggerated. The visceral pleura is torn, there is haemopneumothorax, and there are extensive haemorrhages into the lung substance.

On May 7th, 1934, a girl, aged 15, collided whilst cycling with an on-coming motor-car, and was thrown into the air. She sustained fractures of the right femur, right humerus, and right clavicle. The ribs and sternum were intact. No bruises could be found on the chest, nor was there any effusion of blood into the muscles of the thorax. The ribs, as one expects in a girl of this age, were very resilient. There were two and a half pints of blood in the right pleural cavity, and the right lung was collapsed. There was a laceration, extending deeply into the lung tissue, six inches in length, on the posterior surface of the right lower lobe, and another two inches in length on the internal surface. The right upper lobe presented a tear, three inches in length, on its anterior surface. In front of this laceration a piece of the upper lobe, two square inches in extent, was almost completely severed. Haemorrhages of various sizes, with disruption of the lung tissue, were present, and numerous ecchymoses mottled the lung fields. The left lung presented similar haemorrhages and ecchymoses, but there was no rupture of the pleura.

Smith³ mentions a case of a boy, aged 8 years, who, in 1896, was run over by a heavy cart. There was not the slightest trace of abrasion or bruising of the skin of the chest, nor behind the ribs and sternum, but the upper lobe of the right lung had been cut completely off from the root of the lung, and was floating freely in a pleura full of blood.

Similar cases are recorded by Le Conte,⁴ Wideroe,⁵ Kerr,⁶ MacTaggart,⁷ and Spiers.⁸

Discussion

Injuries of the type described above must occur frequently, but rupture of the lung is comparatively rare. A convincing hypothesis on the physics of traumatic rupture of the lung is difficult to propound, especially when the characteristics of the normal lung are considered. The recorded cases are almost all in young persons. With the exception of the case mentioned above, MacTaggart's case seems to be the oldest—a man, aged 35, fell from a scaffold thirty-five feet high; his lungs were ruptured, but no ribs were fractured. This suggests that resilience of the ribs may be an important factor. But however resilient the ribs it is unthinkable that direct violence without gross damage to them could rupture the tough, spongy, non-resistant lung unless some other factor or factors were present.

It is reasonable to suppose the other factors to be an expanded lung and a glottis closed at the moment of impact. Given these three conditions—resilient ribs, expanded lungs, and a closed glottis—the effect of sudden violence might be likened to the bursting of a blown-up paper bag between the hands. Both lungs are affected, but greater damage might be expected on the side receiving the greater force.

The absence of bruising in some cases of internal injury and in violence to other parts of the body is a constant source of surprise to the legal mind and to juries. The factors governing the production of bruises are not fully understood, and all that can be said is that violence, and extreme violence, is frequently unaccompanied by bruising.

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MORBIDITY IN A FAMILY TREE

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The study of human heredity is a relatively unexplored part of the young science of genetics, and for obvious reasons progress therein must be slow. Thus it is important to record even a single family tree, to afford material for later research. The present case is brought forward for this reason, and also in the hope that it may interest some practitioners in the investigation of the vast amount of similar material to which they have special access.

The bulk of the records were obtained from the matron of a large hospital, who had kept old family diaries, and who could speak personally of many of the members.

Squares indicate males. Circles indicate females. Blacked-in signs indicate cardiovascular cases. Shaded signs indicate tuberculosis. Signs containing cross indicate malignancy. Half shading indicates individuals still alive. Diamonds indicate siblings of indeterminate history, and the number within the diamond the number of such siblings.

Some individuals were known to the investigator, and in some instances the family physician supplied valuable details. The rest of the data were obtained from others of the family, independent confirmation being sought in all cases. The subject studied was the cause of death, although it is admittedly not an ideal one for such investigations, since it is so frequently uncertain, and post-mortem reports are not commonly available. It can, however, yield a certain amount of information, particularly in instances such as this, where many of the records give clear indication of the major morbid factors.

The tree is given graphically in the figure, and the international convention of nomenclature has been adopted. One generation has been arbitrarily named I, and the ancestor and descendant generations are called AI, DI, and so on. The individuals in each generation are numbered consecutively. Siblings of negative or unobtainable history are indicated by diamonds, the number within showing the number of children. The progeny have not been shown when all the offspring yield no

data of any kind. In-comers by marriage are only given when necessary. Cardiovascular cases are indicated by blocked-out signs, tubercle by cross-shading, and malignancy by a cross. All the members are Scottish, except where otherwise stated.

NOTES ON INDIVIDUAL CAUSES OF DEATH

Case	Cause of Death	Notes
AII.1 ...	Paralytic stroke	Over 80
AII.2 ...	Died in childbirth	4-para. Italian
AII.3 ...	Apoplectic seizure	Aged 71
AII.4 ...	Malignant disease (abdominal or pelvic)	Over 60. French
AI.1 ...	Series of paralytic strokes	Aged 68
AI.2 ...	Several paralytic strokes	Over 70
AI.4 ...	"Heart disease"	Aged 64
AI.6 ...	Adult tubercle	—
AI.7 ...	Paralytic stroke	Aged 70
AI.8 ...	Sudden death	Aged 65
AI.9 ...	—	Second wife of AI.8. Cousin of the first wife, AI.7
AI.11 and AI.12	—	Died at 82 and 86 respectively
AI.13 ...	Tumour, probably malignant	—
AI.15 ...	—	Twin; died a few days after birth
AI.16 ...	—	Enlargement of the liver
AI.17 ...	Typhus	Between 40 and 50
AI.18 ...	From uterine fibroid	18 years' invalidism; aged 50
AI.19 ...	Pneumonia	Aged 52
I.1 ...	Tubercle	—
I.2 and I.3	"Strokes"	Over 60
I.5... ..	—	V.D.H. following on acute rheumatism; alive, over 60
I.6... ..	—	Hemiplegia; alive, aged 75
I.7... ..	Cerebral haemorrhage	Just over 60
I.8... ..	Apoplexy	Aged 48
I.9... ..	V.D.H. and Bright's disease	Aged 59
I.11 ...	—	Alive and well; aged 77
I.12 ...	Tubercle	Husband of I.13, who also died of tubercle
I.14 ...	Tubercle and pneumonia	When adult
I.16 ...	Cardiac failure	Aged 75
I.17 ...	Cerebral haemorrhage	Aged 80
I.18 ...	Malignant disease	Over 60
I.19 ...	Apoplexy	Aged 73
I.20 ...	Sunstroke in India	Between 40 and 50
I.21 ...	—	Aged 16
I.22 ...	Following upon operation	Nature unrecorded
I.23, I.24, and I.25	? Heart failure	No details
I.27 ...	Tubercle	Aged 12
I.28 ...	Cardiac failure	About 70
I.29 ...	—	Aged 71
I.30 ...	Carcinoma of stomach	Aged 74
I.31 ...	Following prostatectomy	Aged 79
I.32 ...	—	Alive and well; aged 87
I.33 ...	Angina pectoris	Over 60
I.34 ...	Angina pectoris and carcinoma of oesophagus	—
DI.1 ...	Tubercle	When a young adult
DI.2 ...	—	V.D.H. following on acute rheumatism; alive, aged 49
DI.3 ...	—	D.A.H.; alive, aged 35
DI.6 ...	Diphtheria	Aged 12
DI.7 ...	—	D.A.H. and pulmonary congestion; alive, aged 55
DI.8 ...	—	Tuberculous glands in neck; alive, aged 37
DI.9 ...	—	Tuberculous glands in neck; meningitis in infancy; alive, aged 28

NOTES ON INDIVIDUAL CAUSES OF DEATH (continued)

Case	Cause of Death	Notes
DI.10 ...	Tubercle	Over 50
DII.1 ...	—	Acute rheumatism (heart?); alive, aged 31
DII.3 ...	—	Tuberculous glands in neck; alive, aged 38
DII.4 ...	Infantile tubercle	—
DII.6 ...	Died soon after birth	—
DII.7 ...	Pneumonia and tubercle	Aged 16
DII.8 ...	—	Tuberculous hip; alive, aged 34
DII.9 ...	Died in early infancy	—

Summary

Summarizing these results, the notes of 108 individuals are recorded, of whom twenty-one are known to have died from cardiovascular disease (this does not include the case of sunstroke), and five cardiovascular patients are alive; five had malignant tumours, one of whom also had angina pectoris; of thirteen tuberculous cases four patients are alive. Sixteen patients are known to have attained the age of over 70 years, and a further twelve to have passed 60.

In the absence of data regarding the members of whom nothing is recorded, an extended analysis of this tree is scarcely justified. At the same time some support seems to be afforded to Weitz's¹ conclusion that hypertension is a hereditary diathesis. He says:

"It appears to me beyond question that a dominant heredity accounts for the majority of the cases. For most of the others a dominant heredity is probable but not certain; there is a small residue of instances in whose production heredity has not been shown to play a part."

It must also be noted that many of the individuals reached an advanced age, and so the expected incidence of cardiovascular degeneration among them would be much increased. But at the same time, if a hereditary tendency to hypertension is indeed present, as would seem to be the case both in the main stock—that is, progeny of AII.3, and in the subfamily of AI.1 and AI.2—then it would be expected to manifest itself at the weakest point of the constitution when old age advanced nearer to dissolution.

The hereditary aspects of malignant disease and rheumatoid diathesis are now attracting much attention, while the relation of inheritance to tuberculosis has been extensively investigated. For a full discussion of these subjects, and also of the inheritance of cardiovascular defect, reference should be made to Baur, Fischer, and Lenz,² who deal with the whole topic in a wide and inclusive manner.

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K. O. Evensen, writing in the August issue of *Norsk Mag. f. Laegevid.*, reports a case of aortic rupture in a man aged 52 who lived two and a half days after the rupture had taken place. The clinical symptoms were haemopericardium and ascites. The blood pressure was high, the systolic being 170 and the diastolic 80. The urea in the blood serum was 155 mg. per c.cm. Electrocardiographic examination showed negative T waves in the first two leads. Post mortem, a large heart, weighing 1,000 grams, was found. The pericardial sac contained 800 c.cm. of blood. A nearly complete rupture was discovered 2 cm. above the aortic valve. Macroscopically no morbid changes could be seen on the aortic wall, but, microscopically, slight inflammation changes in the media and some arteriosclerotic changes were detected.