ABSTRACTS OF CARDIOLOGY

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A Study of the Beneficial Effects of Anticoagulant Therapy in Congestive Heart Failure. G. C. GRIFFITH, R. STRAGNELL, D. C. LEVINSON, F. J. MOORE, and A. G. WARE. *Ann. intern. Med.*, 37, 867–887, Nov., 1952.

Anticoagulants have been given to patients with congestive heart failure at the Los Angeles County Hospital since emboli were found to be the direct cause of death in 20% of cases of rheumatic heart disease coming to necropsy. In the present paper the authors report the results of anticoagulant therapy in 416 out of 627 patients with congestive heart failure. Some patients received dicoumarol, others "tromexan" or "depo-heparin"; a number did not receive anticoagulant therapy and served as controls. Treatment was continued until heart failure had disappeared. An attempt was made to maintain the prothrombin level at 20% of normal, the level being estimated daily in patients given dicoumarol and twice daily in those given tromexan. Rapid fluctuation in the pro-thrombin level in patients given tromexan made it necessary also to give the drug in divided daily doses.

In the treated group thrombo-embolism occurred in 10% of cases as compared with 30% in the control group, the reduction being greatest in patients with rheumatic heart disease or coronary arterial disease without hypertension. There was no significant difference in the efficacy of the various anticoagulants used. Hæmorrhage occurred in 2·8% of the control and 2·9% of the treated patients; it was not observed, however, with dicoumarol or tromexan when the prothrombin level was above 10%. Protection from embolism seemed to be achieved when the prothrombin level was below 60% and the authors suggest that adequate prophylaxis can be secured with a prothrombin level of 45%.

It is concluded that administration of an anticoagulant is a useful adjuvant to the general treatment of congestive heart failure. *C. W. C. Bain*

Œsophageal Contraction and Cardiac Pain. W. EVANS. *Lancet*, 2, 1091–1097, Dec. 6, 1952.

If a diagnosis of angina pectoris is based on the patient's recital of his symptoms alone there is a good chance that the diagnosis is wrong. Pain in the chest closely resembling the pain of coronary disease may in fact not be associated with heart disease at all, but with a form of disordered action of the œsophagus which the author calls "œsophageal arrhythmia." This phenomenon is easily recognizable during cardioscopy while the patient swallows a thick barium paste. the normal rhythmical progression of the meal gives way to irregular movements, the meal is held up at the diaphragmatic opening, and the lower end of the œsophagus becomes distended; there is then a sharp recoil upwards of the meal, the œsophagus resuming its normal calibre for "a brief period" and then becoming distended again. This process may be repeated several times before the meal passes on into the stomach.

In order to determine the relation of this phenomenon to angina-like pain, the author has sought it in two groups of patients over a period of 15 years. The first group, the control series, consisted of 1400 subjects, 700 of whom were healthy persons without pain or evidence of heart disease, 200 were identified as having painless heart disease, and 500 were patients with a history of præcordial pain and electrocardiographic evidence of cardiac infarction. Of these 1400 patients 37 (2·6%) exhibited œsophageal arrhythmia, 25 being patients with coronary arterial disease and only 7 being healthy subjects. The second group, the test series, consisted of 332 patients complaining of pain closely resembling classic angina in nature and site, in being provoked by exertion, and in distribution to the chin and shoulder, but in whom scrupulous clinical and electrocardiographic examinations had failed to provide any evidence of heart disease. In 133 (40%) of these patients œsophageal arrhythmia was demonstrated.

The mechanism of œsophageal arrhythmia and the origin of the pain are discussed and the author points out that œsophageal spasm, except where it is segmental, is not a direct cause of pain. Œsophageal arrhythmia should not, therefore, be regarded as the cause of the chest pain; it is merely a radiological sign that, when present, suggests for the pain a dyspeptic source.

The treatment recommended is to give glyceryl trinitrate in an emergency of pain or distress, followed by a regimen consisting in reassurance and explanation, measures to promote better digestion, carminatives, reduction in consumption of alcohol and tobacco, and slimming. *G. F. Walker*

Results of Sympathectomy for Peripheral Vascular Disease, with a Fifteen-year Evaluation of Cases. J. W. HENDRICK and E. G. GUY. *J. int. Coll. Surg.*, 18, 668–684, Nov., 1952.

This paper records the personal views and experience of the authors, who have studied the value of sympathectomy performed by them in the treatment of peripheral vascular disease. A useful working classification is given: (1) vasospastic functional disease; (2) vasospastic

organic disease; (3) organic degenerative disease with or without vasospasm; and (4) hyperactivity of the vasomotor system. The commonly recognized peripheral vascular diseases are described and the value of sympathectomy in each is discussed. The place of sympathectomy in treating claudication is also described: the authors point out its value and the persistence of relief in many cases. They draw attention to the frequency with which claudication in foot muscles is misdiagnosed as foot strain.

It is their opinion that in certain cases—notably in the acute phase of thrombo-angiitis obliterans—sympathectomy seemed to hasten the progress of gangrene. [It seems more likely that the disease was so overwhelming that nothing could arrest its progress.] Sympathectomy in the treatment of causalgia, thrombophlebitis, frostbite, and acute arterial obstruction is also described.

[This paper is well worth reading in full. The authors' opinion that " the various diseases caused by [vascular] obstruction are frequently not well defined and seem to merge with one another " will appeal to all who have to manage these cases. *Cecil Flemming*

Dietary Treatment of Hypertension. I. H. PAGE and A. C. CORCORAN. *J. clin. Nutrit.*, **1**, 7–16, Sept.–Oct., 1952.

The authors, writing from Cleveland, Ohio, briefly review the history and experimental basis of treatment of hypertension by restriction of sodium in the diet. They point out that experimental work regarding the relation between vascular reactivity and sodium retention has been clouded by species differences. It is their opinion that in the clinical evaluation of dietary treatment of hypertension in human beings it is essential to have a preliminary control period, during which repeated blood-pressure determinations are made, with the aim of achieving a stabilized state while the patient is on a relatively constant, normal intake of sodium and protein. Some patients may reach this condition in a few weeks, whereas in others it may take some months. During part of this time the diet should contain 0·2 g. sodium with added salt in enteric-coated tablets. Sodium restriction can be started by replacing these by a placebo in tablets of identical appearance. Estimations of urinary sodium concentration are made throughout the study. Sodium intake should be increased to a normal level in a post-test control period.

It was found that under these conditions a minority of hypertensive patients respond to sodium restriction by fall in blood pressure and improvement in the pattern of hypertensive vascular disease. Similar studies with the rice diet of Kempner showed that its efficacy is related only to its low sodium content. It is not considered possible to forecast which cases will respond to sodium restriction, nor is it common to get a speedy answer to the question during the period when the patient is receiving the diet, even when, in addition to sodium restriction, mercurial diuretics are given, as the beneficial action of the diet may be long delayed. Many social, economic, and personality factors were found to militate against successful continuance of the diet; there is, moreover, in the presence of moderate or severe renal failure, the danger of sodium depletion.

The authors conclude that the practical choice lies between rice diet and other diets of low sodium content (less than 200 mg. per day). Extradietary sources of sodium (stomach powders, water-softeners) should be avoided; salt substitutes are unsatisfactory and, in the case of lithium chloride, dangerous. Cation-exchange resins are less satisfactory in the treatment of hypertension than in that of congestive heart failure or nephrotic syndrome. Low-sodium diet can with advantage be combined with hexamethonium therapy. In malignant hypertension speedier measures than sodium restriction are, as a rule, needed. *K. G. Lowe*

Nephrectomy and Other Treatment for Hypertension in Pyelonephritis. G. W. PICKERING and R. H. HEPTINSTALL. *Quart. J. Med.*, **22**, 1–22, Jan., 1953.

The results of excision of one diseased kidney for the relief of hypertension in 12 patients treated during the last 11 years at St. Mary's Hospital, London, are reported and the ætiology, pathology, and clinical features of chronic atrophic pyelonephritis—the lesion most commonly found in the excised kidney—are reviewed, the typical morbid changes being illustrated by one photograph and 5 photomicrographs.

Nephrectomy brought about considerable and persistent reduction of blood pressure in 4 of these 12 patients.

There are four possible explanations for the failure of nephrectomy to relieve hypertension in cases of apparently unilateral kidney disease; (1) hypertension of more than about 2 years' duration may have become irreversible; (2) it may have entered the malignant phase; (3) it may not have been the consequence of the renal lesion; or (4) undetected pyelonephritis may have been present in the other kidney. From this series and others reported the authors conclude that bilateral disease is the commonest reason for failure. Hence, if the condition is bilateral, excision of the more severely affected kidney is unlikely to do good, and may do harm. On the other hand if hypertensive neuroretinopathy (albuminuric retinitis) is present, urgent measures for the relief of the hypertension are required, such as: (1) subtotal adrenalectomy, which proved successful in the 3 cases reported here, but failed in 3 other cases in adults whose malignant hypertension was not the result of pyelonephritis; (2) sympathectomy, which was not adequately investigated in the present series; and (3) the parenteral administration of hexamethonium compounds.

Adrian V. Adams

The Natural History and Course of Hypertension with Papilledema (Malignant Hypertension). M. F. SCHOTTSTAEDT and M. SOKOLOW. *Amer. Heart J.*, **45**, 331–362, March, 1953.

The natural history of hypertension associated with papillœdema was studied in the records of 104 cases seen at the University of California Hospital between 1936 and 1940. The average age of the 63 men and 41

women was 42 years. There was a family history of hypertension in about one-half of the patients, and a history of significant illness (scarlet fever, frequent sore throat, glomerulonephritis, pyelonephritis, and toxœmia of pregnancy) in rather more than one-half. In 74 patients hypertension had been diagnosed 5 months to 27 years before the onset of the malignant phase. The authors estimated the average duration of the malignant phase to be 9 months.

Renal symptoms were observed in 87 patients, and chronic pyelonephritis was found in 12 of 37 cases in which necropsy was performed. The part played by the kidney in malignant hypertension was reflected in the response to treatment; only in those cases where renal function was good was sympathectomy or a low-sodium diet successful; in other cases the treatment of choice was administration of such drugs as hexamethonium and " dibenamine." The rapid deterioration in renal function noted in some cases was striking, indicating the need for frequent observation of patients with malignant hypertension so that treatment may be started promptly.

In the authors' cases the appearance of papillœdema indicated a phase of the disease which, if left untreated, was usually rapidly progressive, though in 3 cases recession occurred spontaneously. A raised cerebrospinal-fluid pressure was not considered to be responsible for the papillœdema, which was sometimes unilateral. Patients with papillœdema who had impaired renal function rarely recovered. The heart was affected in nearly every case in the series. *J. L. Lovibond*

Results with Oral Hexamethonium Alone and in Combination with 1-Hydrazinophthalazine (Apresoline) in the Therapy of Hypertension. J. H. MOYER, H. B. SNYDER, I. JOHNSON, L. C. MILLS, and S. I. MILLER. *Amer. J. med. Sci.*, **225**, 379–398, April, 1953.

In this paper are described the results in 58 cases of hypertension in out-patients treated with hexamethonium chloride by mouth. There were 28 men and 30 women aged between 29 and 71 years, in each of whom the pre-treatment blood pressure was higher than 160/100 mm. Hg. The initial dose of hexamethonium was 250 mg. given 4 times daily—a dose before each meal and another at bedtime. This was gradually increased until an adequate response was obtained or side-effects led to discontinuance of the drug. The lowering of the mean blood pressure (taken as the diastolic pressure plus one-third of the pulse pressure) by 20 mm. Hg was considered to be a good result. In only 18 patients was there failure to attain this without untoward side-effects, and in these cases " apresoline " (hydralazine) was given with rather less hexamethonium chloride. Apresoline was given in a dosage of 25 mg. 4 times a day, and this was increased by 25 mg. a day at weekly intervals. The combined therapy led to a satisfactory fall in blood pressure in 11 of the 18 patients.

Studies of renal hæmodynamics did not suggest that these drugs caused any permanent impairment of renal function. The relief of headaches, and of angina pectoris if present, was marked and the improvement of cardiac function striking. The drugs were given with benefit in cases of hypertension of renal origin, and the authors conclude that they may be safely used in such cases provided the blood urea level is not rising.

G. S. Crockett

The Prognosis of Subacute Bacterial Endocarditis Treated with Antibiotics. Results of Treatment in 202 Cases. E. DONZELOT, J. M. LE BOZEC, H. KAUFMANN, and J. E. ESCALLE. *Arch. Mal. Cœur*, **46**, 97–107, Feb., 1953.

The results of treatment during 1945–51 of 202 cases of subacute bacterial endocarditis are reviewed, and the prognosis in such cases discussed. Blood cultures (made in 201 of the cases) were positive in 134 cases, *Streptococcus viridians* being isolated in 67 cases, non-hæmolytic streptococcus in 43, *Str. faecalis* in 15, and various organisms in the remaining 9 cases. In 67 cases (33%) no positive blood culture could be obtained after at least 5 examinations. This proportion is considerably higher than that reported by other workers, but was due to the fact that the group was heavily weighted with serious cases referred to the authors from other centres. Of the whole group, 79% had rheumatic and 13% congenital heart disease. In 16 cases (8%) there was no evidence of previous heart disease, but the majority of these were men who had been deported to Germany during the war and in whom there was clinical or serological evidence of infection with typhus. In 14 of these the aortic valve was affected, and in 2 the mitral valve.

At first treatment was with penicillin (1,000,000 units per day), but later one of the newer antibiotics was given, the choice of drug and dose depending on the organism and on the clinical response. Of the 202 patients, 49 (24%) died within 3 months, and a further 21 (11%) within the next 7 years, giving an over-all mortality of 35%. Of the 21 delayed deaths 17 were due to heart failure; of the 132 survivors, the results were good in 120 and fair in 4; 8 patients did not reply to an inquiry.

Unfavourable features affecting the prognosis were: age under 10 (mortality 33%) or over 60 (mortality 40%); the abacteriæmic form of the disease; the presence of resistant bacteria, especially enterococci; affection of the aortic valve; the absence of previous heart disease; and the development of congestive failure. In the classic form of the disease, however, in patients between 10 and 60 years of age with compensated rheumatic disease and a positive blood culture, 83% were cured and more than 70% remained well from 4 to 7 years after the end of treatment. *Keith Ball*

Mechanism of Flutter and Fibrillation. D. SCHERF, A. I. SCHAFFER, and S. BLUMENFELD. *Arch. intern. Med.*, **91**, 333–352, March, 1953.

The authors discuss the two principal theories of the mechanism of flutter and fibrillation, the circus-movement theory and the ectopic-focus (tachysystole) theory, and review their findings in experiments carried out at New York Medical College following the discovery that these disorders can be induced in the heart by the topical application of aconitine (Scherf, *Proc. Soc. exp. Biol. (N.Y.)*, 1947, **64**, 233).

In experimental animals fibrillation or flutter will appear within a few seconds of the application of aconitine to any part of the auricles; if the area treated is isolated from the rest of the auricle by clamping, the arrhythmia ceases except in the isolated area, but returns in both auricles when the clamp is removed. An arrhythmia thus induced can be abolished by cooling the aconitinized focus, while by cautious cooling it may be possible to transform fibrillation progressively through flutter and extrasystoles to sinus rhythm; if the cooling thermode is removed, arrhythmia returns. Stretch and pressure applied to the aconitinized area increase the rate of existing flutter, and fibrillation ensues; the arrhythmias are immediately arrested by applying potassium chloride or quinidine to the aconitinized site. By faradization or the topical application of acetylcholine to the auricle similar " tachysystolic centres " can be induced not only at the site treated, but also at a distance—usually in the sinoauricular or auriculoventricular nodes; it is not clear why aconite does not induce such multiple centres. The authors suggest that these observations cannot be explained by the circus-movement theory, and provide strong evidence that auricular flutter and fibrillation originate in one or more ectopic foci.

The application of aconitine to any area of the ventricle of the dog induces a ventricular tachycardia indistinguishable from flutter which, as in the auricle, can be slowed or stopped by cooling the treated site. In certain cases, however, this flutter changes suddenly to fibrillation, whereupon cooling becomes ineffective. In explanation of this, the authors suggest that the tachysystolic centre set up by aconitine bombards the ventricle with rapid stimuli, causing the formation of innumerable tachysystolic centres in all parts of the ventricular myocardium, the simultaneous activity of which is seen as fibrillation. Thus ventricular fibrillation would appear to be invariably multifocal, whereas auricular fibrillation may be either multifocal or unifocal.

The characteristic feature of fibrillation, as opposed to flutter, seems to be repetitive action occurring in all parts of the myocardium in a totally irregular manner. This is apparently initiated in some way by stimuli from one or more self-sustaining tachysystolic centres, and is non-persistent in the absence of such stimuli in the auricle, but persistent in the ventricle. Thus fibrillation differs fundamentally from flutter, which appears to result from rapid stimulus-formation in a focus without the induction of repetitive action elsewhere in the myocardium.

R. S. Stevens

Paroxysmal Ventricular Tachycardia Treated with Intravenous Injections of Quinidine. L. E. JANUARY, H. E. HAMILTON, and D. W. SINTON. *Arch. intern. Med.*, **91**, 325–332, March, 1953.

The potentially serious nature of paroxysmal ventricular tachycardia calls for prompt measures; some patients cannot tolerate quinidine given by mouth, while it may be too long in taking effect in patients who are urgently ill. Hitherto the intravenous injection of quinidine has been regarded as dangerous, although its successful use in terminating attacks of ventricular tachycardia has been reported on a number of occasions.

At the State University of Iowa Hospitals during the past 12 years intravenous quinidine lactate or gluconate (65 to 650 mg.) has been used in treating 28 attacks in 11 patients. Six of these cases are described in detail. An immediate response occurred in 24 attacks, with dramatic clinical improvement in 18 of these, a delayed response occurred in 2 cases, and failure is reported in 2 cases, with the death of one already moribund patient.

The authors recommend dilution of the 10-ml. ampoules of quinidine solution (containing 65 mg.) with an equal volume of normal saline and stress the importance of injecting not more than 65 mg. of quinidine a minute. During the injection continuous auscultation and continuous direct-writing electrocardiography are carried out, any change in quality of the heart sounds, in the heart rate, or in the QRS complex being an indication for its termination. The most frequent side-effects noted were nausea and vomiting, with sweating in a few; one patient had a convulsive seizure during the injection but this was not repeated on subsequent occasions. Electrocardiograms illustrating the restoration of normal rhythm are reproduced. *R. S. Stevens*

Surgical Closure of Interauricular Septal Defects. R. E. GROSS. *J. Amer. med. Ass.*, **151**, 795–797, March 7, 1953.

Although a small interatrial septal defect may be tolerated fairly well, the presence of a large opening results in a severe left-to-right shunt and may lead to early heart failure and death unless the defect can be closed surgically.

The author describes a method of treatment that entails closure of the defect by means of a stitch or prosthesis through an opening in the right atrium. Blood loss is prevented by attaching to the edges of the incision in the atrial wall a rubber cone or " well " open at both ends and tall enough to allow the blood to rise to a height corresponding to the intra-atrial pressure without overflowing.

In more than 100 experimental operations in dogs it was found that blood rose only a few centimetres in the well and that the addition of small amounts of heparin prevented clotting. Moreover, the heart tolerated the opening satisfactorily for periods of more than an hour. Closure of interatrial openings was attempted with excised atrial muscle, plastic buttons and sheets, and simple sutures.

The operation has been carried out 7 times in children who had been shown by cardiac catheterization to have a left-to-right shunt of 6·7 to 22·3 litres a minute. A wide right thoracotomy is used and the pericardium opened. The large right atrium is exposed and, after incision, the " well " (a conical sleeve of rubber with a rigid upper rim) is attached by interrupted sutures, being held firm by a special clamp as blood rises into it. The hand is introduced into the well and the index finger is used to palpate the defect and to place and guide sutures and appliances.

In 3 cases the defect was closed with the Hufnagel button (two disks screwed together, one on either side

of the opening), but these worked loose because of the absence of an adequate rim of tissue to maintain them in position, the children dying within a few weeks of operation. In another 3 cases closure was carried out with nylon or polythene sheet, cut to size and sutured to the edges of the defect; there were 2 successes and one death, the latter caused by clot forming beneath the projecting edge of the closing sheet, which had been made too large. Finally, one child was treated by suture of the opening with silk stitches; this gave an excellent result, though a small shunt still persists.

T. Holmes Sellors

Six Cases of Single Ventricle with Pulmonary Stenosis. M. CAMPBELL, G. REYNOLDS, and J. R. TROUNCE. *Guy's Hosp. Rep.*, **102**, 99–139, 1953.

In this paper 6 cases of single ventricle with pulmonary stenosis are described in detail and 78 reported cases are reviewed. It is pointed out that a single ventricle with two auriculoventricular orifices, or a large single orifice, results from failure of the ventricular septum to develop normally. This abnormality is distinct from the apparently single ventricle that accompanies tricuspid or mitral atresia, where the septum is pushed far to the right or left to enclose a small chamber from which one or other of the great vessels arise; in these cases there is a single valvular orifice, not a true single ventricle in the anatomical sense.

On clinical examination there are no signs to distinguish cases of single ventricle with pulmonary stenosis from those of the tetralogy of Fallot, to which there is a functional similarity, and the electrocardiogram, angiocardiogram, and cardiac catheterization do not generally furnish decisive information. Bundle-branch block is rare, so that despite a gross abnormality of the ventricular septum, conduction tissue is still present; a strong right ventricular picture is the rule. The atrial septum is intact in about one-sixth of the cases, and transposition of the great vessels of varying degree, with some form of pulmonary obstruction, is seen in about half the cases. [Much valuable anatomical information is given in this paper, which should be read in its entirety.]

James W. Brown

The Diagnosis of Infarction of the Interventricular Septum. H. L. OSHER and L. WOLFF. *Amer. Heart J.*, **45**, 429–440, March, 1953.

In 35 fatal cases of cardiac infarction the electrocardiogram was analysed in the light of the extent and severity of the pathological lesions, as revealed by injection of the coronary arteries, gross examination of the heart, and histological examination of multiple sections.

The authors conclude that a diagnosis of infarction of the interventricular septum may be made when bundle-branch block and high-grade auriculo-ventricular block occur during the course of acute myocardial infarction, when complete right bundle-branch block is associated with Q waves in the right pærcordial leads, and when complete left bundle-branch block is associated with Q waves over the left ventricle. *J. F. Goodwin*

Changes in the Leucocyte Count in Myocardial Infarction. (Über das Verhalten der Leukocyten im peripheren Blutbild beim Myokardinfarkt). L. SLAPAK. *Cardiologia (Basel)*, **22**, 101–117, 1953.

The author has studied the leucocyte count in 228 cases of recent myocardial infarction seen at the Municipal Polyclinic, Vienna. Total counts were estimated daily for 6 weeks after the onset of the attack, and in 53 of the cases daily differential counts were made as well. A leucocytosis was seen in all the cases in which there was no other disease. This started on the 1st day, reached its maximum by the 8th day, and then fell rapidly. The degree of leucocytosis seemed to be correlated with the extent of the infarct; counts of over 30,000 per c.mm. were seen in two serious cases. In general, the smaller the leucocytosis, the better seemed the prognosis. Leucopenia was present in cases complicated by chronic rheumatoid arthritis.

The differential counts varied greatly. The appearance of immature leucocytes in the peripheral blood usually portended a grave prognosis. In most cases neutrophil granulocytes predominated in the first 2 weeks, and this was followed by a relative and absolute lymphocytosis during the 3rd week. [No explanation is offered for this.] *H. David Friedberg*

Clinical Study of Shock following Myocardial Infarction. T. R. FINK, C. J. D'ANGIO, and S. BILOON. *J. Amer. med. Ass.*, **151**, 1163–1165, April 4, 1953.

In the authors' view the shock-like state occurring in patients with acute myocardial infarction is due either to (1) severe heart failure with a raised venous pressure, or (2) a true state of shock like that which follows trauma, with marked arterial and venous hypotension. They therefore so classified 15 consecutive cases of myocardial infarction with shock and treated them accordingly—with intravenous lanatoside C in the former group and with intravenous phenylephrine (" neo-synephrine ") in the latter. Oxygen, rest, and sedation were used as indicated.

In the 10 patients with high venous pressure digitalis administration resulted in a rise in blood pressure and in some cases a fall in venous pressure. In the 5 patients with low venous pressure given phenylephrine, clinical improvement and a rise in both venous and arterial pressures ensued. It is considered that if the venous pressure be high, intravenous infusion of plasma, intra-arterial transfusion, or administration of phenylephrine is contraindicated. *H. David Friedberg*

The British Cardiac Society and the Editors of the *British Heart Journal* would like to congratulate their Printers, Messrs. William Clowes and Sons, on the recent celebration of their 150th anniversary of the founding of the firm. This has been commemorated in *Family Business*, 1803–1953, by the present William Clowes.