Transient Cerebral Ischaemic Attacks

A.C. YEUNG LAIWAH, MSc, MB, MRCP(UK)
Lecturer in Medicine, Glasgow Western Infirmary

The recognition that transient cerebral ischaemic attacks (TIAs) constitute an important risk factor in the subsequent development of a stroke has created a dilemma for clinicians. The controversy and confusion reigning over the management of the TIA patient[1-6] have, for the past 25 years, hindered the development of a uniform and rational approach to this condition. Much of the literature is based on uncontrolled and unsatisfactory clinical studies. This article highlights the numerous difficulties arising from an attempt to draw rational guidelines in the management of the TIA patient.

Definition of TIAs

The most widely accepted definition is that proposed by the *ad hoc* Committee for the Classification of Cerebrovascular Disease[7]; 'a TIA is a temporary and focal episode of neurological disturbance of presumed vascular origin which typically lasts for 2 to 15 minutes; it should not last longer than 24 hours or leave any neurological deficit.' However, the distinction between a prolonged TIA and a minor stroke is often a moot point, hence both conditions are frequently managed in a similar way.

TIAs are classified as carotid or vertebrobasilar, depending on the vascular territory of presumed ischaemia. Amaurosis fugax, a brief episode of partial or total visual loss in one eye, forms part of the carotid TIA definition. Differentiation between a carotid and a vertebrobasilar TIA can be difficult because the latter also causes visual disturbances such as blurring of vision, diplopia and blindness. In some instances, the motor or sensory features of vertebrobasilar insufficiency localise unilaterally, simulating a carotid TIA.

Isolated neurological symptoms like dizziness, vertigo, diplopia or dysarthria are too often wrongly attributed to vertebrobasilar TIAs. All these symptoms have causes that may not be readily identifiable and unless additional signs of brainstem involvement are present, a firm diagnosis of vertebrobasilar TIA is impossible. Furthermore, syncope is not to be confused with the 'drop attacks' of vertebrobasilar insufficiency unless the description of such attacks is absolutely clear.

The differential diagnosis of TIA often creates difficulties: the following conditions must be excluded—

1. the prodromal features of migraine
2. focal epilepsy
3. local eye diseases such as glaucoma or retinal detachment
4. intra-cranial neoplasm
5. giant-cell arteritis
6. hypoglycaemia
7. labyrinthine disorders

Natural History of TIA

In order to assess the potential benefit of any treatment, it is necessary to understand the natural history of the disease. The incidence of TIAs has been estimated to be between 0.3-1.3 per 1,000 adults/year in the Western population[8,9]. While it is agreed that TIAs constitute a risk factor for the subsequent development of a stroke, the magnitude of this risk is uncertain, as it depends, among other factors, on the working definition of a TIA. Many authorities accept that about one in three patients will develop a stroke within five years of the initial TIA. In fact, from published studies, the cerebral infarction rate at 3 to 5 year follow-up varied from < 2 per cent[10] to 53 per cent[11]. Of those patients who developed a stroke within five years, more than 20 per cent did so within the first month and 50 per cent within the first year of the initial attack. After the first year, the stroke incidence was about 5 per cent, equivalent to a five-fold increase in the expected rate for a control population with similar age distribution[12]. The time interval from the first TIA is therefore an important determinant in the subsequent occurrence of a stroke. Neither the average duration of each attack nor the frequency of such attacks had such predictive value[10,13]. Carotid TIAs have been given a worse prognosis than vertebrobasilar TIAs[10] but this view has been challenged[14].

Causes of TIAs

Reversible cerebrovascular insufficiency was originally thought to cause TIAs as a result of posturally determined changes in blood flow in patients with inadequate intracranial collateral circulation[15]. The importance of atheromatous disease of the large neck arteries became apparent in the early 1950s[16,17].The clinico-pathological study of Gunning *et al.*[18] provided strong support for the theory that TIAs could be due to recurrent microemboli arising from mural thrombi formed upon the atheromatous plaques of the carotid and vertebrobasilar arteries. Such artery-to-artery emboli are occasionally seen in the retinal arteries and may consist of platelets and fibrin[19] or atheromatous debris containing cholesterol crystals[20,21].

Although the contribution of other embolic sources

such as the heart was previously thought to be relatively small, this view is currently changing. Haemodynamic disturbances induced by cardiac arrhythmias[22] and postural hypotension[23] appear to be infrequent causes of TIAs in that focal areas of cerebral ischaemia might conceivably become manifest in a patient with co-existing unilateral carotid artery disease or with more diffuse intracranial atheroma.

Less common remediable factors include thrombocytosis, anaemia, polycythaemia, hyperviscosity syndromes and subclavian steal syndrome[4]. Although hypertension is a well-established risk factor in strokes, it is not clear whether hypertensive TIA patients are more likely to have a stroke than those who are normotensive. Hypertension should be treated irrespective of TIA.

Importance of Disease of Extracranial Arteries

As the concept of thromboembolism from the neck vessels became popular, attention was focused on disease of the carotid arteries, spurred by the first report of successful reconstructive surgery of the internal carotid artery in a 66-year-old woman with recurrent hemiplegic attacks[24]. In some earlier studies, significant extracranial vascular disease was reported in as many as 90 per cent of patients with TIAs or strokes as assessed by arteriography or at autopsy[25]. With improvements in carotid surgery, many enthusiasts, especially in the USA, have come to consider carotid angiography as an essential investigation in the work-up of the TIA patient.

Clinical Assessment

Assessment of the internal carotid pulse over the neck for obstruction is often inaccurate as pulsation in the external carotid artery may be mistaken for a diminished or even absent internal carotid pulse[26]. Palpation of the temporal or facial pulses has also been used to detect augmentation of external carotid flow in the presence of internal carotid obstruction[27], but few clinicians rely on this manoeuvre. A cervical carotid bruit is commonly defined as a localised bruit heard over the mid-cervical region just behind and below the angle of the mandible[28], yet bruits heard solely in the supraclavicular region can also originate from a low carotid artery stenosis[29]. The cervical carotid bruit has to be differentiated from a cervical venous hum, transmitted cardiac murmurs and, less commonly, from arteriovenous fistula, angiomatous malformations and stenosis of brachiocephalic, subclavian or vertebral arteries[30]. Bruits are unlikely to be heard if the degree of stenosis is less than 35 per cent and they may actually disappear if stenosis is in excess of 85 per cent. Furthermore, stenosis is often demonstrated angiographically in the absence of an audible bruit. The overall correlation between demonstrable carotid disease and carotid bruits has been estimated at about 60 per cent[28]. Despite these limitations, surprisingly perhaps, the presence of a carotid bruit is still widely regarded as an important clinical sign demanding further investigation of the patient[28,29].

Investigations

Numerous non-invasive tests have been devised in an attempt to select patients' suitability for carotid endarterectomy. These tests can assess luminal patency at the carotid bifurcation either directly, or indirectly by measuring differences in flow rate or pressure distal to the site of artery stenosis (Table 1).

The direct methods of measurement include:

1. Phonoangiography[33,34]—a quantitative analysis of the sound spectrum recorded from the carotid bruits. The measurement of lumen diameter correlates well with angiographic findings provided the bruit has the spectrum characteristics of turbulent blood flow.
2. Pulsed Döppler ultrasonic imaging[34-36], which registers reflected intra-luminal echoes that are related to flow velocity. The image can even be colour-coded to display relative flow velocity. When compared to angiography, the colour-coded method can achieve a sensitivity of 91 per cent and a specificity of 90 per cent when the stenosis is greater than 25 per cent[44].

The main indirect methods of assessment are:

1. Ophthalmodynamometry[40] detecting ophthalmic artery pressure difference. It often fails to detect severe carotid artery stenosis probably because of collateral blood flow. It has been superseded by oculopneumoplethysmography.
2. Oculopneumoplethysmography[42,43]. The difference in ophthalmic artery systolic pressure and ocular pulse volume between the two eyes can be used quantitatively to assess carotid artery stenosis. At best, the false positive rate is about 3 per cent and the false negative rate 6 per cent[45].

Table 1. Possible non-invasive tests for assessing carotid artery patency [31,32].

Direct Tests	*Indirect Tests*		
	Cerebral Circulation	Orbital Circulation	
		Superficial	Deep
1. Palpation	1. Radionuclide angiography	1. Thermography [37]	1. Ophthalmodynamometry [40]
2. Bruit auscultation	2. EEG with carotid compression	2. Directional u/s flow studies [38, 39]	2. Oculoplethysmography [34, 41]
3. Phonangiography [33, 34]		3. Supraorbital photoplethysmography	3. Oculopneumoplethysmography [42, 43]
4. Döppler B-mode Scan [35]		4. Supraorbital fluorescein testing	4. Oculotonography
5. Döppler Imaging ± colour coding [34, 36]			5. Oculosonography
6. Radionuclide angiography			6. Ocular pulse wave analysis

From the battery of tests available, the direct methods of measurement are more accurate than the indirect tests. But, as all these investigations have certain disadvantages, several studies have shown that a combination of direct and indirect tests can considerably improve the accuracy of assessment[31,39,41,42,46]. Nevertheless, 4-vessel angiography of the neck arteries remains the definitive investigation at the present time. The development of computerised video-substraction techniques[47] in obtaining improved angiograms after peripheral intravenous dye injection would provide a major advance in this area.

In spite of the high degree of accuracy achieved in assessing luminal patency of the carotid arteries, the treatment data available from published studies are confusing and difficult to interpret. This is because there is a singular lack of uniformity as to what should constitute a remediable surgical lesion. A degree of stenosis greater than 30 per cent is considered by some to be worthy of consideration; others have operated only on those lesions that are 'haemodynamically significant' (more than 60 per cent stenosis or a residual lumen of less than 2.5 mm[48,49]). The difficulty is further compounded in that severe carotid artery disease is often asymptomatic but symptoms can arise from minimal arterial lesions that hardly encroach upon the lumen and are undetectable by the tests available[50]. The possible importance of these minimal lesions has often been over-shadowed by the emphasis placed upon the increased risk of strokes in patients with asymptomatic carotid bruits[29] or demonstrable arterial stenosis[51]. Other workers have claimed that the prognosis of TIA patients without demonstrable vascular abnormalities was no different from those with stenosis or occlusion[52,53].

Limitations of Carotid Investigations

The main problem is not so much the detection of carotid stenosis as the investigations that should be used to indicate those patients who should have their carotid atherosclerosis treated. In this area, technological advances have surpassed clinical knowledge. The development of a technique that can detect small ulcerated plaques, irrespective of appreciable stenosis, would probably not resolve the issue because the arguments hitherto produced are based on the theory of thromboembolism from the neck arteries.

Several angiographic studies have shown that a significant number of TIA patients have normal neck arteries or minimal arterial stenosis[48,54-58]. In a recent prospective study of 117 patients with carotid TIAs, only 37 per cent of the patients who underwent angiography had significant stenosis whereas 25 per cent had normal arteries[58]. Although a small atheromatous plaque not appreciated radiologically could still act as a source of emboli, two other explanations should be considered. First, the platelets might be functionally abnormal although quantitatively adequate. So far, no consistent or specific platelet abnormality has been reported in TIA patients[59]. Second, there may be another important source of emboli that hitherto has been unsuspected. Barnett's group[60] have provided circumstantial evidence in support of this possibility. Echocardiography revealed mitral valve prolapse in 40 per cent of their younger patients with TIAs (mean age 23.9 years) as compared to 6.8 per cent in the control group. A causal role was attributed to this cardiac lesion after other known causes of TIA were excluded. None of these patients was previously known to have any mitral valve lesion. Furthermore, the prevalence of mitral valve prolapse in the older TIA patients (mean age 64.5 years) was similar to that of the control group, in keeping with the observation that it is unusual for a carotid lesion amenable to surgery to be discovered before the age of 50[57]. Arrhythmias[61] and thrombus formation[62,63] are recognised complications of mitral valve prolapse but their overall clinical significance remains to be established. Recurrent thromboembolism may be more important in causing TIAs, as arrhythmias cause features of generalised cerebral dysfunction rather than focal neurological deficit[22]. Cerebral infarction associated with mitral valve prolapse also occurs more frequently in younger patients[63]. Apart from mitral valve prolapse, other forms of cardiac disease predispose to cerebral embolism, especially in the elderly[64,65].

Treatment

Carotid Endarterectomy

Carotid endarterectomy as a prophylactic operation is only justifiable if the risks of future stroke or death are clearly reduced and the risk of the operation is slight. The effectiveness of surgery remains controversial, despite numerous claims to the contrary (Table 2). With the

Table 2. Results of carotid endarterectomy for TIAs.

Author	Year	Number of patients	Operative mortality (%)	Follow-up	Subsequent Strokes (1)*	(2)**
Fields *et al.* [66]	1970	169	3.6	mean 42 months	15	4
Wylie and Ehrenfeld [67]	1970	129	?	1-10 years	?	5.7
Thompson *et al.* [68]	1970	592	1.1	1-13 years	?	5.4
De Weese *et al.* [69]	1973	103	1	5 years	17	10.6
Toole *et al.* [70]	1975	82	6.1	mean 46 months	16	7
Nunn [71]	1975	170	1.2	mean 39 months	8.8	?

*Assessed from 'medical' review [1]
**Assessed from 'surgical' review [3]

exception of the American Joint Study reported by Fields *et al.*[66], controlled randomised studies are lacking. A review of the surgical literature from 1968-1977 analysing the results of 3,820 endarterectomies estimated an average peri-operative stroke rate of 5.7 per cent and a mortality rate of 3.4 per cent[72].

In some specialist centres, long experience coupled with technological advances have led to a great reduction in the risks associated with both angiography and endarterectomy. Angiography can now be performed with little morbidity (less than 2 per cent) and without mortality. Similarly, carotid endarterectomy is possible with a peri-operative stroke complication rate of less than 2 per cent and mortality of less than 1 per cent[3,72,73]. Careful selection of the surgeon or institution is critical because in less experienced hands a combined morbidity and mortality rate of 21 per cent has been reported[74].

In the 1970 report of Fields *et al.*[66], the risks of stroke and death were comparable in both medically and surgically treated groups by the end of $3\frac{1}{2}$ years. The recent improvement in surgical techniques has reduced the combined peri-operative stroke and mortality risk to 3 per cent or less against the currently assumed stroke rate of 15 per cent in the first year after the onset of symptoms and 5 per cent per year thereafter[12]; thus, it has been argued that therapy should now be surgical. Granted that the risks of endarterectomy are acceptable[75-77], patient selection remains a problem. For instance, Martin and his co-workers[50] observed significant neck artery stenosis in 40 per cent of 100 unselected autopsies of patients over 50 years, the majority of whom had no symptoms attributable to these lesions. On the other hand, postmortem studies of patients who had clinical evidence of cerebrovascular diseases also revealed a similar frequency of neck artery involvement[78,79]. It is therefore difficult to ascertain whether or not incidentally discovered atherosclerotic disease is clinically significant or if a demonstrated lesion in a symptomatic patient is the actual source of his symptoms. An exciting prospect for the future could be the development of radionuclide-labelled platelets or fibrinogen in identifying active thrombogenesis in the neck arteries.

Unsatisfactory patient selection probably accounts for the poor long-term survival after surgery, five-year mortality being about 30 per cent[80-82]. Although myocardial infarction is the main cause of death in these patients, recurrent, cerebrovascular disease is next in importance. Recurrence of neurological symptoms has been reported in up to 25 per cent of patients within five years of operation[81,83].

Anticoagulant Therapy

The value of anticoagulation is similarly controversial. Numerous studies have claimed that anticoagulation both reduces the recurrence rate of TIAs and prevents strokes (Table 3). Confusion prevails, however, because the data available are open to different interpretations, depending on whether importance should be attributed to favourable trends or only to statistically significant results, and whether only controlled, randomised studies should be considered[1,2,5,6,90]. To date, four randomised prospective anticoagulant trials in TIA patients have been published[54,85,86,88] and only one was double-blind[54]. None showed a statistically significant benefit in favour of such therapy. Unfortunately, all these studies suffered from having small numbers of patients or an inadequate follow-up period, and little consideration was given to other co-existing factors such as diabetes, hypertension and current drug therapy, which could possibly have affected the outcome of the trials.

Table 3. Anticoagulant therapy and TIAs.

Study	Year	Number of patients	Follow-up months	Cerebral Infarcts (Total)
Fisher [84]	1958			
control		23		8 (34%)
treated		29	30	1 (3%)
VA Co-op study [85]	1961			
control		15	12.8	0
treated		22	9.3	1 (4.5%)
Baker *et al.* [86]	1962			
control		20	20	4 (20%)
treated		24	18	1 (4%)
Siekert *et al.* [87]	1963			
control		160	60	51 (32%)
treated		175	60	7 (4%)
Pearce *et al.* [54]	1965			
control		20	10.6	2 (10%)
treated		17	11.1	1 (6%)
Baker *et al.* [88]	1966			
control		30	40.6	7 (23%)
treated		30	37.9	2 (7%)
Friedman *et al.* [9]	1969			
control		22	27.4	7 (32%)
treated		22	27.4	1 (5%)
Toole *et al.* [70]	1975			
control		56	46	7 (13%)
treated		21	46	6 (29%)
Olsson *et al.* [89]	1976			
no control				
treated		178	25	1 (0.6%)*

*Minor strokes separately grouped with recurrent TIAs.

In a non-randomised study, the Mayo Clinic group emphasised that anticoagulation reduced the risk of stroke for only two months after the initial TIA[91]. Long-term anticoagulation carries a risk of bleeding, and in one study 10 per cent of patients developed this complication[89]. Elderly patients, in particular, had an eight-fold increased risk of intracranial haemorrhage[92]. Nonetheless, the Mayo Clinic group recommended a three-month course of anticoagulation in patients seen within two months of the initial attack, the risks of bleeding being considered minimal in the short term[2]. In a recent prospective study involving 156 patients, Olsson *et al.*[93] concluded that anticoagulation not only had a prophylactic effect against cerebral infarction but was actually more effective than anti-platelet therapy; they would now use a longer initial course of anticoagulation, i.e. 3 to 12 months. Other experts have lately made similar definite

recommendations in favour of anticoagulants[1,94]. Despite such reassurance, clinicians will continue to view anticoagulant therapy with apprehension because of the possibilities of haemorrhage and the unsettled controversies surrounding this therapy[95].

Anti-platelet Therapy

The recognition of platelet-fibrin emboli in TIA patients encouraged the use of so-called 'anti-platelet drugs' even though the term 'anti-platelet' has no precise meaning[96]. One of the principal mechanisms believed to control platelet/endothelial interaction is the dynamic equilibrium between the pro-aggregatory prostaglandin thromboxane and the anti-aggregatory prostacyclin (PGI_2)[97]. Thromboxane is mainly produced in platelets and prostacyclin in the endothelial cells.

The three anti-platelet agents that have undergone the greatest clinical evaluation are aspirin, sulphinpyrazone and dipyridamole.

Aspirin inhibits platelet cyclo-oxygenase irreversibly by acetylation of the enzyme. Its effects last for the life-span of the platelets. Aspirin can also block endothelial cyclo-oxygenase, thereby impairing production of prostacyclin which inhibits platelet adherence to the endothelium[98]. The dose of aspirin required to inhibit platelet cyclo-oxygenase *in vitro* appears to be much lower than that needed to inhibit endothelial cyclo-oxygenase. It has therefore been suggested that low-dose aspirin may be more effective in preventing thrombosis than high dose aspirin[99]. This hypothesis has not been confirmed in recent studies of human subjects in whom little difference was noted between the inhibitory response of platelet cyclo-oxygenase and that of the endothelial cells to 150 and 300 mg doses of aspirin[100,101]. Perhaps the use of even smaller doses of aspirin might show this differential cyclo-oxygenase inhibition.

Sulphinpyrazone appears able to block cyclo-oxygenase in a reversible manner, and may also protect the endothelium against injury and prolong platelet survival in man.

Dipyridamole, introduced as a cardiac vasodilator, inhibits platelet function by raising platelet cyclic AMP concentration through inhibition of phosphodiesterase.

There have been two major controlled trials of anti-platelet agents in cerebrovascular disease. In the first, the American Co-operative study[102], 187 patients with carotid TIAs were given aspirin (625 mg b.d.) or placebo. At follow-up, carried out at an average of 24 months, there was a beneficial but not statistically significant trend in favour of aspirin with regard to cerebral infarction and deaths. When several clinical end-points grouped as 'favourable clinical outcome' were combined and compared to unfavourable end-points (consisting of multiple TIAs, stroke and death) a significant benefit in favour of aspirin was noted after six months of study.

In the second Canadian Co-operative Trial[103] 585 patients with carotid and vertebrobasilar TIAs were allocated to one of four drug regimes and followed for an average of 28 months. The drugs were: aspirin 1,300 mg/day, sulphinpyrazone 800 mg/day, aspirin and sulphinpyrazone combined, and placebo. Both regimes containing aspirin caused a significant reduction of recurrent stroke and deaths, but in men only. This sex-related benefit of aspirin had previously been noted and discussed in other studies[104,105]. Sulphinpyrazone did not confer any benefit on either sex. At present there is no evidence that dipyridamole is clinically effective when used alone[106].

The current widespread popularity of aspirin therapy for TIAs stems from the results of the well-designed Canadian study. Detailed statistical analysis of the results has purported to show a highly significant benefit for the use of aspirin in men, but at first glance these data appear much less impressive (Table 4). Other experts have expressed reservations about the conclusions of the Canadian study[107,108], yet the results of a recent prospective study in Sweden support the prophylactic value of anti-platelet therapy against cerebral infarction[93]. Comparison with the Canadian study is unfortunately not possible, as all the patients were initially treated with a two-month course of anticoagulation before randomisation into groups. The anti-platelet regime used consisted of a combination of aspirin and dipyridamole, the efficacy of which may be superior to that of either drug used alone[109].

Table 4. The Canadian Co-operative Study (1978). Summary of first events—stroke or death in men.

Treatment	Sulphin-pyrazone	Aspirin	Both	Neither
No. of subjects	115	98	102	91
Death without prior stroke				
vascular cause	6	3	4	5
non-vascular cause	3	0	2	2
Stroke (died later)				
minor	1	1	0	0
moderate	1	0	1	0
major	1	3	0	2
Stroke (alive at end of study)				
minor	10	3	3	4
moderate	7	5	1	6
major	5	2	1	3
Total with eligible events	34	17*	12	22

*Risk reduction for stroke or death = 48% ($p<0.005$)

Many physicians have already been guided by their 'gut-feeling' in using aspirin as the drug of first choice in the treatment of TIAs. The rationale for aspirin therapy has a firmer basis on current theoretical grounds than that for anticoagulation. Moreover, the risks with aspirin, especially when used in low dosage, seem more acceptable than with the other treatments. Yet the case for aspirin, on present objective evidence, is no stronger than that for the alternative therapies discussed, and should perhaps not be regarded as established until more information becomes available.

Conclusions

Within this therapeutic morass, it is clear that many clinicians have already chosen a particular mode of

treatment and neglected others. Unless the cause of TIAs can be identified for each individual patient, and well-designed trials carried out to determine the best treatment for defined sub-groups, any therapeutic guidelines that can at present be offered for the patient with TIAs will be strongly coloured by the therapist's bias. More concerted multidisciplinary efforts, such as that recently reported by de Bono and Warlow[58], are necessary to reassess the basic facts; otherwise, the results of even large multi-centric trials based on currently available information are likely to be disappointing, and treatment will remain more intuitive than scientific.

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A Caribbean Odyssey

Of the numerous medical men who are in some way commemorated in London Sir Hans Sloane has probably been accorded more recognition than any other, with thoroughfares like Sloane Avenue, Gardens, Street and Square, the statue in Chelsea Physic Garden, the blue plaque in Bloomsbury, the British Library and Museum, and the Natural History Museum in South Kensington. The last named was enriched with collections that in part originated from his voyage to Jamaica. This came about when the Duke of Albemarle was going out there to succeed the erstwhile buccaneer, Sir Henry Morgan, as governor. His physician, Peter Barwick, approached Hans Sloane to enquire whether he could suggest anyone who would accompany the Duke as his physician. After consideration Sloane offered himself for the position.

The journey began on 12th September 1687, and Madeira was reached on 21st October. 'When we came near to the Tropick [of Cancer],' wrote Sloane, 'we were called upon for our Tropick-money, that is to say, we who had never before crossed that line to the south, must now give either so much money as by the usage of seamen we shall be tax'd to make them drunk, or to be duck'd thrice into the sea from the yardarm, we chose rather the first and so were free.' They arrived at Barbados on 25th November, and after various other stops, Jamaica was reached on 19th December.

In the College library are two large handsome volumes 'bound in Turkey leather and guilt' and published in 1707 and 1725; they were a gift from the author, Sir Hans Sloane, who was by then President of the College and was presently to become President of the Royal Society—the only man to have held both offices. The two volumes contain a description of the voyage to Jamaica, including the account of crossing the line, and an elaborate account of animals and plants on the island. In addition there is an account of his treatment of Henry Morgan, and many other case histories together with information about the history and geography of the island, and the customs and food of its inhabitants. Besides a map, there are 156 plates, most of which were executed by Michael van der Gucht. Others are the work of J. Savage, a less known draughtsman. Some are unsigned and may well be the work of Garrett Moore, a clergyman of Irish origin, and one of the best draughtsmen on the island. Sloane had employed him to draw in crayons fruits, fish, birds and insects, being in the habit of taking him on his Jamaican outings so that he could draw specimens on the spot. In his travels Sloane collected 800 species of plants, and dried the best specimens he could find. As a practising physician Sloane was careful to include the medical virtues of plants he described. He had been told that the diseases of Jamaica were all different from those of

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