effort all these years, not only to attract an enormous number of readers, but have endeavored and are of the firm opinion that the papers they select for publication are preferably in the citation lists of the forthcoming relevant articles. The aforementioned criteria have enabled the journals to reach the heights of zenith and thereby achieve professional laurels. Many factors contribute to the number of citations per paper, including the science; the nature of the research; the style of communication (papers vary in the number of references they include, from none to over 100); editorial policy (several journals ration the number of references per article); circulation and readership (more readers equals more citations); citation bias comparable to submission bias and publication bias); conformism (scientists often cite those papers that are currently cited); authors' tendency to cite their own work; and referees' self promotion (referees' tendency to recommend inclusion of references to their own work).[2]

Among the sources available, MEDLINE has been accepted as an important information source to search journals and is rather the first available option to conduct a thorough literature review before embarking on writing an article. The author selects the pertinent and relevant articles pertaining to his or her work from MEDLINE and later endeavors to get the full texts. Apparently, an article with a title and an abstract available on MEDLINE would attract more authors than an article with no abstract. Obviously, a small abstract provides the reader with reasonably more ample information about an article than a simple one-sentence title. This appears to be an established fact and requires no further elaboration. Most articles published in reputed journals have simple or structured abstracts, and you encounter this reality while carrying out a search. Case reports, however, are exceptions, as some journals entertain them with an abstract, while others accept them without.

It appears that case reports with abstracts can be more easily approached, read and thereby cited than case reports without abstracts or the colloquial summary for that matter. While making a search, frequently you come across case reports apparently suiting your work, but since they are not abstracted, you nonchalantly forego searching the full texts and delete them from your citation list. Thereby many case reports with sterling impact remain untouched and never cited because they are not abstracted, and appear in the MEDLINE only in titled form. Certainly, if case reports had abstracts (although short and concise), it would increase their citations and thereby result in improvement of the impact factor of the pertinent journal.

Some authors have stepped beyond the aforementioned notion and are of the opinion that more informative abstracts improve the retrieval properties and are of value for citation purposes.[3] Although the editorial policy of most journals preoccupies itself with modalities, procedural issues, this is occasionally at the expense of fundamental issues, namely, the case reports, the letters and their apparent role on the impact factor.

Having considered the issue at length, it is suggested that case reports need to be abstracted, even, if presently the policy matter appears to be academically or financially inexpedient. This approach would have a significant impact on the quality of papers. This is not a vacuous slogan but can be justified by the veterans in this field.

Received 24th March 2002. Accepted for publication in final form 30th October 2002.

From the Departments of Anesthesia (Khan) and Neurosurgery (Saberi, Kashfi), Imam Khomeini Hospital, Tehran University of Medical Sciences, Tehran, Iran. Address correspondence and reprint requests to Dr. Zahid H. Khan, Associate Professor, Department of Anesthesia, Imam Khomeini Hospital, Tehran University of Medical Sciences, Keshavarz Boulevard, Tehran 14197, Iran. Tel. +98 (21) 935061 Ext. 3570. Fax. +98 (21) 929977. E-mail: arash_kashfi@hotmail.com

References

1. Garfield E. How can impact factors be improved? ***BMJ*** 1996; 313: 411-413.
2. West R. Impact factors need to be improved. ***BMJ*** 1996; 313: 1400.
3. Wilczynski NL, Walker CJ, McKibbon KA, Haynes RB. Preliminary assessment of the effect of more informative (structured) abstracts on citation retrieval from MEDLINE. ***Medinfo*** 1995; 8 (Pt 2): 1457-1461.

.....

Spironolactone responsive familial hypertension. *A potentially high prevalence of mineralocorticoid disease in Oman*

Nicholas J. Woodhouse, *MD, FRCP,*
Omiema T. Elshafie, *MD,* ***Ali Mehar,*** *MRCP,*
W. J. Johnston, *FRCP,* ***Juma M. Al-Kaabi,*** *MRCP, DTM&H.*

Mineralocorticoid (MC) hypertension may result from one of 4 inherited disorders, glucocorticoid remediable aldosteronism (GRA), otherwise referred to as familial hypertension type 1 (FH1), non-glucocorticoid suppressible disease, familial hypertension type 2 (FH2),[1] apparent mineralocorticoid excess (AME) and certain forms of congenital adrenal hyperplasia all of which are thought to be rare in the West.[2] Detailed questioning of patients attending our general endocrine clinics with established hypertension, or who developed hypertension during follow-up, revealed a large proportion (>90%) with a positive family history of the disease. Metabolic disorders are much more common in Arabia than in Europe, due primarily to consanguineous marriages (approximately

Table 1 - Biochemical and radiological findings in 14 patients with familial hypertension and their suggested diagnoses.

Plasma renin activity 0.2-2.8 ng/ml/hr	Aldosterone <440 pmol/L	Potassium 3.5-5.0 mmol/L	Steroid suppression	CT scan	Diagnosis
-	710	3.1	negative	Bulky L. adr	FH2
0.3	143	4.4	negative	N	Non MC FH
0.6	575	4.3	negative	N	HRFH
<0.2	3330	3.2	negative	N	FH2
0.5	558	3.6	negative	N	HRFH
3.8	632	4.5	negative	N	HRFH
2.7	702	-			
<0.2	1650	2.8	negative	L. adr nodule	Conn's FH
-	582	3.2	positive	Bulky L. adr	FH1
1.1	347	5.1	negative	N	Non MC FH
<0.2	499	2.9	negative	N	FH2
-	302	4.1	positive	N	FH1
<0.2	453	2.7	negative	N	FH2
2.7	316	4.3	negative	N	Non MC FH
2.0	580	4.3	negative	N	HRFH

CT - computerized tomography, L. adr - left adrenal nodule, N - normal, FH - familial hypertension, FH1 - familial hypertension type 1, FH2 - familial hypertension type 2, MC - mineralocorticoid, HRFH - hyperreninemia familial hypertension

30% in this country) and lack of population mobility.[3] We therefore postulated that their hypertension might be due to MC hypersecretion. To investigate this possibility we carried out a therapeutic trial using spironolactone to identify affected patients, as this drug selectively blocks the MC receptor. Our preliminary results suggest that in Arabia and probably worldwide, inherited forms of MC hypersecretion are surprisingly common.

Of 64 hypertensive patients, seen mainly over an 8 month period, having at least one or more affected parents and or siblings, 45 had completed a therapeutic trial of spironolactone at the time of writing. There were 29 females and 16 males with a mean age of 48 years ranging from 24 to 77 years. Twenty-nine were Omanis, 9 Sudanese, 2 Britons and one each from Egypt, Malaysia, Morocco, Philippines and South Africa (caucasian). The duration of their hypertension was from one month to 33 years and 28% were the product of consanguineous marriages. Before starting treatment, routine serum potassium, calcium and creatinine measurements were obtained together with a renal ultrasound. The only 6 patients found to be hypokalemic (K<3.5 mmol/L) were admitted together with 8 normokalemic individuals for aldosterone and renin measurements and had computerized tomography (CT) scans of the adrenals (patients 1-14). Bloods for renin and aldosterone were taken at 0800 hours after lying flat for 8 hours, and for aldosterone at 0600 hours after dexamethasone 2 mg daily for 4 days. These patients (1-14) and the remainder (15-45) were started on spironolactone 50-100 mg twice daily, given alone to newly diagnosed patients or in addition to other antihypertensives. If the blood pressure (BP) was controlled after 2-4 weeks (<140/85), spironolactone was then continued either alone or after the withdrawal of one antihypertensive agent. Again, after a further 1-2 weeks with good BP control, another medication was discontinued and so on until the patient was taking spironolactone only. A responder was defined as a patient whose BP remained at or below 145/85 mmHg after one or more month's treatment with spironolactone. The outpatient blood pressure readings were obtained using the same 2 nurses and sphygmomanometer throughout. Individual patients' BP was calculated by averaging the combined systolic and diastolic values obtained on at least 3 occasions before and during spironolactone. The group response was expressed as a mean percentage fall in BP with ranges.

Plasma renin activity (PRA) was measured in 11 of the 14 patients who were admitted and is shown together with the serum aldosterone and K levels and adrenal CT scan findings in **Table 1**. Plasma renin activity was suppressed in 4, unsuppressed or raised in 4, and normal in the 3 patients with normal aldosterone levels. Blood pressure control was achieved using spironolactone in 39 of the 45 patients so far studied. This includes the 11 patients, 10 with documented aldosterone hypersecretion and one with normal levels, who were investigated in the ward (data not shown) and 28 of the 31 patients given spironolactone alone, whose mean fall in BP was 19% from baseline (range 5-34). Serum aldosterone levels were completely suppressed in patients 8 and 11 only (data not shown) during dexamethasone administration.

Gynecomastia occurred in most of the male patients who responded to treatment; they were then restarted on their previous or new antihypertensives. Blood pressure was controlled satisfactorily in the 2 patients so far started on moduretic (amiloride 5 mg, hydrochlorothiazide 25 mg once or twice daily). Menstrual irregularities were reported in 4 patients.

These preliminary observations are quite provocative and if confirmed, indicate that inherited forms of MC hypertension are surprisingly common. In this therapeutic trial using spironolactone to block the MC receptor, 84% of the patients responded with a fall in BP. Many had previously been poorly controlled requiring 2 or more antihypertensives. We have interpreted this response to indicate MC hypersecretion, an idea which is supported by finding raised aldosterone levels in 4 of the 8 normokalemic patients randomly admitted for study. Furthermore, BP control was achieved only in one of the 4 with normal aldosterone: as the responder (patient 11) had undetectable levels during steroid suppression a diagnosis of FH1 seems likely. As might be expected, all the patients with a raised aldosterone responded to medical therapy.[2] Of the 14 patients so far admitted, 3 had non MC induced hypertension, and the remainder a presumptive diagnosis of FH2 (4 patients), FHI (2 patients), based on steroid suppression data, HRFH (4 patients) and one a Conn's tumor. The latter was removed during laparoscopic surgery and has resulted in normal aldosterone levels and BP control. The patient's father, mother and sister, now deceased, were all hypertensive as is his only brother suggesting the possibility of familial Conn's syndrome. We hope to obtain an adrenal CT scan from the brother shortly. Computerized tomography scans were normal in the 4 patients with HRFH, and as yet we have no explanation for their raised renin values.[4] A secondary increase resulting from hypertensive kidney damage seems unlikely as all had normal creatinine levels (50, 57, 58 and 41 μmol/L) and only mild to moderate hypertension.[5] Family screening is underway to document whether or not they have familial hyperreninemic MC excess. These findings indicate that the prevalence of MC induced disease may be much higher than previously suspected. In evolutionary terms, this makes sense as the ability to retain salt in extreme climates might be expected to have a definite survival advantage. We conclude that patients with familial disease should undergo a therapeutic trial of an MC receptor blocking drug, before embarking on expensive endocrine investigations. The advent of a new MC receptor blocking agent not affecting the estrogen receptor is awaited with interest.[2] The fact that we identified responders from 3 continents also argues that MC induced hypertension is worldwide and not just a Middle Eastern problem. The identification of increasing numbers of patients with primary hyperaldosteronism, in the United States, Europe, Australia and elsewhere and the realization that many of them are normokalemic at presentation[2] suggests to us that the so called "primary" or "idiopathic" disease may in fact be FH2 in disguise.

Received 2nd July 2002. Accepted for publication in final form 16th November 2002.

From the Department of Medicine, Sultan Qaboos University Hospital, Sultanate of Oman. Address correspondence and reprint requests to Prof. Nicholas J. Y. Woodhouse, Department of Medicine, Sultan Qaboos University Hospital, PO Box 35, Al Khod 123, Muscat, Sultanate of Oman. Tel. +968 513355. Fax. +968 513419.

References

1. Stewart PM. Dexamethasone-suppressible hypertension. ***Lancet*** 2000; 356: 697-699.
2. Stowasser M. How common is adrenal-based mineralocorticoid hypertension? ***Current Opinion in Endocrinology & Diabetes*** 2000; 7: 143-150.
3. Ozand PT. Department of Pediatrics, King Faisal Specialist Hospital, Riyadh, Saudi Arabia. Personal Communication January 2000.
4. Keberle E, Brenneis H, Haag M, Keberle M. Reninoma: a rare cause of hypertension. ***Urologe A*** 2000; 39: 352-355.
5. Oelkers, W, Diederich, S, Bahr, V. Primary hyperaldosteronism without suppressed renin due to secondary hypertensive kidney damage. ***J Clin Endocrinol Metab*** 2000; 85: 3266-3270.

.....

The penile support. *A new method for the treatment of impotence (erectile dysfunction)*

Ahmed N. Al-Maleky, *MBChB, DDV.*

The penile support (PS) is a newly designed instrument to treat the erectile failure, it is indicated for the treatment of all cases of psychological impotence caused or accompanied by weakness of erection in which the fear of failure (vicious-cycle effect) is preponderant and inaccessible to psychological treatment and it does not make sense to waste time with fruitless procedures and to put off a helpful form of treatment for so long that the patients detrimental auto-suggestion have becomes enhanced and fixed, thereby considerably impairing the prognosis. For approximately 2 years, 21 cases of psychological impotent males were treated by the PS, the success rate was more than 80%. According to the review articles obtained from the medline there was no previous similar method. I recommended the PS method for the treatment of psychological erectile dysfunction. Erectile failure is one of the most rewarding of all sexual problems to treat.[1] Impotence is the inability to obtain or maintain an erection of sufficient firmness to permit coitus to be initiated or completed, and can be classified as either primary or secondary. The male with primary impotence has never been able to have intercourse, whereas the male with secondary impotence is experiencing erectile dysfunction after a previous period of normal function.[2] Impairment of erection may result from a variety of organic and psychogenic disorders but in the majority of cases of erectile dysfunction the cause is usually multifactorial.[3] Psychological factors have a role to play in every case of erectile failure whatever the cause, such factors are not always deep in origin and can often be