RESEARCH LETTERS

A Practice Brochure: Complement to, Not Supplement for, Good Physician-Patient Interaction

Patient satisfaction affects patients' compliance with prescribed regimens and their clinical outcomes.[1] Based on the results of patient satisfaction surveys, we suspected that providing an informational brochure to patients regarding their physician's qualifications and desires to provide high-quality care would improve patient satisfaction.

Methods. We surveyed 50 new adult patients attending a dermatology clinic visit for various dermatologic conditions. The 25 patients in the intervention group received a short brochure containing information about their dermatologist's training, desire to provide high-quality health care, and contact information; 25 control patients did not receive the brochure. Both groups completed a postvisit survey of 6 questions related to patient demographics, including age, sex, ethnicity, education, payment source, and reason for their office visit. They also completed 11 items related to their experience, satisfaction, and comfort level during their visit, each item rated on a numerical scale on which 0 indicated "strongly disagree" and 10, "strongly agree." The survey was analyzed using *t* tests and corresponding means; *P* values were then calculated.

Results. The mean overall satisfaction of the control group, which did not receive an informational brochure, was 8.6 vs 8.4 for the intervention group. Most questions were answered more favorably by the control group, although none of the differences were statistically significant. For example, the control group agreed more strongly than the intervention group with the statement "I know who to contact if I have a question or concern regarding my treatment or appointment" ($P = .06$). The control group mean scores were also slightly higher for the statements "I am satisfied with the care my dermatologist provides"; "my dermatologist is concerned about the skin care I receive"; "I am comfortable speaking to my dermatologist about my questions and concerns"; "my questions about skin care were answered during my office visit"; and "I am confident about my treatment plan." No statistical difference was found demographically between the 2 groups except that slightly more Medicare patients were included in the intervention group.

Comment. A dermatologist's interpersonal skills are the most relevant factor in determining patient satisfaction.[2] To the extent that patients who received the brochure had higher expectations than those who did not, the dermatologist may have been more likely to disappoint them with the service provided. The small size of our sample population was a limiting factor in our study. In addition, we did not account for disease severity. It may be easier to please patients with more severe disease where even small improvements may significantly affect the quality of life.[2]

Patient satisfaction is an integral aspect of providing optimal patient care: high patient satisfaction helps lead to improved health outcomes. Patients generally view their dermatologist as the primary source for information about their skin, and they desire a genuine concern from their physician as well as answers to their questions. A supplemental brochure provided to new patients at the check-in counter did not improve patient satisfaction to a statistically significant degree and is not a substitute for quality time with the physician.

Kristen Fosse, MD
Eleanor Kurtz, MD
Vishal Khanna, MFA
Fabian T. Camacho, MS
Rajesh Balkrishnan, PhD
Steven R. Feldman, MD, PhD

Correspondence: Dr Feldman, Department of Dermatology, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, NC 27157-1071 (sfeldman@wfubmc.edu).
Financial Disclosure: Dr Feldman owns stock in www.DrScore.com, an online patient satisfaction survey service.
Funding/Support: The Center for Dermatology Research is supported by an educational grant from Galderma Laboratories LP, Fort Worth, Texas.

1. Fan VS, Burman M, McDonell MB, Fihn SD. Continuity of care and other determinants of patient satisfaction with primary care. *J Gen Intern Med*. 2005; 20(3):226-233.
2. Renzi C, Abeni D, Picardi A, et al. Factors associated with patient satisfaction with care among dermatological outpatients. *Br J Dermatol*. 2001;145(4): 617-623.

A Randomized Double-Blind Study of the Effect of Botox and Dysport/Reloxin on Forehead Wrinkles and Electromyographic Activity

The difference between the potency units of the 2 main botulinum toxin A products, Botox (Allergan, Irvine, California) and Dysport/Reloxin (Ipsen Ltd, Slough, England), is still a subject of discussion even after 15 years of clinical use. The manufacturer of Botox recommends higher ratios than does the manufacturer of Dysport/Reloxin. Herein, we report the findings of a randomized, double-blind, split-face study of forehead wrinkles and electromyographic (EMG) activity following application of the 2 products at a 3:1 dose ratio, independent of the support of either manufacturer.

Methods. Patients included in the study were aged 30 to 70 years, had moderate to severe hyperfunctional forehead wrinkles at rest and maximum contraction, and gave written informed consent. The study was approved by the institution's human research review committee.

Twenty-six patients were enrolled, 2 of whom were lost to follow-up and thus excluded from the study. Of the 24 remaining patients, 20 were women (83%), with a mean±SEM age of 41.9±1.4 years.

Patients were randomly assigned to receive the 2 different formulations in each side of the frontalis muscle in a 3-injection site pattern: 12 U of Botox and 36 U of Dysport/Reloxin were diluted in 0.3 mL of isotonic sodium chloride solution, and the syringes were prepared according to a computerized randomization by an assistant, who passed the unmarked syringes to the treating physician. The EMG electrode was placed 3 cm above the upper medial eyebrow, and the baseline frontalis muscle voltage was measured. All measurements of electric muscle activity were performed during maximum voluntary contraction of the frontalis muscle using the MyoSystem 1200 and the MyoResearch XP software package for analysis (Noraxon, Scottsdale, Arizona). The injections were placed 0, 1, and 2 cm lateral to the position of the electrode.

Study end points were assessed at baseline and 0.5, 1.0, 2.0, 4.0, 8.0, and 10.0 weeks after injection, followed by weekly examinations for a total observation period of 20 weeks. Photographs taken during maximum contraction at each follow-up visit were presented randomly to a panel of 3 experts who were blind to the treatment and duration of time since the treatment. Wrinkle severity was assessed by the panel using the photographs and directly at each visit by the investigator and the patient using a simple 3-item rating scale: "more wrinkles on the right side," "no difference between sides," and "more wrinkles on the left side."

After completion of the study, the blind was broken and all scores recorded according to the side of treatment (0, more wrinkles on the Botox side; 1, no difference; and 2, more wrinkles on the Dysport/Reloxin side).

For statistical analysis, a paired *t* test was used (GraphPad Prism 3.0 and GraphPad Instat 3.05; GraphPad Software, San Diego, California, and SPSS 13.0; SPSS, Chicago, Illinois).

Results. Muscle EMG activity was depressed by both products, reaching a minimum 2 weeks after injection. The effect of Dysport/Reloxin was longer lasting, and the difference from the effect caused by Botox was statistically significant beginning 10 weeks after injection and lasting until the end of observation ($P<.001$) (**Figure 1**). Wrinkle severity, as assessed by the panel ($P<.001$ at week 10), investigator ($P<.001$ at week 10), and patients ($P=.03$ at week 10), increased accordingly in the Botox-treated sides.

Comment. After the difference in unit potency between Botox and Dysport/Reloxin was recognized, a conversion factor of 4:1 to 5:1 was hypothesized.[1] This conversion factor remains widely used despite evidence from the manufacturers' assay methods[2] and comparative clinical trials carried out according to Cochrane standards of evidence-based medicine[3] showing that 3:1 is a more appropriate conversion ratio than 4:1. More recent data suggest that the best dose conversion ratio may in fact be less than 3:1.[4-6] The recommended ratio for the treatment of glabella lines in Germany is 2.5:1.0 (50 U of Dysport/Reloxin to 20 U of Botox).

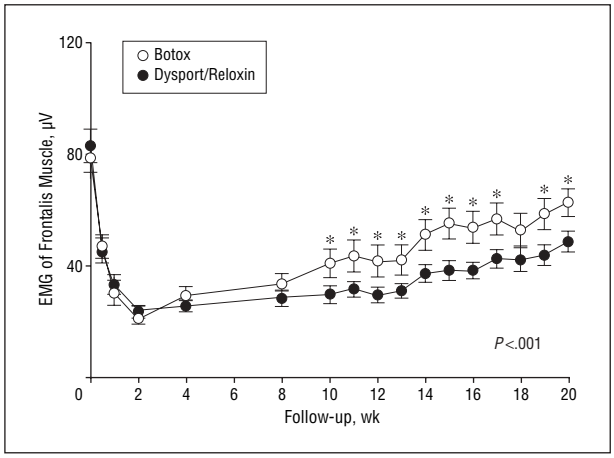

Figure 1. Electromyography (EMG) results from the frontalis muscle prior to injection and during the 20-week follow-up period. Displayed are the mean±SEM microvoltage readings in each group. Botox and Dysport/Reloxin are proprietary names for botulinum A toxin products manufactured by Allergan (Irvine, California) and Ipsen Ltd (Slough, England), respectively. *Statistically significant difference between groups.

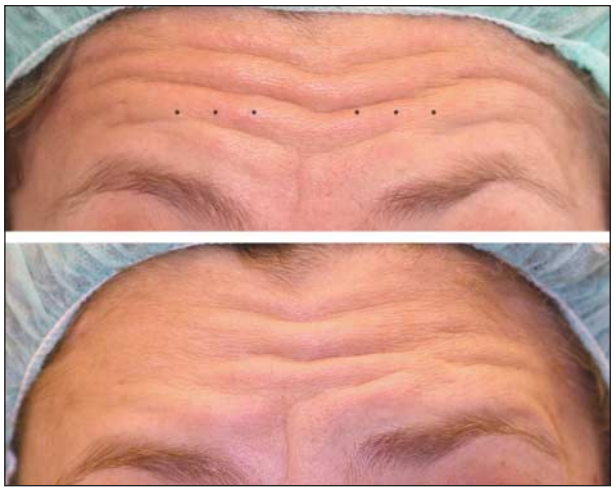

Figure 2. Forehead area of a 41-year-old woman at maximum frown before (A) and 10 weeks after (B) injection of 36 U of Dysport/Reloxin (right side of the forehead) and 12 U of Botox (left side of the forehead). The dots in panel A indicate the sites of injection. Botox and Dysport/Reloxin are proprietary names for botulinum A toxin products manufactured by Allergan (Irvine, California) and Ipsen Ltd (Slough, England), respectively.

Lowe et al[7] published a study in which the effect of Dysport/Reloxin was less sustained than that of Botox in a 2.5:1.0 regimen, which contradicts the results of our study. In addition, the effect of Botox increased 16 weeks after injection, which was not seen in any other study. These results need independent confirmation.

The results of the present study show that Dysport/Reloxin has a longer duration of effect on EMG activity and forehead wrinkles than does Botox at a unit dose conversion ratio of 3:1 (**Figure 2**). This suggests that the conversion ratio for bioequivalence may be less than 3:1.

We recommend this model as a relatively simple and accessible way of obtaining quantitative comparative data in a clinical treatment situation.

Syrus Karsai, MD
Robert Adrian, MD
Stefan Hammes, MD
Jürgen Thimm, MD
Christian Raulin, MD, PhD

Correspondence: Dr Karsai, Laserklinik Karlsruhe, Kaiserstrasse 104, Karlsruhe 76133, Germany (info@raulin.de).
Financial Disclosure: None reported.
Additional Contributions: Jochen Hirsch, MD, PhD, provided expert statistical assistance.

1. Brin MF, Blitzer A. Botulinum toxin: dangerous terminology errors. *J R Soc Med*. 1993;86(8):493-494.
2. Hambleton P, Pickett AM. Potency equivalence of botulinum toxin preparations. *J R Soc Med*. 1994;87(11):719.
3. Sampaio C, Costa J, Ferreira JJ. Clinical comparability of marketed formulations of botulinum toxin. *Mov Disord*. 2004;19(suppl 8):S129-S136.
4. Ranoux D, Gury C, Fondarai J, Mas JL, Zuber M. Respective potencies of Botox and Dysport: a double-blind, randomised, crossover study in cervical dystonia. *J Neurol Neurosurg Psychiatry*. 2002;72(4):459-462.
5. Poewe W. Respective potencies of Botox and Dysport: a double-blind, randomised, crossover study in cervical dystonia. *J Neurol Neurosurg Psychiatry*. 2002;72(4):430.
6. Rosales RL, Bigalke H, Dressler D. Pharmacology of botulinum toxin: differences between type A preparations. *Eur J Neurol*. 2006;13(suppl 1):2-10.
7. Lowe P, Patnaik R, Lowe N. Comparison of two formulations of botulinum toxin type A for the treatment of glabellar lines: a double-blind, randomized study. *J Am Acad Dermatol*. 2006;55(6):975-980.

COMMENTS AND OPINIONS

Infliximab-Induced Palmoplantar Pustulosis in a Patient With Crohn Disease

We read with interest the recent accounts in the *Archives* of paradoxical induction of psoriasislike disease in patients undergoing therapy with tumor necrosis factor α (TNF-α) inhibitors.[1-3] Similar reports have been published elsewhere in the dermatologic[4,5] and rheumatologic literature.[6-8]

Report of a Case. We report the case of a 37-year-old woman with Crohn disease who developed palmoplantar pustulosis (PPP) during treatment with infliximab. Palmoplantar pustulosis has not previously been reported in cases of Crohn disease treated with TNF-α inhibitors. Our patient was diagnosed as having Crohn disease in November 2005, had no history of psoriasis, and was otherwise healthy. A regimen of infliximab was begun at 5 mg/kg, and the patient had 3 infusions, January, June, and July 2006, resulting in complete remission of her bowel symptoms.

One month later, she developed classic PPP together with a mild psoriasiform eruption on the lower legs. In particular, the patient's feet were painful, which adversely affected her mobility. We prescribed betamethasone dipropionate in optimized vehicle ointment twice daily, polythene occlusion at night, and soap-free wash and moisturizer. Over the following 3 to 4 weeks, the patient improved clinically and symptomatically and did not need additional psoriasis treatment. By February 2007, the PPP had cleared, presumably due to the diminishing effects of infliximab, but there was a corresponding slight relapse of Crohn disease. The patient began treatment with azathioprine, and the Crohn disease again passed into remission. However, if this treatment does not provide adequate control in the future, we will reintroduce infliximab therapy and treat any skin changes with aggressive topical therapy, oral agents such as methotrexate, or switch to another anti–TNF-α agent such as adalimumab.

Comment. All anti–TNF-α agents, paradoxically, induce or exacerbate psoriasis, albeit rarely.[6] Although this is considered a class effect,[4] 1 case of etanercept-induced psoriatic lesions did not occur after switching to infliximab.[6] Usually the skin changes are self-limiting,[1,4] but in some cases they have been sufficiently severe to discontinue treatment.[6] The cause is unknown, although hypotheses include the cross-regulation between TNF-α and interferon[1] or the abnormal expression of TNF-α in eccrine sweat glands.[9] Understanding this paradox may help in our further understanding of the mechanisms, causes, and treatment of psoriasis.[3,10]

Michael J. Sladden, MBChB, MAE
Philip J. Clarke, MBBS, FRACGP
John Wettenhall, MBBS, FRACP

Correspondence: Dr Sladden, Department of Medicine, University of Tasmania, Launceston General Hospital, Launceston 7250, Australia (m.sladden@doctors.org.uk).
Financial Disclosure: None reported.

1. de Gannes GC, Ghoreishi M, Pope J, et al. Psoriasis and pustular dermatitis triggered by TNF-α inhibitors in patients with rheumatologic conditions. *Arch Dermatol*. 2007;143(2):223-231.
2. Ubriani R, Van Voorhees A. Onset of psoriasis during treatment with TNF-α antagonists: a report of 3 cases. *Arch Dermatol*. 2007;143(2):270-272.
3. Fiorentino DF. The yin and yang of TNF-α inhibition. *Arch Dermatol*. 2007;143(2):233-236.
4. Pirard D, Arco D, Debrouckere V, Heenen M. Anti-tumor necrosis factor alpha-induced psoriasiform eruptions: three further cases and current overview. *Dermatology*. 2006;213(3):182-186.
5. Lee HH, Song IH, Friedrich M, et al. Cutaneous side effects in patients with rheumatic diseases during application of tumour necrosis factor-alpha antagonists. *Br J Dermatol*. 2007;156(3):486-491.
6. Sari I, Akar S, Birlik M, Sis B, Onen F, Akkoc N. Anti-tumor necrosis factor-alpha-induced psoriasis. *J Rheumatol*. 2006;33(7):1411-1414.
7. Kary S, Worm M, Audring H, et al. New onset or exacerbation of psoriatic skin lesions in patients with definite rheumatoid arthritis receiving tumour necrosis factor alpha antagonists. *Ann Rheum Dis*. 2006;65(3):405-407.
8. Sfikakis PP, Iliopoulos A, Elezoglou A, Kittas C, Stratigos A. Psoriasis induced by anti-tumor necrosis factor therapy: a paradoxical adverse reaction. *Arthritis Rheum*. 2005;52(8):2513-2518.
9. Michaëlsson G, Kajermo U, Michaëlsson A, Hagforsen E. Infliximab can precipitate as well as worsen palmoplantar pustulosis: possible linkage to the expression of tumour necrosis factor-alpha in the normal palmar eccrine sweat duct? *Br J Dermatol*. 2005;153(6):1243-1244.
10. Heymann WR. Tumor necrosis factor inhibitor-induced pustular psoriasis? *J Am Acad Dermatol*. 2007;56(2):327-329.

Suppression of the HPA Axis in Pediatric Patients With Atopic Dermatitis

We read with interest the recent article by Schlessinger et al[1] that assessed the potential of the topical corticosteroid fluocinonide to suppress the hypothalamic-pituitary-adrenal