Evidence of a Decrease in Heart Rate and Skin Conductance Responses in PTSD Patients After a Single EMDR Session

Stéphanie Aubert-Khalfa
Hôpital de la Timone, Marseille, France

Jacques Roques
Centre de Traitement des Traumatismes Psychiques, Montpellier, France

Olivier Blin
Hôpital de la Timone, Marseille, France

Patients with posttraumatic stress disorder (PTSD) demonstrate abnormal psychophysiological responses to stressful events. Given that eye movement desensitization and reprocessing (EMDR) therapy appears to be a treatment of choice for trauma victims, the aim of the present study was to determine if psychophysiological responses to stress decreased after a single EMDR session. Six PTSD patients were treated by an EMDR therapist. Their psychophysiological responses (heart rate and skin conductance) were recorded before and after the EMDR session under two conditions: (a) in a relaxed state and (b) while visualizing their own traumatic event. At the end of the session, all patients had a significant reduction in their PTSD symptoms, which confirms previous results demonstrating the efficacy of the EMDR approach. Second, after only one EMDR session, heart rate and skin conductance during the trauma recall decreased significantly as compared to a relaxing state.

Keywords: EMDR; PTSD; psychophysiology; heart rate; skin conductance; treatment

Eye movement desensitization and reprocessing (EMDR) has been successfully used in patients with posttraumatic stress disorder (PTSD), and its efficacy is recognized in many international guidelines and the *Cochrane Review* (Bisson & Andrew, 2007). EMDR is a comprehensive psychotherapy approach that is guided by an information processing model (Shapiro, 2001). During this treatment, clients attend to a traumatic memory and its associated elements, in short sequential exposures, while simultaneously engaging in a dual-attention task, such as eye movements. While there is no definitive explanation as to how EMDR works, several hypotheses have been proposed (Christman, Kilian, Propper, & Phaneuf, 2003; MacCulloch & Feldman, 1996; Shapiro, 2001; Stickgold, 2002). Possible neurophysiological, psychophysiological, and neurobiological changes related to EMDR should especially be evaluated to better understand biological mechanisms involved in this treatment.

Generally, in anxiety disorders, panic attacks are accompanied by an increase on physiological measures (Bystritsky, Craeske, Maidenberg, & Shapiro, 1995; Wilhem & Roth, 2001). PTSD is also associated with abnormal psychophysiological responses in a resting state and with exposure to stressful events (Cohen et al., 2000; Hopper et al., 2006; Liberzon, Abelson, Flagel, Raz, & Young, 1999; Sack, Hopper, & Lamprecht, 2004). This is one reason why some researchers have attempted to explore the autonomic nervous system functioning before and after EMDR treatment. Barrowcliff, Gray, Freeman, and MacCulloch (2004) have shown a reduction in electrodermal arousal in healthy participants engaging in eye movements following elicitation of a negative memory. In patients with PTSD, heart rate (HR) and galvanic skin response decreased during EMDR (Wilson, Silver, Covi, & Foster, 1996), suggesting a relaxing effect of the eye movements. Psychophysiological arousal during trauma recall was also diminished after a successful

 DOI: 10.1891/1933-3196.2.1.51

therapeutic exposure in Vietnam veterans with PTSD (Boudewyns & Hyer, 1990). More recently, HR was shown to be significantly reduced during a trauma script after EMDR treatment in patients with PTSD (Sack, Lempa, & Lamprecht, 2007). This result appears to be related to heightened parasympathetic tone.

In order to replicate previous results and verify the efficacy of a single EMDR session, the present study aimed to further explore the psychophysiological responses to trauma after a single EMDR session by recording both HR and skin conductance responses during a relaxing state and during trauma recall. The skin conductance responses reflect rapid fluctuations in eccrine sweat gland activity in response to the liberation of acetylcholine by the sympathetic nervous system (Boucsein, 1992). In contrast, neural control of the heart depends on both sympathetic and parasympathetic pathways (Levy & Warner, 1994; Randall, 1977). According to previous results (Barrowcliff et al., 2004; Sack et al., 2007), we expected decreased sympathetic activity and increased parasympathetic activity after the EMDR session. We thus hypothesized that HR and skin conductance responses to the trauma event recall would significantly decrease.

Method

Participants

Six patients (one male and five females) not under medication, with an age ranging from 19 to 73 years (mean age = 31.1 ± 20.9 [*SD*]) and education ranging from 6 to 16 years (mean = 11.17 ± 3.37 [*SD*]), participated in the present study. All had suffered from traumatic events, and their symptom presentation fulfilled diagnostic criteria for PTSD according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM-IV;* American Psychiatric Association, 1994). One patient was widowed, one patient had a boyfriend, and the four others were single. The ethnic background of all patients was White. Two female patients were victims of rape, two patients had experienced the sudden death of a close relative, and the male patient had been physically attacked (by a knife stabbing). The last patient had been harassed over the course of 1 year and showed signs of complex trauma and suffered from anorexia.

Three of these six patients had multiple traumas. One of the females had been raped four times and treated for all but one of these, which was the target for this study. Two other patients also had multiple traumas necessitating additional EMDR sessions after completion of this study. All patients were prospectively studied as part of their clinical care. Informed consent was obtained from each patient prior to the therapeutic session.

Procedure

Before the onset of the session and 2 weeks later, patients had to complete French versions of psychometric questionnaires assessing their symptoms related to their posttraumatic state. The Posttraumatic Checklist Scale (PLCS) for the *DSM-IV* (Weathers, Litz, Herman, Huska, & Keane, 1993) and the Impact of Event Scale Revised (Weiss & Marmar, 1996) were completed in French (Brunet, King, & Weiss, 1998; Ventureyra, Yao, Cottraux, Note, & De Mey-Guillard, 2002). Two patients did not complete the two questionnaires 2 weeks after the treatment.

HR and skin conductance were recorded for each PTSD patient before and after the single EMDR session for a 30-second duration while patients were in a relaxed state and while they were visualizing their own traumatic event. For the relaxing period, participants were asked to breathe calmly, to close their eyes, and to relax. They were required to indicate when they felt relaxed and to maintain this state until asked to stop. Physiological recordings were obtained only when each participant indicated that he or she was in the relaxed state. Patients then visualized their own traumatic event and indicated when this was experienced. In a manner similar to the relaxed physiological recording, physiological measures were recorded for trauma recall during 30 seconds after the onset indicated by the patients. We did not record more than 30 seconds, thereby avoiding suffering for the patients.

Treatment

The EMDR treatment was administered by a therapist licensed in psychotherapy and who had received levels I and II of training in EMDR as provided by the French EMDR Institute (Paris, France) and who had 13 years of clinical experience with the EMDR method. Treatment was administered according to the standard protocol (Shapiro, 1995). EMDR's process measures were collected at the beginning of the session and following treatment. These include obtaining a score on the Subjective Units of Disturbance Scale (SUDS) to assess the level of distress in relation to the targeted traumatic memory (0 = no disturbance to 10 = the most disturbance possible). The validity of the desired positive cognition using the Validity of Cognition Scale (VOC) was also assessed (1 = completely false to 7 = completely true). The EMDR treatment was not finished for two patients, as the target memory changed. Thus, VOC and SUDS could not be provided for these two patients.

Physiological Measures

HR was acquired at 1,000 Hz, whereas skin conductance was acquired at 125 Hz, using the MP 30 system and Acknowledge software 3.6.7 (Biopac Systems Inc.). Electrocardiogram (ECG) was recorded using a standard three-leads configuration (Einthoven lead 2 configuration) (Biopac EL503). Skin conductance was recorded from the left hand with a pair of Ag-AgCl electrodes (0.8-cm diameter) attached to the palmar surface of the medial phalange of the index and middle fingers and filled with a 0.050 molar NaCl paste. All recording procedures followed the recommendations by Fowles et al. (1981).

Physiological Signals Processing

All physiological measures were continuously monitored during the recording and visually inspected off-line. Recording artifacts were identified and corrected by intrapolation, and otherwise were discarded. Corrections were necessary for only few seconds in two patients' recordings. Instantaneous R-R intervals were calculated from the ECG using a peak detection algorithm to obtain a continuous RR tachogram. Careful examination of the ECG and the tachogram ensured that the automatic R-wave detection procedure had been performed correctly. Skin conductance was smoothed at 0.5 seconds, and the Biopac peak detection function allowed the detection and counting of skin conductance responses that were superior to 0.02 μSiemens per second.

Statistics

Because of the small number of participants and an absence of normal distribution of the data, the nonparametric Wilcoxon test was used to compare psychophysiological differences between the relaxing state and the trauma recall, before and after EMDR treatment. Nonparametric Mann–Whitney tests were also performed to analyze differences between self-report measures before and after treatment because of the absence of two self-report measures.

Results

Prior to the single EMDR session, all patients had scores on the PLCS (mean 63.8 ± 7.0 [*SD*]) and Impact of Event Scale Revised (mean 60.6 ± 10.3 [*SD*]) tests that were in the range of PTSD patients (Brunet et al., 1998; Ventureyra et al., 2002). After the EMDR treatment, scores were significantly reduced for the PCLS (mean 49.25 ± 10.3 [*SD*]) ($z = 2.03$, $n = 10$, $p = 0.038$) and for the Impact of Event Scale–Revised (mean 33.0 ± 17.6 [*SD*]) ($z = 2.13$, $n = 10$, $p = 0.38$). These results are consistent with the VOC and SUDS changes respectively, from 1.8 ± 0.9 (*SD*) to 6.25 ± 1.5 ($z = 2.51$, $n = 9$, $p = 0.016$) and from 8.0 ± 1.4 (*SD*) to 0 ± 0 ($z = 2.57$, $n = 9$, $p = 0.016$), indicating improvement of the PTSD symptoms in patients.

The reduction in symptom scores following the EMDR session stands in line with a complete decrease of the psychophysiological responses. As illustrated in Figure 1, the difference of skin conductance ("during the trauma recall" minus "during the relaxing state") was significantly greater before the EMDR treatment (mean 7.64 ± 4.6 [*SD*]) than after the treatment (mean –0.67 ± 2.9 [*SD*]) ($z = 1.99$, $n = 6$, $p = .046$). This indicates that the skin conductance responses to trauma recall as compared to the baseline relaxing state have significantly diminished after the EMDR session. The same result was obtained with the HR since HR differences ("during the trauma recall" minus "during the relaxing state") were significantly higher (mean 16.97 ± 8.0 [*SD*]) than the HR difference between the two states after the treatment (mean 2.43 ± 3.4 [*SD*]) ($z = 2.2$, $n = 6$, $p < .028$) (see Figure 2).

Discussion

Patients' scores on all scales (SUDS, VOC, PCLS, and the Impact of Event Scale–Revised) were significantly reduced after the single EMDR session. This strengthens the results of previous studies on the efficacy of EMDR (Maxfield & Hyer, 2002). However, the mean scores remained relatively high since three patients had multiple traumatizations necessitating other EMDR sessions. The three other patients reported complete recovery after the single-session therapy.

Despite the small number of patients, physiological responses (HR and skin conductance responses and traumatic event compared to the relaxing state) were significantly decreased after only one EMDR session. After this single EMDR session, physiological differences between both conditions were equal (skin conductance responses) or close (HR) to zero. Results highlight the effects of EMDR therapy on the autonomic nervous system, especially the sympathetic activity reflected by the reduction in skin conductance responses. Physiological hyperarousal described in PTSD patients (Liberzon et al., 1999) could then no longer be observed after the EMDR session.

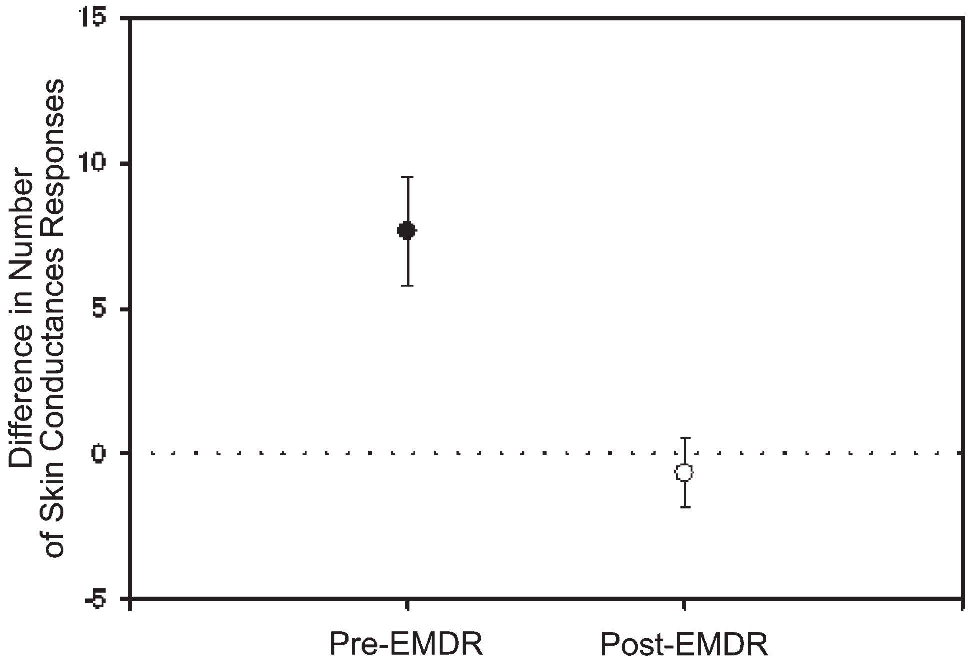

FIGURE 1. Skin conductance. Means and standard errors of the number of skin conductance responses during the trauma recall minus during the relaxing state (used as a baseline) before and after the EMDR session.

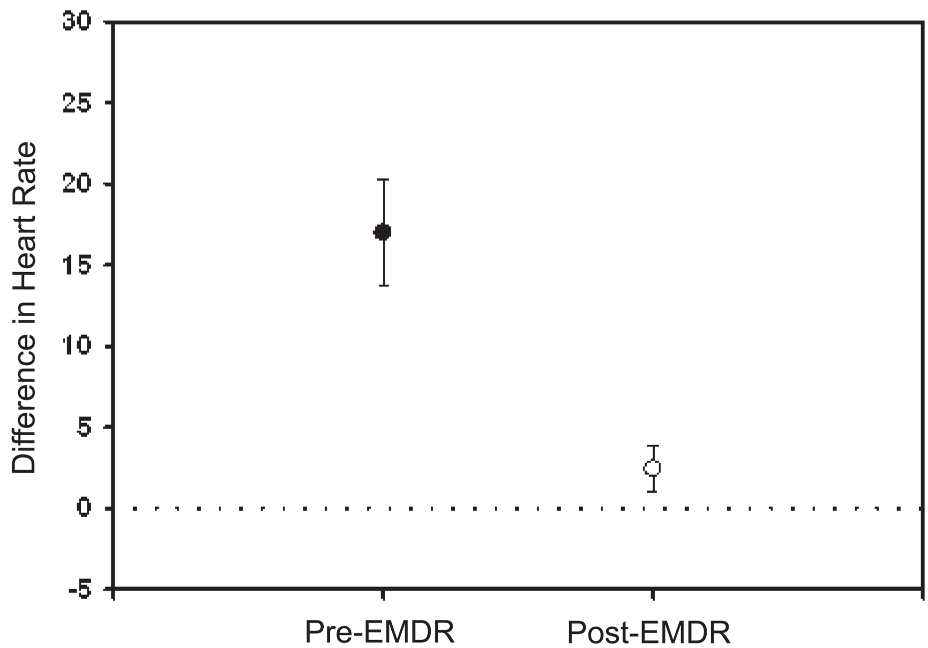

FIGURE 2. Increase in heart rate (HR) during trauma recall. Means and standard errors of HR during the trauma recall minus during the relaxing state (used as a baseline) before and after the EMDR session.

The skin conductance response is known to be influenced by the reticular formation, the anterior hypothalamus, and, at a higher level, the limbic system (Venables & Christie, 1980). The amygdala seems to have an excitatory effect on electrodermal activity, whereas the hippocampus has an inhibitory effect (Bagshaw, Kimble, & Pribram, 1965; Pribram & McGuinness, 1975). The hyperarousal reduction after the EMDR session may then reflect changes in brain function, such as the inhibitory control of the amygdala. Further experiments should test for this hypothesis.

One of the limitations of this study is the small number of patients. In spite of this, the impact of the EMDR session on psychophysiological responses during visualization of trauma events in patients with PTSD was statistically significant. In addition, these results are in accordance with previous results (Barrowcliff et al., 2004; Sack et al., 2007).

A second limitation is that the trauma memory was repeated two times (before and after the EMDR session). The decreased skin conductance responses and HR differences after the EMDR session may reflect habituation to the stimulus. We suggest that this is not the case; at least three stimuli presentation are required to observe habituation in electrodermal activity (Levinson & Edelberg, 1985). In addition, prior to the EMDR treatment, the patients had observed neurovegetative reactions (e.g., tears or stomachache) as they would think or speak of their trauma. The autonomic hyperactivity is part of the PTSD symptoms (Weathers et al., 1993; Weiss & Marmar, 1996). However, further experiments should include a control group to test for the effect of repeated measurement of psychophysiological responses to trauma recall.

Conclusion

Despite the limitations of this study, previous results have been replicated. In order to explore the neural mechanisms underlying EMDR treatment and its effect on physiological modifications, future experiments should simultaneously record the patients' brain activity via more sophisticated scans, such as PET, fMRI, or DTI, to assess the involvement of amygdala, hippocampus, and frontal structures in the decrease of sympathetic nervous activity in PTSD patients after EMDR treatment. Functional neuroimaging studies have already demonstrated that PTSD is associated with activity changes in frontal and limbic systems (for a review, see Francati, Vermetten, & Bremner, 2007). It should be determined whether the EMDR treatment effect relies on neural mechanisms involving these brain structures.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Bagshaw, M. H., Kimble, D. P., & Pribram, K. H. (1965). The GSR of monkeys during orienting and habituation after ablation of the amygdala, hippocampus and inferotemporal cortex. *Neuropsychologia, 3,* 111–119.

Barrowcliff, A. L., Gray, N. S., Freeman, T. C. A., & MacCulloch, M. J. (2004). Eye-movements reduce the vividness, emotional valence and electrodermal arousal associated with negative autobiographical memories. *Journal of Forensic Psychiatry and Psychology, 15,* 325–345.

Bisson, J., & Andrew, W. (2007). Psychological treatment of post-traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews,* Issue 3, Article No. CD003388.

Boucsein, W. (1992). *Electrodermal activity.* New York: Plenum Press.

Boudewyns, P. A., & Hyer, L. A. (1990). Physiological response to combat memories and preliminary treatment outcome in Vietnam veteran PTSD patients treated with direct therapeutic exposure. *Behavior Therapy, 21,* 63–87.

Brunet, A., King, S., & Weiss, D. S. (1998). *Version française de l'Impact of Event Scale Revised.* Unpublished manuscript.

Bystritsky, A., Craeske, M., Maidenberg, T. V., & Shapiro, D. (1995). Ambulatory monitoring of panic patients during regular activity: A preliminary report. *Biological Psychiatry, 38,* 684–689.

Christman, S. D., Kilian, J. D., Propper, R. E., & Phaneuf, K. A. (2003). Bilateral eye movements enhance the retrieval of episodic memories. *Neuropsychology, 17,* 221–229.

Cohen, H., Benjamin, J., Geva, A. B., Matar, M. A., Kaplan, Z., & Kotler, M. (2002). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrun analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research, 96*(1), 13.

Fowles, D. C., Christie, M. J., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal measurements. *Psychophysiology, 18*(3), 232–239.

Francati, V., Vermetten, E., & Bremner, J. D. (2007). Functional neuroimaging studies in posttraumatic stress disorder: Review of current methods and findings. *Depression and Anxiety, 24,* 202–218.

Hopper, J. W., Spinazzola, J., Simpson, W. B., & van der Kolk, B. A. (2006). Preliminary evidence of parasympathetic influence on basal heart rate in posttraumtic stress disorder. *Journal of Psychosomatic Research, 60*(1), 83–90.

Levinson, D. F., & Edelberg, R. (1985). Scoring criteria for response latency and habituation in electrodermal research. *Psychophysiology, 22,* 417–426.

Levy, M. N., & Warner, M. R. (1994). *Parasympathetic effects of cardiac function.* In J. A. Armour & J. L. Ardell (Eds.), *Neurocardiology* (pp. 53–76). New York: Oxford University Press.

Liberzon, I., Abelson, J. L., Flagel, S. B., Raz, J., & Young, E. A. (1999). Neuroendocrine and psychophysiologic responses in PTSD: A symptom provocation study. *Neuropsychopharmacology, 21,* 40–50.

MacCulloch, M. J., & Feldman, P. (1996). Eye movement desensitisation treatment utilises the positive visceral element of the investigatory reflex to inhibit the memories of post-traumatic stress disorder: A theoretical analysis. *British Journal of Psychiatry, 169,* 571–579.

Maxfield, L., & Hyer, L. A. (2002). The relationship between efficacy and methodology in studies investigating EMDR treatment of PTSD. *Journal of Clinical Psychology, 58,* 23–41.

Pribram, K. H., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. *Psychological Review, 82,* 116–149.

Randall, W. C. (1977). Sympathetic control of the heart. In W. C. Randall (Ed.), *Neural regulation of the heart* (pp. 43–94). New York: Oxford University Press.

Sack, M., Hopper, J. W., & Lamprecht, F. (2004). Low respiratory sinus arrhythmia and prolonged psychophysiological arousal in posttraumatic stress disorder: Heart rate dynamics and individual differences in arousal regulation. *Biological Psychiatry, 55,* 284–290.

Sack, M., Lempa, W., & Lamprecht, F. (2007). Assessment of psychophysiological stress reactions during a traumatic reminder in patients treated with EMDR. *Journal of EMDR Practice and Research, 1,* 15–23.

Shapiro, F. (1995). *Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures*. New York: Guilford Press.

Shapiro, F. (2001). *Eye movement desensitization and reprocessing* (2nd ed.). New York: Guilford Press.

Stickgold, R. (2002). EMDR: A putative neurobiological mechanism of action. *Journal of Clinical Psychology, 58,* 61–75.

Venables, P. H., & Christie, M. J. (1980). *Electrodermal activity*. In I. Martin & P. H. Venables (Eds.), *Techniques in psychophysiology* (pp. 3–67). New York: Wiley.

Ventureyra, V. A. G., Yao, N., Cottraux, J., Note, I., & De Mey-Guillard, C. (2002). The validation of the Post-Traumatic Checklist Scale (PCLS) in posttraumatic stress disorder and non-clinical participants. *Psychotherapy and Psychosomatics, 29*(3, Pt. 1), 232–238.

Weathers, F. W., Litz, B. T., Herman, D. S., Huska, J. A., & Keane, T. M. (1993, October). *The PTSD checklist: Reliability, validity and diagnostic utility*. Paper presented at the annual meeting of the International Society for Traumatic Stress Studies, San Antonio, TX.

Weiss, D. S., & Marmar, C. R. (1996). The impact of Event Scale Revised. In J. P. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD: A practitioner's handbook* (pp. 399–411). New York: Guilford Press.

Wilhem, F. H., & Roth, W. T. (2001). The somatic symptom paradox in DSM-IV anxiety disorders: Suggestions for a clinical focus in psychophysiology. *Biological Psychology, 57,* 105–140.

Wilson, D. L., Silver, S. M., Covi, W. G., & Foster, S. (1996). Eye movement desensitization and reprocessing: Effectiveness and autonomic correlates. *Journal of Behavior Therapy and Experimental Psychiatry, 27,* 219–229.

Acknowledgments. We thank Krista Hyde for English editing and comments.

Correspondence regarding this article should be directed to Stéphanie Aubert-Khalfa, CIC-Unité de Pharmacologie Clinique et d'Evaluation Thérapeutique, Assistance Publique Hôpitaux de Marseille, Institut des Neurosciences Cognitives de la Méditerranée, UMR 6193 CNRS, Université de la Méditerranée, Faculté de Médecine, Hôpital de la Timone, 13385 Marseille Cedex 5, France. E-mail: skhalfa@skhalfa.com