

PAPER**GENERAL; PATHOLOGY/BIOLOGY**

Vito Borraccia,¹ M.D.; Sara Sblano,² M.D., Ph.D.; Felice Carabellese,³ M.D.; Rosalisa De Sario,¹ M.D.; Roberto Zefferino,⁴ M.D.; and Francesco Vinci,² M.D.

Stress-Related Temporary Hearing Loss—Evaluation of Bio-Humoral Parameters: Forensic and Criminological Applications

ABSTRACT: The body-alarm reaction results from the activation of hypothalamic–pituitary–adrenal axis, which can lead to physio-psychological phenomena such as an exclusion/occlusion of the sense of hearing. One hypothesis to explain this alteration consists in a hydromechanical dysfunction of the internal ear attributable to antidiuretic hormone. In this study, we evaluated the perception of acoustic stimuli administered in stressful conditions in 14 phobic patients and in 20 healthy subjects, in order to assess the influence of stress on perceiving capabilities. We also measured the concentration of salivary cortisol and IL-1 β and neurovegetative parameters to objectivise and quantify the physiological reactions. Our results show a worse perception of the frequencies of the human voice under stress; these findings could have a dual value: in the legal field, concerning criminal liability, and on the operative context, regarding the efficiency of verbal communication among law enforcement officers in situations inducing intense emotional stress.

KEYWORDS: forensic science, forensic pathology, stress, hearing loss, hydromechanical dysfunction, police force responsibility, cortisol, IL-1

The present study owes its origin to a question posed during an assessment made in the course of a criminal investigation. We were asked whether a situation of acute stress arising during a shooting could have caused a police officer to fail to hear the verbal cease-fire order, given from very short distance and in a loud voice, so that he continued to fire causing fatal injury to a malefactor.

It is well known that one of the most ancient phylogenetic human responses is the reaction to the perception of danger (real or imagined). This reaction, known as a body-alarm reaction, can give rise to the different behavioral and psychophysical responses. These are attributed to the emotional activation induced by the negative stimulus, organized in the limbic area, that manifests at the biological–somatic level by producing neurovegetative and endocrine alterations, for example, and at the psychological–behavioral level by triggering the motor sequences allowing the subject to turn and fight or else to flee the danger (1). This body-alarm reaction resulting from the activation of one of the main neuroendocrine axis, hypothalamic–pituitary–adrenal axis, prepares the organism for the fight or flight reaction (2). It is characterized by a first phase consisting of hypersecretion of adrenaline, noradrenaline and antidiuretic hormone (ADH) and then after a few minutes, also of cortisol. The production of these substances by the pituitary gland and adrenal cortex induces

a number of alterations in the organism such as an enhanced attention and better body reaction to a threat, and an increased blood supply to the muscle masses to equip the body for greater functional demands, as well as a reduced sensitivity to pain.

In association with these alterations, which can be considered adaptive reactions, some physio-psychological phenomena can also develop, such as aberrations of psychogenic type, whereby the mind, being totally concentrated on the threat, can partially or totally exclude, in a completely unconscious fashion, other sensory information arriving in the same context. In this way, an exclusion/occlusion of the sense of hearing, for example, can occur. This phenomenon is tantamount to a complete distortion of the hearing processes and is typically associated with a lowered threshold of perception. One suggestive hypothesis that could justify the occurrence of this phenomenon in the course of acute stress seems to be a hydromechanical dysfunction of the internal ear, with the onset of cochlear hydrops, an excess of fluid in the internal ear. This would be attributable to ADH. In fact, even if the main physiological role of ADH is exerted at the level of the renal glomeruli, where it is involved in regulating the reabsorption of fluids through specific channels called aquaporins, ADH receptors and aquaporins have also been identified in the internal ear (3–5), where they may take part in modifying the cochlear fluids.

In light of the above, the operative and legal implications of such alterations of the perceptions can easily be appreciated in cases where they could occur in police officers or, in general, in members of rescue teams. Difficulty in hearing an order during the operative phase, perhaps during combat, or failure to hear the sound of an impending threat (e.g., the arrival of a train, or gunshots) poses a very high risk and, from a medico-legal standpoint, could be a factor of primary importance when assessing possible liability. An

¹Ufficio Sanitario XI Reparto Mobile, Polizia di Stato, Via Cacudi, 3, 70123 Bari, Italy.

²Sezione di Medicina Legale (Di.M.I.M.P.), Università di Bari, Piazza G. Cesare 11, 70124 Bari, Italy.

³Sezione di Criminologia e Psichiatria Forense (Di.M.I.M.P.), Università di Bari, Piazza G. Cesare, 11, 70124 Bari, Italy.

⁴Dipartimento di Scienze Mediche e del Lavoro, Università di Foggia, Viale L. Pinto 1, 71100 Foggia, Italy.

Received 27 Oct. 2010; and in revised form 29 Jan. 2011; accepted 1 Mar. 2011.

investigation of altered perceptions during acute stress conducted in armed subjects before and during a shooting revealed a reduction in the hearing capacity in no <88% of cases, while in only 15% of cases did the opposite occur, that is, enhanced hearing (6,7).

Moreover, even in the clinical setting, an analysis of these phenomena is of extreme interest in our view, especially in the study of subjects suffering from phobias, such that in the course of unexpected, sudden exposure to the phobic stimulus, they could find themselves endangered by this condition of dysperception.

Many attempts have already been made to objectivise and quantify the physiological reactions to psychological stress (ambulatory monitoring of the autonomic nervous system parameters, such as the heart rate, as well as the hypothalamus–pituitary axis and the immune functions, by assessing leukocyte proliferation and cytotoxicity induced by the natural killer cells). Nevertheless, the parameter shown to be most efficacious in this context has been the dosage of salivary cortisol. Salivary cortisol is closely correlated to circulating free cortisol, the biologically active form, and the levels rise within a very few minutes in response to acute stress (8,9), reaching a peak after 15–30 min and having a half-life of approximately 1 h. Activation of the hypothalamic–pituitary–adrenal axis, during intense physical and psychological stress, thus results in a temporary increase in circulating cortisol. There is also a reciprocal influence between the immune system and the neuroendocrine activity modulated by some interleukins (IL-1, IL-2, and IL-6) (10).

Therefore, in this study, we aimed to assess whether variations in the levels of salivary cortisol and IL-1 β , induced by deliberately provoked acute stress, could affect the hearing of a human voice. This was studied on the basis of the frequencies perceived best and/or worst, and of the different gain.

Materials and Methods

After receiving a full illustration of the purpose and aims of the study, two groups of voluntary subjects gave written informed consent to take part. One of the groups, defined as controls, included 20 subjects, 10 of whom were students or junior residents at the ENT Clinic at the Faculty of Medicine and Surgery, Bari University Hospital, with a negative history for phobias. Mean age in this group was 29.30 ± 2.74 years (min 24–max 34). The other 10 subjects in this group included five men aged 28.00 ± 2.74 years (min 24–max 31) and five women aged 30.60 ± 2.97 (min 27–max 34). None of the subjects were currently taking medications.

The other group consisted of subjects affected by specific phobias followed up at the Psychiatric Clinic of Bari Polyclinic. There were 14 subjects in this group, five men and nine women, with a mean age of 31.33 ± 3.21 years (min 29–max 35). In each of them, the diagnosis had been made after full clinical and psychodiagnostic assessment (BSSRSPP [an Italian test for agoraphobia]). None of them was receiving pharmacological therapy. A preliminary study of the sensitivity to specific elements was made by showing the subjects filmed images of the relative phobic stimulus. This administration was an integral part of a course of treatment detailed in a study protocol approved by the appropriate ethics committee.

A complete clinical history was taken of all members of both groups, paying particular attention to the presence/absence of phobias, and all underwent an ENT visit to exclude any hearing deficiency.

Using a special helmet for 3D vision, all participants were shown images that can trigger brief, intense fear (*terrifying visual stimuli*), interposed in a neutral film sequence. This helmet provides 3D stereovision and is connected to a stereo audio system to make the experience as realistic as possible. To identify any phases of

hearing loss in moments of stress, some acoustic stimuli (previously recorded in digital format) were then administered.

The acoustic stimuli were regulated by the researcher using a wired digital sound reproduction system; they included different frequency patterns (achieved by applying audio filters) and different volumes to verify whether these variables had any effect on the level of perception of the acoustic stimuli during the acute stress phase. Changes in the volume (gain) were made at the moment of administration of the stimuli using the digital potentiometer provided in the device.

First of all, five conventional numerical sequences (1249, 6166, 3826, 7667, 3264) were recorded in the laboratory, as well as two opposite orders with the same final sound (“ceasefire” and “fire”) in MP3 format on a single base file. These were spoken by a male voice, recorded using a *Logitech* digital microphone (Logi-tek Ltd., Stamford, Lincolnshire, UK) and a dual processor AMD computer (Advanced Micro Devices, Inc., Sunnyvale, CA). For experimental purposes, sequences with a *Doppler* effect were also recorded.

Then, using audio processing software (Adobe Audition 2006; Adobe Systems Inc., San Jose, CA), the sequences were processed by altering the frequencies to obtain three traces (*normal frequency*, *high frequency*, and *low frequency*).

The operations carried out on the base file were as follows:

- Volume increase around the frequency 1.6 K by +15.
- Volume reduction around the frequency 46 K by –15.
- Volume reduction around the frequency 93 K by –15.
- Volume reduction around the frequency 187 K by –15.
- Volume reduction around the frequency 375 K by –15.
- Volume reduction around the frequency 4 K by –4.7.
- Volume reduction around the frequency 12 K by –10.

The low-frequency MP3 file was generated using a filter for cutting down high frequencies and amplifying low ones using the fast Fourier transform (FFT). In practice, a filter is applied to the base file (normal frequency) to amplify frequencies ranging from 350 to 500 Hz and reduce the remainder. The high-frequency MP3 file was obtained by applying a filter for cutting down low frequencies and amplifying high ones using the FFT. To the base file (normal frequency), a filter was applied to amplify frequencies ranging from 880 to 3650 Hz and lower the remainder. To use the sequences during the course of the experiments of the subject’s ability to recognize sounds, the single numerical sequences were isolated by Adobe Audition software to obtain 15 numerical sequences and six sentences (two normal frequency, two high frequency, and two low frequency) in MP3 format. The acoustic stimuli employed are resumed in Table 1.

The autonomic nervous system parameters (heart rate using a digital pulse oximeter; arterial pressure) were continuously monitored, using the values obtained at the time when the salivary samples were taken as reference for correlations with variations in the endocrine parameters.

The salivary samples were collected with Sarstedt Salivettes (SARSTEDT Group, Numbrecht Rommelsdorf, Germany), on the day before the experiment and again on the day of the experiment, at 8 AM and 3 PM (8.00 and 15.00 hours). Participants were asked not to eat or drink any substance (apart from water) and to refrain from smoking for 1 h before salivary sampling, so as to reduce food residues and salivary stimulation to a minimum. Salivary sampling was carried out by the standard method:

- The mouth was rinsed with water.
- A sterile Salivette (removed from the container) was gradually lowered into the mouth without touching it with the hands.
- The subject chewed the Salivette (for 2 or 3 min).

TABLE 1—Acoustic stimuli employed.

(1)	(7)	(13)	(16)
1-2-4-9 NF	3-8-2-6 NF	3-2-6-4 NF	Ceasefire NF
(2)	(8)	(14)	(17)
1-2-4-9 HF	3-8-2-6 HF	3-2-6-4 HF	Ceasefire HF
(3)	(9)	(15)	(18)
1-2-4-9 LF	3-8-2-6 LF	3-2-6-4 LF	Ceasefire LF
(4)	(10)		(19)
6-1-6-6 NF	7-6-6-7 NF		Fire NF
(5)	(11)		(20)
6-1-6-6 HF	7-6-6-7 HF		Fire HF
(6)	(12)		(21)
6-1-6-6 LF	7-6-6-7 LF		Fire LF

NF, normal frequency; LF, low frequency; HF, high frequency.

- The Salivette was placed in the test tube.
- The sample was transported in a thermal container at 5°C and then preserved at -20°C until arrival at the laboratory of the Department of Medical and Occupational Sciences of the University of Foggia.

For quantitative analysis of salivary cortisol, the Salimetrics HS-Cort kit was used. As salivary cortisol is not affected by the percentage of salivary flow, only the concentration was used in the analysis. IL-1 β was determined using an ELISA kit (Roche, Indianapolis, IN). Total proteins were analyzed with the Bradford method using a commercial kit. The results for IL-1 β are expressed as the percent ratio of IL-1 β and salivary proteins. Inter- and intra-sample percent variations of <8% were obtained.

In the second phase of the experiment, within 20–30 sec of sight of the stressful stimulus and administration of the acoustic stimulus, the subject's perception of these was measured using two different systems:

- The subject was given a portable electronic device model Pr-110 μ P/S that measures the reaction time, but set in manual touch-button mode: he/she should press the button corresponding to the perceived acoustic stimulus among the three alternatives: (a) order n. 1: "fire"; (b) order n. 2: "ceasefire"; and (c) numerical sequence.

In this study phase, it was decided not to measure the reaction time, although this will be measured in the continuation of the present study. Instead, the subject was informed of what would happen, so as to increase the level of stress during the experiment.

- At the end of the experiment, the subjects underwent a brief structured interview, preceded by technical memory reinforcement exercises, to ascertain whether the claimed failure to perceive the orders was attributed to the effect of the stressful event on the recall capacity or else on the hearing capacity. Finally, it should be noted that in the data assessment, the Professional Stress Scale (PSS), modified for use with students, was used to estimate the individual perception of the stress.

Statistical Analysis

The difference between the cortisol concentrations found at 08.00 and 15.00 hours was calculated by taking into account the concentration at 08.00 minus the concentration at 15.00 hours.

DIFF1 indicates the difference found on the previous day, and DIFF2 the difference on the experimental day.

The percent cortisol difference was calculated by dividing the cortisol difference between 08.00 and 15.00 hours by the final concentration. PERC1 indicates the percent ratio of the cortisol difference during the experiment, and PERC2, the percent difference on the previous day.

The results for IL-1 β are expressed as the ratio between IL-1 β and salivary proteins. Student's *t*-test was used to assess the differences between the means; the chi-squared test was used to determine statistically significant associations between the PSS subscales and the levels of salivary cortisol and IL-1 β .

Results

No significant variation was observed in control groups either as regards the neurovegetative parameters (increased heart rate) or the salivary cortisol and IL-1 β values. All the hearing stimuli administered during the terrifying audiovisual stimuli were well perceived by all the subjects in this group.

Instead, in the group of phobic subjects, we obtained the following results. Arterial pressure (systolic and diastolic) measured before and after the experiment was higher than the measurements made at the same times on the previous day (Table 2); this difference was statistically significant ($p < 0.05$).

The changes in the heart rate at the start of the experiment and the maximum values recorded during the experimental phase are shown in Table 3. A statistically significant increase was obtained during the experiment ($p < 0.05$).

In the group of phobic subjects, the acute stress provoked by the administration of terrifying audiovisual stimuli was revealed by differences in the values of salivary cortisol and IL-1 β in the two experimental phases.

The double sampling, carried out on the previous day so well before the time of the experiment and then during the experimental phase, reduced the errors as compared to control values. The differences observed and the association with increases in heart rate and arterial pressure were significantly different ($p < 0.05$) from those obtained during measurements made on the previous day, confirming the acute stress provoked by the administration of the terrifying audiovisual stimuli to the group of phobic subjects.

Table 4 indicates the mean, median, and standard deviation for the salivary cortisol concentrations during the experimental day and the previous day.

TABLE 2—Arterial pressure: indices of central tendency and dispersion.

	Start of the Experiment	End of the Experiment	Previous Day 08.00 AM	Previous Day 03.00 PM
Mean	135.3/89	135.3/89	133.2/84	131.8/84
Median	136.5/90	135/90	130/80	130/83
Standard deviation	15.4/10.2	14.6/9.9	12.5/7.2	14.6/8.7

TABLE 3—Heart rate: indices for central tendency and dispersion.

	Start of the Experiment	Maximal Cardiac Frequency	Previous Day 08.00 AM	Previous Day 03.00 PM
Mean	84	102	78	80
Median	88	96	76	76
Standard deviation	0.74	1.2		

TABLE 4—Cortisol concentrations ($\mu\text{g}/\text{dL}$).

	Start of the Experiment	End of the Experiment	Previous Day 08.00 AM	Previous Day 03.00 PM
Mean	0.774	0.895	0.419	0.157
Median	0.524	0.812	0.316	0.125
Standard deviation	0.645	0.630	0.249	0.096

TABLE 5—Indices of cortisol concentrations.

	DIFF2	DIFF1	PERC1	PERC2
Mean	0.262	0.121	2.076	0.075
Median	0.202	0.000	1.835	0.093
Standard deviation	0.213	0.722	1.600	0.711

Table 5 shows the mean, median, and standard deviations for the differences in salivary cortisol at the start and end of the experiment (DIFF1), and mean, median, and standard deviation for the differences in cortisol concentrations in the morning and afternoon of the previous day (DIFF2), the cortisol index during the experiment (PERC1), and the cortisol index during the previous day (PERC2).

Table 6 shows the mean, median, and standard deviation of IL-1 β salivary concentrations during the experiment and during the previous day. Mean concentrations of IL-1 β were higher at the end of the experiment than at the start, and this increase was statistically significant.

As to the perception of acoustic stimuli, we obtained the results shown in Table 7 for the perception of the numerical sequences, and in Table 8 for the perception of the orders.

Discussion

Hydromechanical dysfunction of the ear occurs as a reaction to numerous phenomena and may or may not be associated with neurosensorial hearing loss. It is characterized by an increased production of fluid in the internal ear, secondary to hypersecretion of ADH in response to either exogenous stimuli, largely of an environmental nature (weather, temperature, and atmospheric pressure

TABLE 6—Concentrations of IL-1 β (pg/dL).

	Start of the Experiment	End of the Experiment	Previous Day 08.00 AM	Previous Day 03.00 PM
Mean	3.286	4.474	2.824	3.682
Median	2.531	3.612	2.325	3.005
Standard deviation	2.492	3.303	1.922	2.860

TABLE 7—Numerical sequences: percentage of failure to respond and/or error.

	Men, %	Women, %
Low frequency	80	100
Normal frequency	60	80
High frequency	10	30

TABLE 8—Orders: percentage of failure to respond and/or error.

	Men, %	Women, %
Low frequency	80	100
Normal frequency	40	40
High frequency	0	0

changes), or endogenous stimuli (stress) (11,12), and by an anomalous response by the aquaporins (13,14).

The individual response to stimuli that trigger feelings of fear, anger, or happiness depends on cultural and idiopathic factors. The system that processes the incoming data and determines the relative decision-making flow is, as stated previously, the limbic system consisting of the amygdala, hippocampus, thalamus, hypothalamus, etc., that can influence the responses of both the autonomic nervous system and the neuroendocrine system (1,2).

It is the interaction between the amygdala and the prefrontal neo-cortex that lies at the heart of emotional intelligence. The amygdala is able to set off a sort of “neural trigger” that can produce an autonomic response, while the cortex processes a more sophisticated type reaction. Some researchers have demonstrated that in the first milliseconds of perception of an object, not only does the subject unconsciously realize what the object is but also whether he/she likes it or not. However, the recall mechanism triggered by the amygdala is a primitive, relatively simple mechanism that therefore gives rise to errors of perception when the subject is faced with a similar but not identical element. This can induce a reaction based on the emotions before conscious thought intervenes (precognitive emotions), in other words, even before the information is processed at the cortical level. In this sense, the emotional circuits, and especially those relative to anxiety, play an essential role in clinical psychology issues.

This emotional state, which is not completely understood and about which no consensus has yet been reached, underlies many psychopathological pictures such as panic attacks, phobias (specific and social), the obsessive-compulsive disorder, the stress-related posttraumatic disorder and generalized anxiety, as well as some situations that are not necessarily pathological, mainly associated with fear. Fear, in the sense of an emotional response to a recognizable threat or danger, generally coming from outside, lies at the root of all anxiety responses. It is characterized by an immediate recognition of impending danger and by a reaction that is normally proportional to the level of the stimulus. From the physiological standpoint, in humans, there is a true cerebral circuit of fear. According to LeDoux (15), there is a “high” cortical pathway (slower, implying awareness) and a “low” subcortical pathway that does not involve the cortex (faster and less aware), and then conscious processing occurs. When the response is experienced as a conscious feeling, other higher-level cognitive systems offer further opportunities for regulating the emotional reaction. This would explain why some phobic patients feel a strong anxiety: despite being aware that they suffer from an irrational, unreasonable fear of something, they are still not able to control this anxiety.

The results of our experiment showed a worse perception of acoustic stimuli under stress, with a rate of 40–60% (depending on the type of stimuli) of failed or wrong responses in the normal frequency range of the human voice; the extent of this stress-induced misperception is even greater for the low frequencies, while it appears to decrease in the high range. Moreover, the combined study of neurovegetative parameters (changes in heart rate and arterial pressure) and salivary cortisol and interleukin values, which in our experimental sample showed a rise during stressful situations, proved to be a useful method. In addition, the investigation opens out interesting fields of inquiry into the possible incidence that psycho-physical conditions related to feelings of anxiety—so, not necessarily pathological—could have on questions of criminal liability, although further work needs to be carried out on this experiment to confirm the trend.

The findings of this investigation could therefore have a dual value. First, in the legal field if this experimental evidence was found in a suspect, it could provide useful elements, suggesting

extenuating circumstances or even absolving the subject from criminal liability.

In particular, in light of our findings, in the case that gave rise to the experiment the hypothesis that the police officer could, in conditions of intense emotional disturbance induced by acute stress, have failed to hear the ceasefire order given by the commanding officer, from very short distance and in a loud voice, appears more likely. This would be attributable to a selective temporary hearing loss of some frequencies normally audible to the human ear. In this case, he might have failed to obey the order for reasons outside his own volition, as supported also by the claims of the witnesses at the time of the preliminary hearing.

This would imply the possibility of applying, during a trial, one of the causes of absolution from blame, as it would invalidate, in part or in toto, the psychological element of the crime. In fact, if the psychological link between the author of a crime and her/his conduct is lacking, the action can no longer be regarded as a conscious one taken with full awareness of the consequences, as it loses the subjective component and retains only the objective elements that are not normally sufficient for the attribution of accountability. Moreover, although against the law, the action can be justified by the extenuating circumstances, that is, the legitimate use of arms (“...no punishment is impossible on the person who commits an act because they are obliged to do so in order to save themselves or others from a danger of serious bodily harm, if this danger was not voluntarily caused by themselves, nor otherwise avoidable, provided the action is proportional to the danger...”; art. 53 Italian Penal Code, “Legitimate use of weapons”). In the present case, the justification would be the compulsive force of the instinct of preservation when faced with a danger of serious, unavoidable, and involuntary bodily harm. The offense would thus be downgraded from murder to manslaughter, in other words, the abuse of a right by exceeding the limits established by law or by order of the commanding officer, or even the failure to make a correct assessment of the true situation and what was required to deal with it. In this case, the action or omission, despite being legally classified as a crime, is attributable to an error: a false interpretation of a situation affects the determination of the will and can induce a subject to commit actions that they would not have performed if they had not involuntarily made an error. If these scientific data are confirmed in other studies, they could introduce a case for extenuating circumstances, because selective temporary hearing loss during acute stress would be an accidental, accessory element causing the crime to be viewed in a different light and hence justifying a reduction of the sentence normally passed for the relative crime.

The other implication of our experiment seems to be on the operative context, especially in cases where the forces of order are involved in situations inducing intense emotional stress, when communication among the members of special forces and/or rescue teams is particularly vital. In fact, the use in operative contexts of suitable digital equipment able to produce a range of frequencies and gain (volume) that is better audible to the human ear could combat the onset of selective temporary hearing loss, thus safeguarding both the operators and the success of the operation itself.

In this sense, it should be noted that in the same period as our experiment was being conducted, at the “29th Shot Show,” the most important weapons fair, held in Dallas (U.S.A.) last February, an important American company producing weapon systems exhibited the “SWAT EARS” system, that consists of an ear-phone kit for use by operators potentially involved in shootings, that fosters better hearing of sounds that would otherwise be inaudible to

humans. In fact, the sophisticated circuit of SWAT EARS allows the immediate identification of sounds of danger like gunshots, as well as notably amplifying the range of spoken sounds. Unfortunately, we were unable to discuss the matter with this company during the course of our study.

Moreover, in the wake of the case at hand, in the future we may further investigate the different influence of stress on perceiving abilities in comparison with distraction stimuli (such as the use of cell phones during driving causing inattention blindness): both of them can produce sensorial dysperceptions, but with different brain mechanisms. In fact, when subjects switch between two (or more) competing tasks, there is a “reaction-time switching cost,” which is the time that the brain takes to switch its attention and focus from one task to another (16). Several studies demonstrated that there is only so much the brain can do at one time, even if the tasks draw on different areas and neural networks of the brain, because the brain has a capacity limit. This may help explain why talking on cell phones could affect what a driver, or a pedestrian, sees because these two tasks compete for our brain’s information processing resources (17,18). On the other hand, when someone undergoes stressful situations, real or perceived, the limbic system immediately responds, via autonomic nervous system and endocrine glands, causing metabolic, psychological, and behavioral changes, which can produce significant modifications at the sensorial and perceptive levels without significantly involving consciousness. We feel that delving deeper into these different mechanisms might benefit both the neuroscientific and forensic fields.

Another possible future development of our study could be an extension of the investigation to operative staff (special police and military forces), to identify and above all quantify the emotional stress parameters studied in this work, both during interventions in operative scenarios (taking care to safeguard both the operators and the operation) and in training scenarios deliberately set up for the purposes of testing their reaction capacities under stress. The biochemical parameters obtained in this study could be used as indices for training programs to control reactions to stress, as well as in later comparative studies with nonprofessional groups of subjects lacking specific training in this sense.

References

1. Cassano GB, Pancheri P, Pavan L, Ravizza L, Rossi R, Smeraldi E, et al., editors. *Trattato italiano di psichiatria*. Milan, Italy: Masson, 1999;313.
2. Mason J. Emotions as reflected in patterns of endocrine integration. In: Levi L, editor. *Emotions—their parameters and measurement*. New York, NY: Raven Press, 1975;143–81.
3. Lopez IA, Ishiyama G, Lee M, Baloh RW, Ishiyama A. Immunohistochemical localization of aquaporins in the human inner ear. *Cell Tissue Res* 2007;328:453–60.
4. Huang D, Chen P, Chen S, Nagura M, Lim DJ, Lin X. Expression patterns of aquaporins in the inner ear: evidence for concerted actions of multiple types of aquaporins to facilitate water transport in the cochlea. *Hear Res* 2002;165:85–95.
5. Beitz E, Zenner HP, Schultz JE. Aquaporin-mediated fluid regulation in the inner ear. *Cell Mol Neurobiol* 2003;23:315–29.
6. Blum LN. *Force under pressure: how cops live and why they die*. New York, NY: Lantern Books, 2000.
7. Solomon RM, Horn JM. Post-shooting traumatic reactions: a pilot study. In: Reese JT, Goldstein HA, editors. *Psychological services for law enforcement officers*. Washington, DC: US Department of Justice, 1986;383–93.
8. Kirshbaum C, Hellhammer DH. Salivary cortisol in psychobiological research: an overview. *Neuropsychobiology* 1989;22:150–69.
9. Hellhammer DH, Kirshbaum C, Belkien L. Measurement of salivary cortisol under psychological stimulation. In: Hingten JN, Hellhammer DH, Huppmann G, editors. *Advanced methods in psychobiology*. Toronto, Canada: Hogrefe, 1987;281–9.

10. Dantzer R. Stress and immunity: what have we learned from psychoneuroimmunology? *Acta Physiol Scand Suppl* 1997;640:43–6.
11. Aguilera G. Regulation of pituitary ACTH secretion during chronic stress. *Front Neuroendocrinol* 1994;15:321–50.
12. Aguilera G. Corticotropin releasing hormone, receptor regulation and the stress response. *Trends Endocrinol Metab* 1998;9:329–36.
13. Nishioka R, Takeda T, Kakigi A, Okada T, Takebayashi S, Taguchi D, et al. Expression of aquaporins and vasopressin type 2 receptor in the stria vascularis of the cochlea. *Hear Res* 2010;260:11–9.
14. Gu FM, Han HL, Zhang LS. Effects of vasopressin on gene expression in rat inner ear. *Hear Res* 2006;222:70–8.
15. LeDoux EJ. *The emotional brain: the mysterious underpinnings of emotional life*. New York, NY: Simon & Schuster/Touchstone, 1996.
16. Rogers RD, Monsell S. Costs of a predictable switch between simple cognitive tasks. *J Exp Psychol* 1995;124:207–31.
17. Just MA, Keller TA, Cynkar JA. A decrease in brain activation associated with driving when listening to someone speak. *Brain Res* 2008;1205:70–80.
18. Neider MB, McCarley JS, Crowell JA, Kaczmarek H, Kramer AF. Pedestrians, vehicles, and cell phones. *Accid Anal Prev* 2010;42:589–94.

Additional information and reprint requests:

Sara Sblano, M.D., Ph.D.

Via Giustino Fortunato

4 C/10

70125 Bari

Italy

E-mail: ssblano@gmail.com