FERTILITY AND STERILITY
Vol. 52, No. 6, December 1989

Printed on acid-free paper in U.S.A.

The effect of a specific emotional stressor on prolactin, cortisol, and testosterone concentrations in women varies with their trait anxiety

Koen Demyttenaere, M.D.*†
Piet Nijs, M.D.*
Gerry Evers-Kiebooms, Ph.D.‡
Philippe R. Koninckx, M.D., Ph.D.§

University Hospital Gasthuisberg, Leuven, Belgium

The psychological and the hormonal response to a specific emotional stressor (a video film on treatment of infertility, pregnancy, and delivery) was investigated in 30 women and the responses were correlated with their trait anxiety level. The experiment included a resting period before and after the stressor. The psychological response, i.e., the change in state anxiety, was in phase with the stressor and varied with the trait anxiety level. The endocrinological response, i.e., the time courses of prolactin, cortisol, and testosterone, was not in phase but varied with the trait anxiety level. It is suggested that psychological phenomena as anticipation, mental assimilation, and reflection could explain these findings, and that these should be taken into account when investigating the so-called "psychological" infertility. Fertil Steril 52:942, 1989

Emotional stress has been suggested as a cause of infertility, but the evidence is scanty. Psychosocial contributions described anxiety, depression, lowered self esteem, and social isolation in infertile women, without cause or effect suggestions.[1]

Psychoanalytical studies, based on case reports, suggested that conflicting attitudes toward feminine role and motherhood could cause subfertility. Psychotherapeutical contributions recently suggested a positive effect of psychotherapy on fertility.[2] Recently, we demonstrated that the probability of conception is lower in women with a high trait anxiety.[3]

Received November 14, 1988; revised and accepted August 2, 1989.

* Department of Obstetrics and Gynecology, Psychosomatic Unit.

† Reprints requests: Koen Demyttenaere, M.D., Department of Obstetrics and Gynecology, Psychosomatic Unit, University Hospital Gasthuisberg, Herestraat 49, B-3000 Leuven, Belgium.

‡ Department of Human Genetics.

§ Department of Obstetrics and Gynecology.

The mechanisms, however, by which stress could cause infertility remain unknown. Psychoendocrinological reactions involving prolactin (PRL), cortisol, and testosterone (T) secretion have been suggested. Moderate and severe hyperprolactinemia cause anovulation and amenorrhea. Recently, even temporarily elevated PRL levels (nocturnal, preovulatory, or early-follicular) were described to affect follicular maturation, ovulation, fertilization, and corpus luteum function.[4–6] The hypothalamo-pituitary-adrenal axis could affect fertility: a direct gonadal effect of corticoid hormones, a corticosteroid-mediated decrease in pituitary responsiveness to gonadotropin-releasing hormone (GnRH), and a centrally mediated inhibition of GnRH release was described.[7] The association of infertility and hyperandrogenism is also quite frequent in women.[8]

Hyperprolactinemia is associated with depression and anxiety. Bromocriptin lowers prolactin concentrations and improves depression.[9] The hypothalamo-pituitary-adrenal system is activated in stressful situations. Depression as well as anxiety

can be associated with an altered diurnal variation of cortisol secretion.[10] The interaction between stress and testosterone secretion is controversial.[11]

Emotional stress thus clearly influences the secretion of some hormones, and trait anxiety influences fertility. Because a group of women with "psychological" infertility cannot yet be identified, we investigated the correlation of trait anxiety with psychological and endocrine responses to a specific emotional stressor, i.e., a video on infertility, pregnancy, and delivery, in women attending the infertility clinic.

MATERIALS AND METHODS

Subjects

Women attending the infertility clinic of the Leuven University Hospital Gasthuisberg for artificial insemination (AID, n = 13) or for in vitro fertilization (IVF, n = 17) were invited to participate. The first 30 positive responders out of 90 who were invited actually participated.

All women had regular ovulatory cycles and were treated for primary infertility. The indication for IVF was male subfertility (n = 11), mechanical infertility (n = 4), or unexplained infertility (n = 2). In all women treated for male subfertility, no other obvious causes of infertility were found. The mean age of the women included was 29.7 ± 3.2 (±SD) years and the mean duration of their infertility was 4.2 ± 2.2 years.

The AID couples were found to have no psychological contraindication for AID, as evaluated by an experienced psychiatrist of the Psychosomatic Unit of the Department of Obstetrics and Gynecology during a 1 hour semi-structurized interview. Thus all couples had a stable partner relationship without a manifest psychiatric disease. The decision for AID was harmoniously taken with sufficient elaboration of the psychotrauma of male infertility.

Design

No drugs were taken for the last 72 hours. The psychoendocrinological experiment was performed in the early follicular phase (day 3.3 ± 1.2 of the menstrual cycle) and during the afternoon (between 2 and 6 PM) to prevent menstrual cycle or diurnal variations in hormone concentrations or psychometric characteristics.

Repetitive blood sampling (n = 14, numbered 0 to 13) was performed at 15 minute intervals using an indwelling catheter. Sampling was started half an hour after venipuncture.

A video on infertility, pregnancy, and delivery was used as a specific emotional stressor and was administered on an individual basis. The experiment consisted of 3 periods: a resting period (1 hour; 15 minutes), the specific emotional stressor (1 hour), and a second resting period (1 hour). During the resting periods, women were allowed to read magazines which were specifically checked for the absence of articles and pictures on pregnancy.

The psychometric testing was done during the 30 minutes between the placement of the catheter and the first blood collection. All tests were administered once, except the state anxiety. Because the state anxiety level fluctuates very quickly, it was measured three times, i.e., after the first resting period, after the video, and after the second resting period.

Psychometric Tests

To evaluate anxiety levels, personality, coping styles, and depression scales, the following four psychometric tests were used.

Spielberger State Trait Anxiety Inventory (STAI)

A Dutch translation of the STAI was used.[12] Trait anxiety is defined as the general tendency of an individual to be upset in stressful situations, or as the mean level of anxiety over a longer period. It is considered as a personality trait. The test-retest reliability ranges from 0.92 (after 1.5 hour) to 0.75 (after 118 days). State anxiety is defined as the momentarily experienced anxiety.

Amsterdamse Biografische Vragenlijst (ABV-B)

The ABV-B is an adaptation of the Eysenck Personality Inventory.[13] It is a 117-item scale and different aspects of the personality can be scored: neuroticism (N), neurosomatism (NS), extraversion (E), test attitude (T), social desirability (SW) and masculinity-feminity (MF).

The test-retest reliability in women after 3 weeks is 0.70 for N, 0.80 for NS, 0.83 for T, and 0.87 for E. The validity of these subscales has been extensively documented.[13]

Utrechtse Coping Vragenlijst (UCL)

The UCL is a Dutch adaptation of the Westbrook Coping Scale.[14] Coping mechanisms are con-

ceptualized as relatively stable personality traits, reflecting how a person copes with problems. The UCL consists of seven clusters of coping strategies: active coping, palliative reactions, avoiding reactions, social support seeking, depressive-regressive coping, expression of emotions or anger, and comforting ideas. The test-retest reliability in women ranges from 0.76 to 0.43 after 6 weeks.

Zung Depression Scale

A Dutch translation of the Zung Depression Scale was used.[15] When the index (= standardised transformation of the raw score) is equal or greater than 50, there is depressive disorder.

Radioimmunoassay Methods

Luteinizing hormone (LH) and PRL concentrations were assayed using the nonspecific LH assay and the PRL kits of Medgenix (Institute for radioactive elements, Fleurus, Belgium). Progesterone (P) and 17 B-estradiol (E_2) concentrations were assayed using the P and the E_2 kits of Biomerieux (Lyon, France). Follicle-stimulating hormone (FSH), T, and cortisol concentrations were assayed as described previously.[16]

Statistical Methods

Means ± standard deviation (SD) are listed unless indicated otherwise.

Kendall Tau rank correlation coefficients were used to investigate correlations between psychometric variables, and between psychometric variables and hormone concentrations.

Because the hormonal data have a non-Gaussian distribution, logarithmic transformation was performed before multivariate analysis of variance (manova) and one way analysis of variance (anova) were applicated using trait anxiety level as independent variable.

For the visual clarity of the figures, however, a dichotomy was made, showing the linear values of the group with lower than median trait anxiety (n = 15) and the group with higher than median trait anxiety (n = 15).

RESULTS

The psychometric test profiles are similar in AID and IVF women. Depressive coping, however, is more pronounced in IVF women and their state anxiety is higher after the video ($P < 0.05$).

Table 1 Psychometric Variables in the Investigated Women and the Kendall Tau Rank Correlation Coefficient With Trait Anxiety; for Comparison, the Means of the General Population Are Listed[a]

	General population	All women[b] (n = 30)	r	P
Trait anxiety	38	38.8 ± 8.0	—	—
Neuroticism (N)	50	61.8 ± 27.5	0.55	0.0000
Neurosomaticism (NS)	50	47.9 ± 23.4	0.08	NS
Extraversion (E)	50	55.3 ± 26.6	−0.11	NS
Test attitude (T)	50	46.7 ± 26.6	−0.02	NS
Masculinity femininity (MF)	50	51.9 ± 34	−0.32	0.01
Social desirability (SW)	50	50.2 ± 27	−0.27	0.03
Active coping	16 to 18	17.8 ± 2.8	0.03	NS
Palliative coping	13 to 18	16.9 ± 2.3	−0.06	NS
Avoiding	12 to 16	15.8 ± 3.8	0.32	0.02
Social support	10 to 14	14.2 ± 4.7	0.39	0.005
Depressive coping	10 to 12	12.1 ± 4.0	0.54	0.0001
Expression of emotion	6 to 8	6.9 ± 2.3	0.19	NS
Comforting ideas	11 to 14	12.8 ± 2.2	0.06	NS
Zung depression	<50	44.6 ± 8.1	0.17	NS

[a] NS, not significant.
[b] Values are means ± SD.

Trait anxiety is not related to the cause of infertility. The trait anxiety of the 24 women attending the clinic for male infertility is 38.4 ± 7.0, whereas the trait anxiety of the 6 other women is 40.3 ± 8.5. Trait anxiety is negatively correlated with age ($r = -0.28$; $P = 0.03$) and tends to be positively correlated with the duration of infertility ($r = 0.26$; $P = 0.06$).

The results of psychometric tests and their correlations are listed in Table 1. It can be noted that in the ABV-B, trait anxiety is positively correlated with neuroticism ($r = 0.55$; $P = 0.0000$), negatively with social desirability ($r = -0.27$; $P = 0.03$), and negatively with the masculinity-feminity score ($r = -0.32$; $P = 0.01$). In the UCL, trait anxiety is positively correlated with three coping mechanisms: seeking for social support ($r = 0.39$; $P = 0.005$), depressive coping ($r = 0.54$; $P = 0.0001$), and avoiding ($r = 0.32$; $P = 0.02$).

State Anxiety During the Experiment

Trait anxiety correlates with state anxiety during the whole experiment: i.e., after the first resting period ($r = 0.31$, $P = 0.01$), after the video ($r = 0.34$, $P = 0.009$), and after the second resting period (r

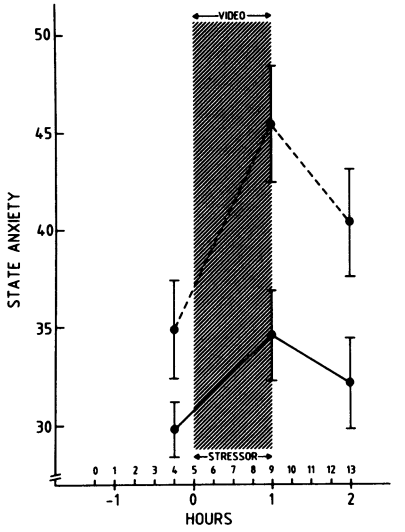

Figure 1 State anxiety (means ± SE) in higher (– – –, n = 15) and in lower (– – –, n = 15) than median trait anxious women.

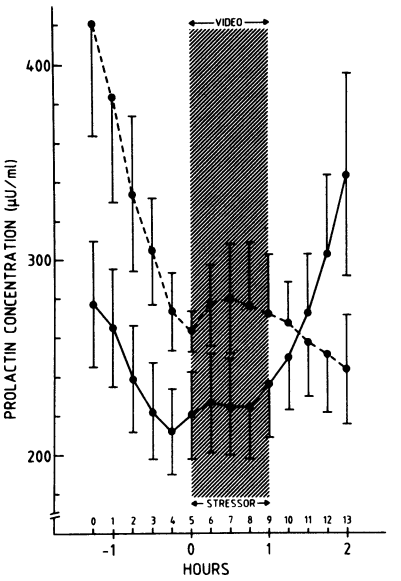

Figure 2 Prolactin concentrations (means ± SE) in higher (– – –, n = 15) and in lower than median trait anxious women (– – –, n = 15), before, during, and after a specific emotional stressor.

= 0.38, $P = 0.004$) (Fig. 1). The increment in state anxiety associated with the administration of the emotional stressor is positively correlated with trait anxiety ($r = 0.26$; $P = 0.04$).

Trait Anxiety and Hormonal Levels

The plasma concentrations of E_2 (61.7 ± 33.5 pg/mL), P (0.21 ± 0.26 ng/mL), LH (7.1 ± 3.2 mIu/mL), and FSH (5.9 ± 1.5 mIu/mL) confirm that all women were in the early follicular phase.

Trait Anxiety and PRL levels

The time course of PRL concentrations varies with the trait anxiety level (Fig. 2). During the first resting period, PRL concentrations decrease in all subjects. As soon as the emotional stressor is administered, the PRL time courses become different. While in higher trait anxious women, the stressor only results in a further decrease, the stressor results in a continuous increase in lower trait anxious women, and this evolution continues during the second resting period. These differences in hormonal profiles are significant. Trait anxiety correlates with the changes in PRL concentrations during the second resting period ($r = 0.39$; $P = 0.03$), and during the whole experiment ($r = 0.32$; $P = 0.01$). The effect of trait anxiety on the whole PRL time course during the first resting period and during the rest of the experiment is significant (Manova: Wilk's criterion: $F = 3.30$; $P = 0.01$).

The effect of trait anxiety on the PRL time course during the video and the second resting period is significant (Anova: $F = 7.52$; $P = 0.002$). Trait anxiety is not correlated with single PRL concentrations except at the very end of the experiment, where they are even negatively correlated (Prl 13: $r = -0.29$; $P = 0.02$).

Trait Anxiety and Cortisol Levels

The time course of cortisol concentrations varies with the trait anxiety level (Fig. 3). During the first

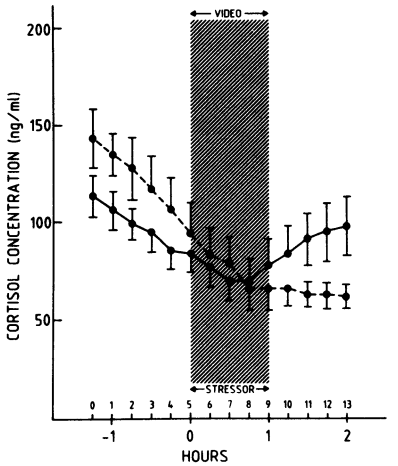

Figure 3 Cortisol concentrations (means ± SE) in higher (– – –, n = 15) and in lower than median trait anxious women (– – –, n = 15), before, during, and after a specific emotional stressor.

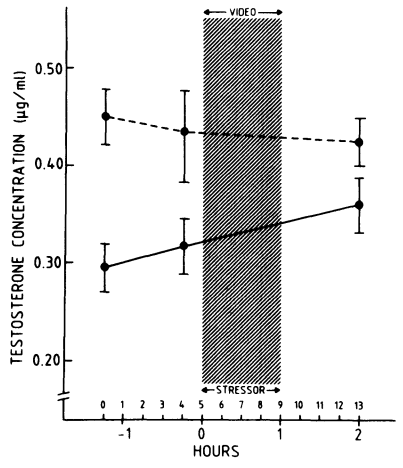

Figure 4 Testosterone concentrations (means ± SE) in higher (– – –, n = 15) and in lower than median trait anxious women (– – –, n = 15), before, during, and after a specific emotional stressor.

resting period, cortisol concentrations decrease in all subjects. As soon as the emotional stressor is administered, the cortisol time courses become different. Although in higher trait anxious women, the stressor only results in a further decrease, the stressor results in a continuous increase in lower trait anxious women, and this evolution continues during the second resting period. These differences in hormonal profile are significant. Trait anxiety correlates with the cortisol changes during the video ($r = 0.37$; $P = 0.004$) and during the whole experiment ($r = 0.37$; $P = 0.004$). The effect of trait anxiety on the cortisol time course during the first resting period and during the rest of the experiment is significant (Manova: Wilk's criterion: $F = 3.40$; $P = 0.01$). The effect of trait anxiety on the cortisol time course during the video and the second resting period is significant (Anova: $F = 5.11$; $P = 0.01$). Trait anxiety is not correlated with single cortisol concentrations.

Trait Anxiety and T Levels

The time course of T concentrations varies with the trait anxiety level (Fig. 4). In higher trait anxious women, T plasma levels remain stable during the whole experiment. In lower trait anxious women, however, T is lower at the beginning and gradually increases during the experiment. These differences in hormonal profile are significant. Trait anxiety correlates with T changes during the whole experiment ($r = 0.53$; $P = 0.0001$). The effect of trait anxiety on the T time course (during the first resting period and during the rest of the experiment) is significant (Manova: Wilk's criterion: $F = 3.28$; $P = 0.01$). Trait anxiety correlates with the T concentrations at the onset of the experiment ($r = 0.54$; $P = 0.0000$) and just before the video is shown ($r = 0.32$; $P = 0.02$).

DISCUSSION

The psychometric test results in the investigated group are comparable with those found in the general population.[12–15] The clinical belief that infertile women are more anxious, more neurotic and more depressed, is thus not confirmed. This conclusion should, however, be interpreted with care because of a possible preselection bias, i.e., it cannot be excluded that the nonparticipants (60/90) present different psychometric characteristics, and because controls were not included. Moreover, 24 women had probably a normal fertility because the investigation revealed only a male infertility. Because the number of women with mechanical infertility (n = 4) and with unexplained infertility (n = 2) is too small for meaningful conclusions, we cannot document the clinically well known fact that emotional distress during infertility is related to the individual woman's perception of who bears responsibility for the infertility. It is interesting, however, to notice that the psychometric test profile was similar in AID (n = 13) and IVF women (n = 17; 11 male subfertility).

The observation that trait anxiety is negatively correlated with age and tends to be positive with the duration of infertility should be interpreted carefully. One could speculate that the women with a higher trait anxiety have a longer duration of infertility because they remain longer in infertility programs.

On the contrary, one should not conclude that trait anxiety is increased by the infertility, since trait anxiety is a stable personality characteristic. Because trait anxiety is known to be not correlated with age, we suggest that our finding of a negative correlation between trait anxiety and age reflects a selection bias. We speculate that older women with high trait anxiety are less motivated to participate in experiments.

The increase in state anxiety during the video film demonstrated that the specific emotional stressor was effective. As could be expected, this increase is more pronounced ($P = 0.04$) in women with higher trait anxiety. Looking at a video on infertility, pregnancy, and delivery was, at least in

higher trait anxious women, experienced as extremely stressful because their state anxiety increased up to decile 8.[12]

Although the emotional changes were in phase with the administered stressor, i.e., low before, high at the end of the video and lower again at the end of the experiment, the endocrinological changes were clearly out of phase. We suggest that all women had an anticipatory PRL and cortisol peak before the experiment and that this anticipation is more explicit in higher trait anxious women.

It cannot be excluded, however, that the venipuncture (placement of permanent catheter) or the psychometric testing itself were experienced as more traumatic or more stressful in higher trait anxious women. Unfortunately, a state anxiety measurement immediately after the venipuncture was not performed. Once the video is shown, the evolution of PRL and cortisol concentrations is different depending on the trait anxiety level. In higher trait anxious women, the conscious stress of the video does not increase in PRL or cortisol secretion. In lower trait anxious women, the video increases both PRL and cortisol concentrations. For PRL, the increase is most important after the video has been shown, i.e., during the second resting period, whereas for cortisol, the increase is most important during the video. The evolution of both hormones is thus similar but their time courses are slightly out of phase. This suggests a hypothalamic or even suprahypothalamic mechanism. The evolution of T concentrations is different from those of PRL and cortisol. Because only three samples per person were assayed because of the lack of plasma, the evolution of testosterone concentrations during the experiment cannot be described exactly. Our data, however, clearly demonstrate that in women with a lower trait anxiety, the initial testosterone concentration is lower, whereas the concentration increases more during the experiment. We can only speculate about the mechanism because the literature on stress and testosterone concentrations is confused.[11] Also in the adrenal, a dissociation between cortisol and androgen levels in stressful situations was recently described.[17]

To explain that the endocrinological response was out of phase, we suggest that the higher trait anxious women respond endocrinologically mainly with anticipatory release, whereas lower trait anxious women respond mainly during and after the stressor, i.e., during mental assimilation and reflection. Thus psychological phenomena as anticipation, reflection, and mental assimilation could modulate hormone secretion during stress. Therefore, a clearcut difference should be made between psychological and pharmacological (e.g., thyroid-releasing hormone, metoclopramid, sulpiride, . . . for PRL) stressors. Although pharmacological stressors interact directly and brutally with the hypothalmo-pituitary axis, psychological stressors modulate physiologically through higher brain centers. This means that psychological stressors are more subtle, involving mechanisms as anticipation, mental assimilation, and reflection. This is important, because it is a main characteristic of anxious women to react with strong anticipation, whereas healthy women react by reflection and mental assimilation.

From our data it is evident that the evolution of hormones should be studied over longer periods to evaluate psycho-endocrinological interactions, and that serial blood sampling with a prolonged baseline is necessary. Moreover, personality characteristics (e.g., trait anxiety) should be taken into account. Although a specific psychological stressor indeed provokes different reactions in higher or lower trait anxious women, it remains unknown whether this also holds through for pharmacological stressors.

Our findings shed new light on conflicting data in the literature. PRL concentrations after masturbation increased in some persons and decreased in others.[18] It was described that prolonged stress induced a reversal of an initially elevated plasma level of PRL and cortisol in some subjects.[19] Although embryo transfer is considered as stressful, PRL concentrations do not change after embryo transfer.[20]

In contrast with the strong psychological impact of the video, i.e., decile 8 for the high-trait anxious women, the changes in hormone concentrations are much less pronounced and remain within the normal range. Because we demonstrated that the probability of conception in an AID program is significantly lower in women with a high-trait anxiety, it cannot be excluded that these minor changes in hormone concentrations interfere with fertility.[3] Indeed, one should realize that these minor changes occur repetitively and frequently because high-anxious women are psychologically overalert. Some infertile women indeed live in a social isolation by the fact that they anxiously avoid other pregnant women or visits in a maternity unit, always in anticipation of a confrontation with their infertility. Moreover, the literature provides argu-

ments that even subtle hormone changes affect fertility. Mean PRL levels are significantly lower in conception than in nonconception cycles.[4] An impaired luteal function can be the consequence of elevated PRL levels at night time only.[21] Metoclopramid induced hyperprolactinemia in the early follicular phase impairs the development of the dominant follicle and in women with a LUF syndrome, only minor differences in plasma hormone levels were described.[22] Women with a LUF syndrome have a higher trait anxiety level than normal controls.[23] Daytime noise stress increases nocturnal PRL levels.[24] Cigarette smoking, which can be a stress-linked behavior, is in men, associated with higher PRL levels.[25]

In conclusion, we demonstrated that the changes in PRL, cortisol, and T concentrations provoked by a specific emotional stressor vary with their trait anxiety. We suggest that typical psychological phenomena as anticipation, mental assimilation, and reflection can explain the differences between hormonal and psychological responses, and that personality characteristics should be taken into account when psychological stressors are investigated. We speculate that personality characteristics determine how women experience and react to the stress of infertility, and that these psychological and endocrinological differences could influence their probability of conception.

REFERENCES

1. Stauber M: Psychosomatische Untersuchungen zur sterilen Partnerschaft. Gynäkologe 15:202, 1982
2. Sarrel PM, DeCherney AH: Psychotherapeutic intervention for treatment of couples with secondary infertility. Fertil Steril 43:897, 1985
3. Demyttenaere K, Nijs P, Koninckx P, Steeno O, Evers-Kiebooms G: Anxiety and conception rates in donor insemination couples. J Psychosom Obstet Gynecol 8:175, 1988
4. Board JA, Storlazzi E, Schneider V: Nocturnal prolactin levels in infertility. Fertil Steril 36:720, 1981
5. Ben-David J, Schenker JG: Transient hyperprolactinemia: a correctable cause of idiopathic female infertility. J Clin Endocrinol Metab 57:442, 1983
6. Ylikorkala O, Kauppila A: The effects on the ovulatory cycle of metoclopromid-induced increased prolactin levels during follicular development. Fertil Steril 35:588, 1981
7. Rivier C, Rivier J, Vale W: Stress-induced inhibition of reproductive functions: role of endogenous Corticotropin-Releasing Factor. Science: 607, 1986
8. Steinberger F, Smith K, Rodriguez-Rigau LJ: Hyperandrogenism and female infertility. In Endocrinology of human infertility: new aspects, Edited by PG Crosignani, BL Rubia. London, Academic Press, 1981, p 327
9. Buckman MT, Kellner R: Reduction of distress in hyperprolactinemia with bromocriptin. Am J Psychiatry 142:242, 1985
10. Mendlewicz J, Charles G, Franckson JM: The dexamethasone suppression test in affective disorder: relationship to clinical and genetic subgroups. Br J Psychiat 141:464, 1982
11. Hellhammer DH, Hubert W, Schürmeyer T: Changes in saliva testosterone after psychological stimulation in men. Psychoneuroendocrinology 10:77, 1985
12. Vanderploeg HM, Defares PB, Spielberger CD: Handleiding bij de Zelfbeoordelingsvragenlijst: een Nederlandstalige Bewerking van de Spielberger State Trait Anxiety Inventory. Swets and Zeitlinger, Lisse, 1980 (Manual)
13. Wilde GJS: Amsterdamse Biografische Vragenlijst. Van de Rossen, Amsterdam, 1970
14. Schreurs PJG, Van de Willige G, Tellegen B, Brosschot JF: De Utrechtse Coping-lijst UCL. Tijdschrift voor Psychologie 12:101, 1984
15. Zung WWK: A self-rating depression scale. Arch Gen Psychiatry 12:63, 1965
16. Koninckx PR, Heyns W, Verhoeven G, Van Baelen H, Lissens W, De Moor P, Brosens I: Biochemical characterisation of peritoneal fluid in women during the menstrual cycle. J Clin Endocr Metabol 51:1239, 1980
17. Parker L, Eugene J, Farber D, Lifrak E, Lai M, Juler G: Dissociation of adrenal androgen and cortisol levels in acute stress. Horm Metab Res 17:209, 1985
18. Steeno O, Coucke W, Koninckx Ph: Prolaktin und Stress durch Masturbation. Andrologia 11:466, 1979
19. Johansson GG, Karonen SL, Laakso ML: Reversal of an elevated plasma level of prolactin during prolonged psychological stress. Acta Physiol Scand 119:463, 1983
20. Boyers SP, Lavy G, Russell JB, Polan ML, DeCherney AH: Serum prolactin response to embryo transfer during human in vitro fertilisation and embryo transfer. J In Vitro Fert Embryo Transfer 4:269, 1987
21. Lenton EA, Brook L, Sobowale OS, Cooke ID: Prolactin concentrations in normal menstrual cycles and conception cycles. Clin Endocrinol 10:383, 1979
22. Koninckx PR, Brosens IA: Clinical significance of the luteinized unruptured follicle syndrome as a cause of infertility. Europ J Obstet Gynec Reprod Biol 13:355, 1982
23. Nijs P, Koninckx PR, Verstraeten D, Mullens A, Nicazy H: Psychological factors of female infertility. Eur J Obstet Gynecol Reprod Biol 18:375, 1984
24. Fruhstorfer B, Fruhstorfer H, Grass P, Milerski HG: Daytime noise stress and subsequent night sleep: interference with sleep pattern, endocrine and neurocrine functions. Intern J Neuroscience 26:301, 1985
25. Hauger Klevene JH, Balossi EC: Prolactin: a link between smoking and decreased fertility? (Letter) Fertil Steril 46: 531, 1986