Eur Arch Psychiatr Neurol Sci (1987) 236:312–318

European Archives of Psychiatry and Neurological Sciences

Changes in Serum Prolactin after Electroconvulsive and Epileptic Seizures

Folke Johansson[1] **and Lars von Knorring**[2]

[1]Department of Neurology, [2]Department of Psychiatry, Umeå University, S-90185 Umeå, Sweden

Summary. Serial serum prolactin (PRL) concentrations were measured after epileptic seizures and seizures following electroconvulsive therapy (ECT). There was a large and rapid rise in PRL after ECT seizures but a much more variable PRL response after spontaneous seizures. Only epileptic seizures of longer duration and of grand mal character resulted in a more substantial rise in PRL. In ECT seizures there was a significant positive correlation between the duration of seizures and the rise in postictal PRL. The postictal PRL curves over 24 h were similar in both spontaneous seizures and ECT seizures. Interictally there were no differences in PRL levels between epileptic patients compared to patients with other neurological diseases or healthy volunteers. Chronic treatment with drugs affecting dopamine transmission had a profound effect on PRL secretion, and a dose-dependent significant increase in PRL with neuroleptics was observed.

Key words: Prolactin – ECT – Epilepsy – Neuroleptics – L-Dopa

Introduction

Several reports have indicated that certain neuroendocrine or muscular markers can be used to differentiate between epileptic and psychogenic seizures (Abbot et al. 1980; Collins et al. 1983; Trimble 1978; Wyllie et al. 1985). The most consistent results have been found with prolactin (PRL) and a rise in postictal PRL levels has been shown both in animal experiments and in humans with spontaneous or electroconvulsive epileptic attacks (Dana-Haeri et al. 1983; Pritchard III et al. 1983; Öhman et al. 1976; Apéria et al. 1985). However, whether the elevated PRL secretion is specifically related to the epileptic discharge or part of a more generalised metabolic or ischemic event is not known. The clinical use of PRL as an "epileptic marker" is also limited by the fact that certain partial attacks or "minor" convulsions do not seem to result in any conspicuous rise in PRL (Collins et al. 1983; Pritchard III et al. 1983; Laxer et al. 1985).

The aim of the present study was to elucidate the consistency and time course of the PRL increase after spontaneous and electroconvulsive seizures. In order to investigate baseline conditions and serum PRL variability, healthy volunteers and psychiatric and neurological patients were included. The serum PRL dependency on dopamine transmission was studied by inclusion of patients treated with L-dopa or neuroleptics. Patients receiving electroconvulsive treatment (ECT) were used as a model for well-controlled seizures.

Offprint requests to: L. von Knorring

Methodology

Patient series

Inpatients from the neurological and psychiatric wards were studied for PRL secretion postictally or under baseline conditions.

Neurological patients. (a) Patients with known epilepsy with seizures during the investigation period ($n = 28$, 8 males and 20 females, age range 17–57 years, mean 29.6 ± 3.7 years). Patients in this group were in hospital because of poor seizure control or for evaluation of the cause of their epilepsy. They all had focal or general interictal epileptic EEG activity and were all taking anticonvulsant medication, either as monotherapy or as a combination of several drugs.

(b) Patients with known epilepsy, investigated interictally ($n = 12$, 5 males and 7 females, age range 17–68 years, mean 30.7 ± 3.9 years). None of these patients had any seizures during the period of PRL sampling. Some of them were also included in (a) and thus served as their own controls for PRL. They were receiving therapy with antiepileptics and were inpatients for the same reasons as those in (a).

(c) Patients with Parkinson's disease ($n = 5$, 2 males and 3 females, age range 42–69 years, mean 59.0 ± 3.8 years). They were all receiving L-dopa in therapeutic doses (more than 400 mg) and two patients also received bromocriptin (less than 15 mg).

(d) Patients with other neurological diseases ($n = 9$, 4 males and 5 females, age range 43–67 years, mean 51.0 ± 3.6 years). They were receiving treatment with various drugs but none was taking L-dopa or antiepileptics.

Psychiatric patients. (a) Patients with chronic schizophrenic syndromes ($n = 15$, 10 males and 5 females, age range 31–80 years, mean 53.5 ± 3.9 years). They had all been on regular neuroleptic treatment for at least 3 years, usually for much longer.

(b) Depressed patients hospitalized during a depressive episode and treated with ECT ($n = 12$, 5 males and 7 females, age range 24–56 years, mean 41.7 ± 3.3 years). In this group PRL was taken immediately before and until 24 h after ECT.

(c) Depressed patients treated with repeated ECTs ($n = 16$, 6 males and 10 females, range 26–65 years, mean

58.8 ± 4.0 years). Here PRL was taken before and after each consecutive ECT.

Healthy Volunteers. Healthy members of the neurological staff ($n = 37$, 13 males and 24 females, age range 19–63 years, mean 36.9 ± 3.2 years) were included for comparison of PRL secretion with other groups. Pregnant or lactating women were excluded.

Serum PRL

Blood samples for PRL determinations were taken at predetermined times before and after ECT seizures and as soon as possible after spontaneous epileptic seizures (the first sample usually within 15–20 min after the seizure). Samples for baseline PRL concentrations were taken at 8 a.m., 2 p.m., 8 p.m., and 2 a.m. Blood was withdrawn from an antecubital vein catheter and stored at −20°C until analysis by a radioimmunoassay procedure using commerciably available PRL and PRL antibody preparations (Kabi AB and Axel Johnson Instrument AB, Sweden). The results are given in micrograms per liter. Due to the limitations of the assay, values below 2 µg/l were given the value of 2 (these extremely low values were found only in patients treated with L-dopa).

ECT Procedure

ECT was given in the usual manner with methylscopolamine as premedication and during Brietal Sodium-suxamethonium chloride anesthesia. Respiration was supported by pure oxygen from a Ruben's bag. Convulsions were induced by electric stimulation (usually 3–7 s through unilaterally positioned electrodes on the nondominant hemisphere. The strength of the convulsions and the seizure time was recorded for each individual. ECT was given in the morning and patients were fasted prior to each treatment. The methodology has been described in detail elsewhere (d'Elia 1970).

Epileptic Seizures

Spontaneous or induced seizures were recorded immediately after the event. The staff were instructed to note the characteristics of each observed or recalled seizure and the duration of the attack. The patients were divided into three groups.

Spontaneous seizures. (a) Patients with seizures of long duration (more than 30 s). All primary or secondary generalized seizures were included in this group and also a few complex partial seizures of longer duration.

(b) Patients with seizures of short duration (less than 30 s). A heterogenous group with absences, myoclonic or tonic minor convulsions, and most of the partial seizures (simple or complex).

ECT seizures. The duration and strength of the tonic-clonic convulsions were recorded. All seizures had a uniform generalized tonic-clonic (grand mal) character.

Neuroleptic medication

The 15 psychotic patients investigated had all been hospitalized for several years and were all kept on stable neuroleptic medication, often with a combination of several drugs. The pharmacological treatment had not been changed in the previous 6 months. The doses of the various neuroleptic drugs were transformed to corresponding doses of chlorpromazine according to the equipotency table suggested by Davis (1976); i.e., 100 mg of chlorpromazine daily is equipotent to 100 mg thioridazine or chlorprotixene, 19 mg acetophenazine, 15 mg prochlorpherazine, 10 mg perphenazine, 3 mg thio tixene, 5 mg trifluoperazine, 2 mg haloperidol, and 2 mg fluphenazine. Thus, the total daily dose of neuroleptics expressed in chlorpromazine equivalents, was 110–6250 mg/day (999.0 ± 385.6 mg/day).

Statistics

Differences between means were calculated by means of the Student's t-test and relationships were sought by means of the linear correlation coefficient. The χ^2 test was used to test the significance of differences in frequency distributions.

Results

Serum PRL in Healthy Volunteers

In 34 healthy subjects, PRL concentrations at 8 a.m. varied from 2 to 48 µg/l. In the males ($n = 12$) the values varied from 3 to 20 µg/l, while the variation in the females was even larger, 2–48 µg/l (Fig. 1a). There were a few abnormally high values in normals, especially at 8 a.m. (Fig. 1a). However, they all had normal concentrations at other times of the day and they had normal values when retested at the same time another day. There was a tendency towards higher PRL values in the females (at 8 a.m., females $n = 24$, 12.8 ± 11.8 µg/l, males $n = 12$, 9.6 ± 5.0 µg/l, $t = 0.90$ N.S., at 2 p.m., females 7.6 ± 4.9 µg/l, males 5.5 ± 4.0 µg/l, $t = 1.32$ N.S.). In the group of healthy volunteers, the sex difference did not reach statistical significance, but in the total sample, the females had significantly higher serum PRL values (at 8 a.m., females $n =$ 44, 15.0 ± 16.6 µg/l, males $n = 34$, 8.6 ± 5.8 µg/l, $t = 2.13$ $P <$ 0.05).

There was a clear tendency towards a circadian variation in baseline serum PRL secretion, both in normal subjects and in the different patient groups, except for Parkinson patients on L-dopa (Fig. 2). The lowest values were found at 2 p.m. while higher values were found between 8 p.m. and 8 a.m.

If the mean ± 2 SD is regarded as the normal range, the normal variation for PRL could be regarded as 0–19.7 µg/l for the males and 0–36.4 µg/l for the females at 8 a.m. At 2 p.m. the range was smaller (Fig. 2) and the normal variation for males was 0–13.5 µg/l and for females 0–17.4 µg/l.

Serum PRL in Clinical Groups

The values for PRL at 8 a.m. are presented in Fig. 1a. In Fig. 1b the PRL values are expressed in SD above or below the mean for the healthy volunteers. The normal values used were: males $n = 12$, 9.6 ± 5.0 µg/l, females $n = 22$, 12.8 ± 11.8 µg/l.

As shown in Fig. 1b, patients on neuroleptic drugs had significantly higher serum PRL values than the healthy volunteers (68% of the patients receiving neuroleptics had serum PRL values above +0.75 SD as compared to 14.7% of the healthy volunteers, $\chi^2 = 17.50$, $P < 0.001$). Also the small group of patients receiving L-dopa differed significantly from the controls (80% had PRL values below −0.75 SD as compared to 14.7% of the healthy subjects, $\chi^2 = 7.11$, $P < 0.01$).

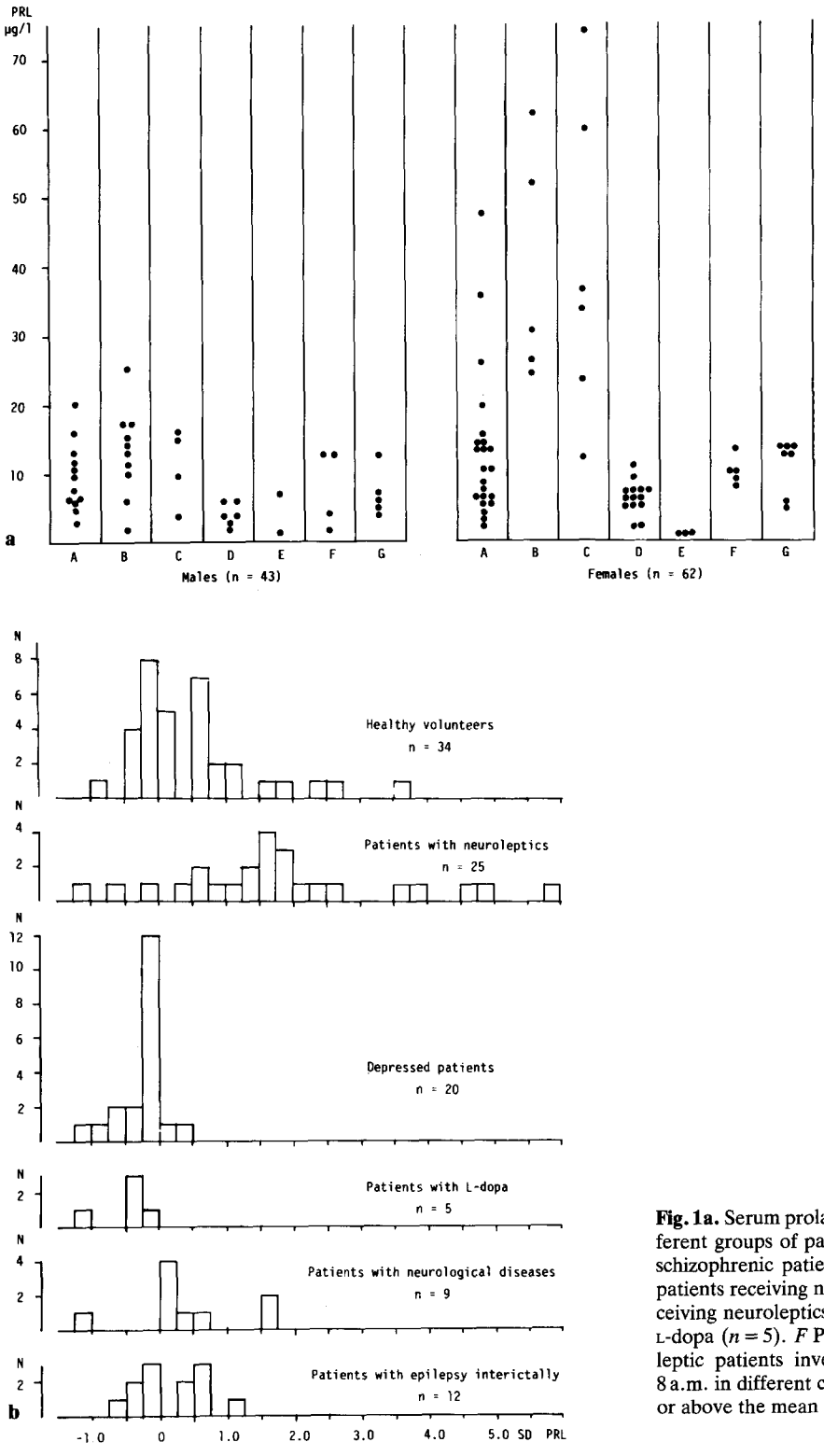

Fig. 1a. Serum prolactin (PRL) at 8 a.m. in healthy volunteers and different groups of patients. *A* Healthy volunteers ($n = 34$). *B* Chronic schizophrenic patients receiving neuroleptics ($n = 15$). *C* Depressed patients receiving neuroleptics ($n = 10$). *D* Depressed patients not receiving neuroleptics ($n = 20$). *E* Patients with Parkinson's disease on L-dopa ($n = 5$). *F* Patients with neurological diseases ($n = 9$). *G* Epileptic patients investigated interictally ($n = 12$). **b** Serum PRL at 8 a.m. in different clinical groups. PRL values expressed as SD below or above the mean of the healthy volunteers

In the patients receiving neuroleptic treatment, the increased serum PRL values was related to the drug and not to the diagnosis of the patient. Patients with chronic schizophrenic disorders and depressed patients receiving neuroleptics had increased PRL values (Fig. 1a). Furthermore, in the chronic schizophrenics, the correlation between the neuroleptic dose (expressed in chlorpromazine equivalents) and the serum PRL values was 0.86, $P < 0.01$. As a wide range of neuroleptic doses was involved and as the distribution of neuroleptic doses was not normal, the dose of neuroleptics was also expressed in log chlorpromazine equivalents (Fig. 3). However, the correlation was still 0.82, $P < 0.01$.

Thus, it seems clear that dopamine blockade results in a dose-dependent increase in serum PRL diurnal secretion, whereas dopamine agonistic drugs decrease the values far below the normal values and with a flat diurnal curve (Fig. 2).

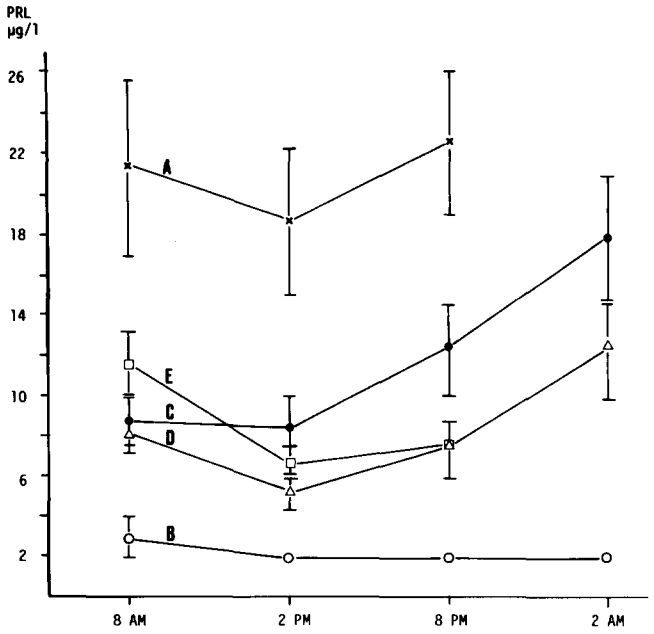

Fig. 2. Circadian curves of serum PRL in psychic and neurological diseases and healthy volunteers ($\bar{x} \pm$ SE). Patients with: *A* Schizophrenic syndromes on neuroleptic treatment *B* Parkinson's disease on L-dopa *C* Neurological diseases *D* Epilepsy, investigated interictally *E* Healthy volunteers

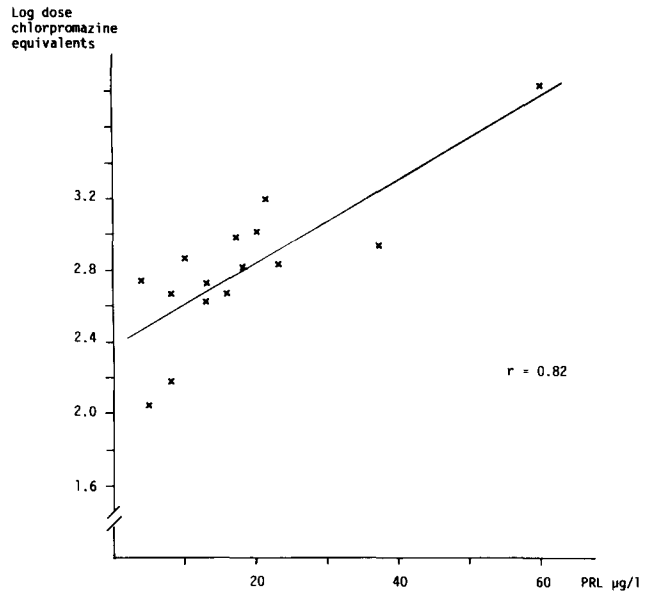

Fig. 3. Relationship between daily dose of neuroleptics (expressed in chlorpromazine equivalents according to the equipotency table by Davis 1976, log scale) and serum PRL levels at 2 p.m. in 15 chronic schizophrenic patients

The patients with depressive disorders, investigated before ECT had somewhat lower serum PRL values compared to the healthy volunteers (90% had values below −0.5 SD as compared to 38.2% of the healthy volunteers, $\chi^2_y = 11.76$, $P < 0.001$, Fig. 1b).

The patients with neurological diseases and the epileptic patients investigated interictally tended to have normal PRL values (Fig. 1b and Fig. 2, at 8 a.m. patients with neurological diseases, $n = 9$, 8.7 ± 3.3 µg/l, healthy volunteers $n = 37$, 11.7 ± 9.7 µg/l, $t = 1.06$ N.S., patients with epilepsy interictally, $n = 12$, 8.2 ± 2.8 µg/l, healthy volunteers, $n = 37$, 11.7 ± 9.7 µg/l, $t = 1.22$, N.S.).

Serum PRL after ECT Seizures

In the present study, ECT has been used as a "model" for epileptic seizures. Baseline values were collected 30 and 15 min before ECT and then samples for PRL were taken at 15, 30, and 60 min after ECT and later at 2, 4, 6, 12, and 24 h postictally. At 15 min after ECT there was a significant rise in serum PRL ($t = 12.51$, $P < 0.001$) and 30 min after the increase was still significant ($t = 9.11$, $P < 0.001$). After 60 min there was greater variability and the increase in PRL was no longer significant ($t = 1.35$, N.S.). At 2, 4, 6, 12, and 24 h after ECT, the serum PRL values had returned to normal levels (Fig. 4).

In one series of patients ($n = 16$), PRL was analyzed during the first 6 ECTs. The maximum serum PRL increase 15 min after each ECT was recorded. For all 6 consecutive ECTs, the rise in serum PRL was significant (ECT-1; $+27.2 \pm 19.2$ µg/l, $t = 5.66$, $P < 0.001$, ECT-2; $+28.1 \pm 33.8$, $t = 3.32$, $P < 0.01$, ECT-3; $+20.3 \pm 14.3$, $t = 5.66$, $P < 0.001$, ECT-4; $+19.4 \pm 14.0$, $t = 5.54$, $P < 0.001$, ECT-5; $+21.4 \pm 20.9$, $t = 4.10$, $P < 0.01$, ECT-6; $+20.5 \pm 17.6$, $t = 4.67$, $P < 0.001$). There was a small tendency towards a greater increase in PRL after ECT-1 and ECT-2 than during the following ECTs, but the difference between the increase in PRL after ECT-1 and the increase in PRL after ECT-6 was not significant ($t = 1.31$, N.S.). In a series of 32 patients recorded at the maximum increase 15 min after ECT the increase in serum PRL was significant both in females and males (females $n = 22$, $+26.7 \pm 31.1$, $t = 4.03$, $P < 0.001$, males $n = 10$, $+14.1 \pm 11.7$, $t = 3.80$, $P < 0.02$). There was no significant difference between the increase in PRL after ECT in females and males ($t = 1.26$, N.S.). The increase in serum PRL after ECT-1 did not correlate with age, ($r = -0.10$ N.S.), serum PRL before ECT ($r = 0.03$, N.S.), stimulation time ($r = -0.09$, N.S.), or treatment response (patients with favorable response after ECT 3–8, $n = 22$, $+25.5 \pm 19.8$, patients with late or poor response after ECT 9–13, $n = 10$, $+16.8 \pm 10.4$, difference between groups, $t = 1.23$, N.S.). However, the maximum increase in serum PRL 15 min after ECT correlated significantly with the seizure time ($r = 0.37$, $P < 0.05$). Thus, a longer seizure, regardless of the stimulation time, resulted in an increased response in serum PRL. The fact that the increase in serum PRL was related to epileptic seizure and not to anesthesia or some nonspecific stress factors is shown in Fig. 5. For technical reasons this patient had no ECT but was otherwise prepared with the same anesthetic and muscle relaxant procedure. No alterations were recorded in serum PRL levels.

Serum PRL after Spontaneous Seizures

In patients with spontaneous seizures there was a rapid rise in PRL levels within 30 min postictally with decreasing concentrations during the next 30 min (Fig. 6 and Fig. 7). When all types of epileptic attacks were looked at together, the PRL levels were widely dispersed, both within the normal and abnormal ranges (Fig. 6). When the seizures were separated according to the duration of the attacks, the difference was more obvious (Fig. 6 and Fig. 7). For the seizures of long duration (more than 30 s), all PRL values but one were in the abnormal high range. In this group there were mostly generalized tonic-

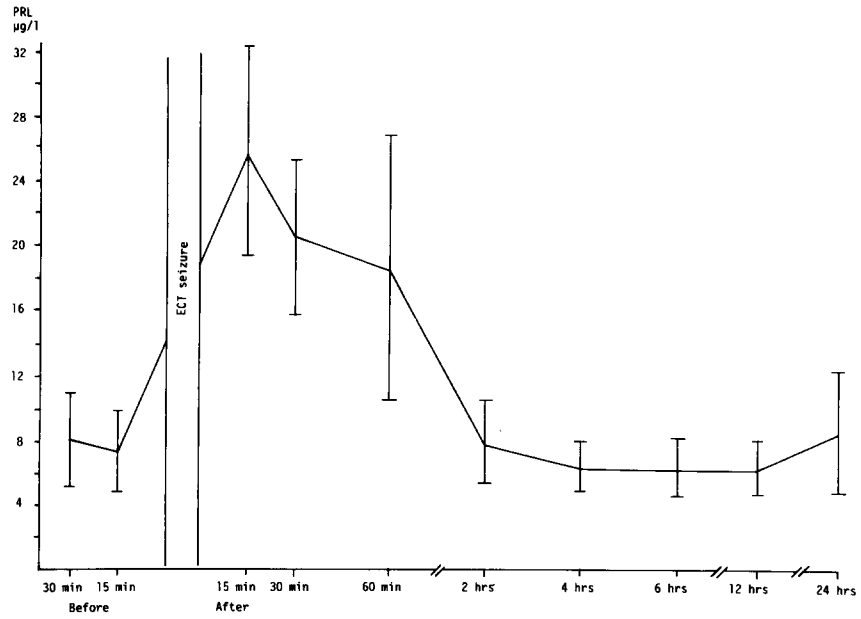

Fig. 4. Serum PRL before and up to 24 h after electroconvulsive therapy (ECT) ($\bar{x} \pm$ SE). Depressed patients ($n = 12$)

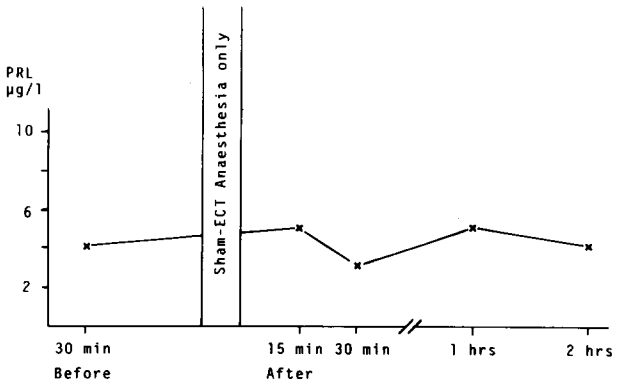

Fig. 5. Serum PRL in sham-ECT (male 31 years)

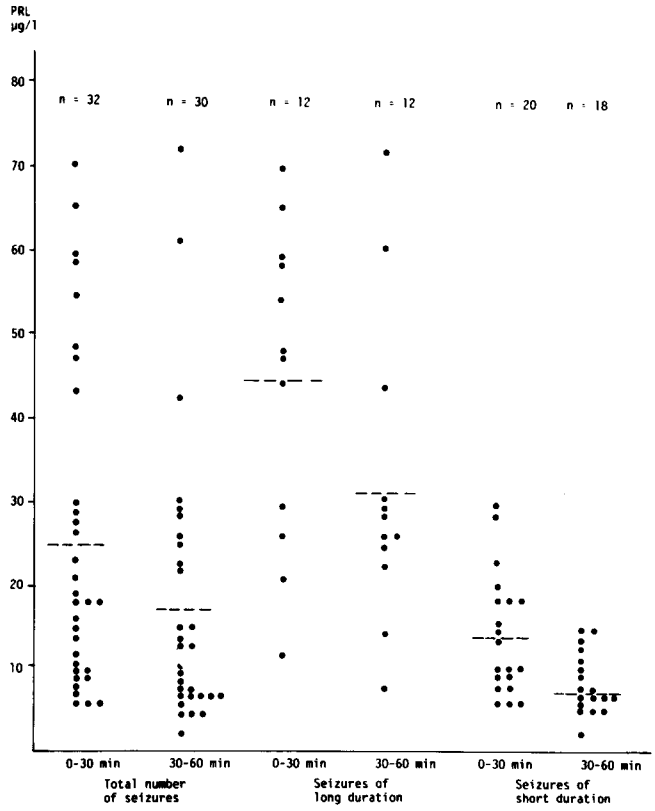

Fig. 6. Postictal levels of serum PRL measured at 0–30 and 30–60 min after epileptic seizures for total number of seizures and for seizures of long (above 30 s) and short (below 30 s) duration

clonic seizures and all patients had a disturbance of consciousness. In the group with short lasting seizures (less than 30 s) the result was not so clear. In this group there were more mixed types of seizures and it was not possible to record more accurately the presence or degree of altered consciousness for each patient. The PRL concentrations were not so variable in this group and most values were in the normal range (Fig. 6). Seizures of long duration had a much slower decrease in PRL levels, which were not normalized until about 2 h postictally (Fig. 7). Once normalized the postictal PRL values were stable up to 24 h. After 2 h, the postictal PRL curves were similar for spontaneous and ECT seizures. A few patients had several types of seizures at different times and it was possible to obtain PRL samples after each separate attack. It was found that the postictal PRL had the same pattern as in general, i.e., generalized and long seizures resulted in high PRL values compared to a more modest or no rise in attacks of short duration, in samples taken from the same patient. It was not possible to calculate a more precise relationship between the duration of the spontanous seizures and the PRL levels but such a positive correlation was found for ECT seizures as stated earlier.

There were only three patients who were strongly suspected of having psychogenic seizures. They were all taking antiepileptics for presumed epilepsy and two of them had focal epileptic EEG discharges and positive CT scans. All patients had long attacks of a typical generalized character and in two patients there were repeated attacks at different times or as a "convulsive status". Most PRL values were normal

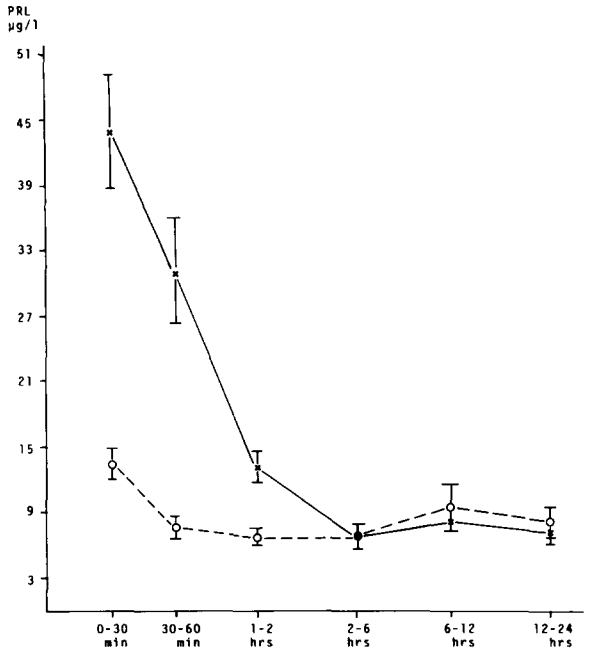

Fig. 7. Postictal levels of serum PRL in epileptic seizures with long (above 30 s) and short (below 30 s) duration. ($\bar{x} \pm$ SE) (×——×) seizures of long duration, (O– – –O) seizures of short duration

postictally, but in one patient there was a clear rise in PRL after two separate attacks but clinically it was not possible to record any features distinguishing these attacks from the many other seizures with normal postictal PRL curves for this patient.

Three patients (females with primary generalized epilepsy) were investigated with an EEG tape recording over 15 to 24 h and PRL samples were taken regularly and after EEG epileptic discharges. The PRL concentrations were quite normal and stable in all patients. The pattern was the same in the patient with no pathological EEG activity as in the other two with frequent bursts of generalized epileptic discharge up to 7 s and with no or doubtful clinical epileptic manifestations.

Discussion

The results of this study confirm the findings of others (Cavallo et al. 1984; Pritchard III et al. 1983) that patients with epilepsy have normal interictal PRL levels and the same circadian pattern as other neurological patients and normal subjects. A nocturnal rise in PRL was shown in a study by Molaie et al. (1985a and b) for primary generalized epilepsy and partial complex epilepsy. However, in a study of sexual dysfunction in temporal lobe epilepsy (Pritchard III et al. 1983) a consistent interictal hyperprolactinemia was not found.

There is no clear indication that various antiepileptic drugs or frequent bursts of epileptiform EEG activity have any influence on interictal PRL secretion, as shown in this study and also by others (Aminoff et al. 1986; Cavallo et al. 1984). Studies on the influence of interictal epileptiform discharges on PRL secretion have shown different results. Molaie et al. (1986) found that there is a significant increase in PRL levels among male patients with complex partial seizures compared to healthy controls. This increase was seen during non-REM sleep and during the awake stages at night. There was no increase during REM sleep periods when the interictal epileptiform activity was significantly reduced. It is obvious that chronic treatment with drugs affecting dopamine transmission substantially alters basal PRL levels, as shown in the present study. During long-term neuroleptic treatment, a persistent hypersecretion of PRL was found, which is in line with earlier findings (Öhman et al. 1980; Öhman and Axelsson 1980). In the present study, a strong significant correlation was observed between the dose of neuroleptics and the increase in PRL. Such a direct correlation has earlier been questioned (Öhman and Axelsson 1980) but may be explained by the fact that the patients in the present study were investigated in a steady-state after very long periods of unaltered neuroleptic treatment. In our study the rise in PRL could not possibly be attributed to the psychiatric disease since there was a clear difference in serum PRL between depressed patients with and without neuroleptics.

In patients with epilepsy there was a rapid rise in PRL postictally, expecially after seizures of duration longer than 30 s, with a peak level within 30 min. These results are in line with the findings in other studies (Abbot et al. 1980; Dana-Haeri et al. 1983; Pritchard III et al. 1983; Trimble 1978). The rapidly increasing postictal blood levels may reflect an instant loss of dopaminergic inhibitory control on the hypothalamic or pituitary PRL neuronal system (Abbot et al. 1980; Apéria et al. 1985).

Alteration of consciousness has been proposed as the crucial factor for the PRL response in generalized seizures and partial complex seizures (Dana-Haeri et al. 1983; Pritchard III et al. 1983). In this study and others (Collins et al. 1983; Dana-Haeri et al. 1983), a large proportion of partial seizures, especially those with elementary symptoms, and other minor convulsions (myoclonic jerks, absences, short tonic or tonic-clonic seizures) did not seem to result in changes in PRL levels large enough to make it possible to separate such epileptic attacks from pseudoseizures or other types of nonepileptic cerebral convulsions.

Besides alteration of consciousness, the duration of the seizures could also be used to separate different kinds of epileptic manifestations. The results of the present study indicate that in epilepsy a large and fast rise in PRL was found in seizures of longer duration (more than 30 s), whereas PRL following short lasting fits was only modestly elevated. In ECT seizures there was a significant positive correlation between the duration of the seizure and the PRL response in our study. This relationship has not been found by others (Öhman et al. 1976; Apéria et al. 1985) but was reported in an ECT study by Skrabanek et al. (1981). Furthermore, in a series of 5 patients with complex partial seizures (Pritchard III et al. 1983) with EEG recording and exact timing of the attacks, there was a somewhat similar correlation between the duration of the seizure and postictal PRL elevation as found in our study.

Since there are seizures of short duration with involvement of consciousness (e.g., absences and complex seizures) and occasional seizures of longer duration with no increase in PRL levels, there must be other explanations for increased PRL levels in epileptic seizures besides involvement of consciousness or the duration of the seizures. Studies using stereotactically implanted electrodes have shown that PRL always rises following complex partial seizures with bilateral limbic involvement (Sperling et al. 1985). During simple partial sei-

zures with no disturbance of consciousness or limbic involvement there was no rise in PRL levels. Unilateral limbic involvement during complex seizures resulted in a postictal PRL rise in 36% of the seizures. Thus, bilateral limbic involvement always and unilateral involvement sometimes results in increased PRL levels. This is in accordance with the present study where the seizures of long duration were of generalized tonic-clonic type whereas most of the partial complex seizures had a short duration with no or only slight elevation in PRL, possibly as a result of unilateral limbic involvement.

The PRL response to ECT was significantly elevated after each ECT, from ECT-1 to ECT-6, and this has also been shown in another study (Linnoila et al. 1984). In the study by Apéria et al. (1985) there was a significant difference in the PRL response between ECT-6 compared to ECT-1 and a similar tendency (not significant) was found in our study. The diminished PRL response after repeated ECTs, parallel to improvement of depressive symptoms, may reflect increased dopaminergic transmission (Apéria et al. 1985). Increased dopamine transmitter function could also explain the positive results with ECT in severe Parkinson's disease (Modigh et al. 1981). In the present study there was no significant difference in postictal PRL levels between those patients who improved quickly (after ECT 3–8) and those with no or a slow response (after ECT 9–13) and these results are in agreement with those of Öhman et al. (1976).

References

Abbot RJ, Browning MCK, Davidson DLW (1980) Serum prolactin and cortisol concentrations after grand mal seizures. J Neurol Neurosurg Psychiatry 43:163–167

Aminoff MJ, Simon RP, Wiedemann E (1986) The effect on plasma prolactin levels of interictal epileptiform EEG activity. J Neurol Neurosurg Psychiatry 49:702–705

Apéria B, Thorén M, Wetterberg L (1985) Prolactin and thyrotropin in serum during electroconvulsive therapy in patients with major depressive illness. In: Apéria B (ed) A psychoendocrinological study of electroconvulsive therapy in major depressive disorder. Dissertation from the Karolinska Institute, Stockholm, pp 85–102

Cavallo A, Moore DC, Nahori A, Meaumanoir A, Sizonenko PC (1984) Plasma prolactin and cortisol concentrations in epileptic patients during the night. Arch Neurol 41:1179–1182

Collins WCJ, Lanigan O, Callaghan N (1983) Plasma prolactin concentrations following epileptic and pseudoseizures. J Neurol Neurosurg Psychiatry 46:505–508

Dana-Haeri J, Trimble MR, Oxley J (1983) Prolactin and gonadotropin changes following generalized and partial seizures. J Neurol Neurosurg Psychiatry 46:331–335

Davis JM (1976) Comparative doses and charts of antipsychotic medication. Arch Gen Psychiatry 33:858–860

d'Elia G (1970) Unilateral electroconvulsive therapy. Acta Psychiatr Scand 215:[Suppl]

Laxer KD, Mullooly JP, Howell B (1985) Prolactin changes after seizures classified by EEG monitoring. Neurology 35:31–35

Linnoila M, Lieowitz G, Scheinin N, Chang MD, Cutler NR (1984) Effects of electroconvulsive treatment on monoamine metabolites, growth hormone, and prolactin in plasma. Biol Psychiatr 19:79–84

Modigh K, Balldin J, Edén J, Granérus AK, Wålinder J (1981) Electroconvulsive therapy and receptor sensitivity. Acta Psychiatr Scand [Suppl] 290:91–99

Molaie M, Culebras A, Miller M (1985) Nocturnal prolactin levels in epileptics, Neurology 35 [Suppl 1]:133

Molaie M, Culebras A, Miller M (1985) Nocturnal plasma prolactin rise in patients with complex partial seizures. Ann Neurol 18:719–722

Molaie M, Culebras A, Miller M (1986) Effect of interictal epileptiform dischanges on nocturnal plasma prolactin concentrations in epileptic patients with complex partial seizures. Epilepsia 27:724–728

Pritchard III PB, Wannamaker BB, Sagel J, Nair R, DeVillier C (1983) Endocrine function following complex partial seizures. Ann Neurol 14:27–32

Skrabanek P, Balfe A, Webb M, Maguiere J, Powell (1981) Electroconvulsive therapy (ECT) increases plasma growth hormone, prolactin, luteinising hormone and follicle-stimulating hormone but not thyrotropin or substance P. Psychoendocrinology 6:261–267

Sperling MR, Pritchard III PB, Engel Jr J, Daniel C, Sagel J (1985) Limbic seizures and prolactin. Epilepsia 26:529

Trimble MR (1978) Serum prolactin in epilepsy and hysteria. Br Med J Dec 16:1682

Wyllie E, Lueders H, Pippenger C, VanLants F (1985) Postictal serum creatine kinase in the diagnosis of seizures disorders. Arch Neurol 42:123–126

Öhman R, Axelsson R (1980) Prolactin response to neuroleptics. Clinical and theoretical implications. J Neural Transm [Suppl] 17:17–21

Öhman R, Balldin J, Wålinder J, Wallin L (1976) Prolactin response to electroconvulsive therapy. Lancet II:936–937

Öhman R, Forsman A, Larsson M (1980) Prolactin response to haloperidol after a single dose and during prolonged administration. Curr Ther Res 27:137–143

Received March 9, 1987