MODEL OF THE NEWBORN'S PHYSICAL DEVELOPMENT*

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In the present work I try to develop a model that predicts the development time of a newborn that suffers a development delay. The paper should be helpful for parents, who apply therapy to their children and often need to know how long will it take to complete the development.

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1. Introduction

Development of a newborn is a complicated, genetically determined process. During the first year of life, among the others, a genetic program of motor development is run that goes through several well defined intermediate stages. Omission of a certain stage results in a delay in development, or, eventually in development of alternative (pathological) patterns of motion [1–4].

In each case, a delay requires to run the development program from the point of delay to the final stage of development, *i.e.* the stage of unaided walking.

Parents often ask physicians how much will it take for the baby to achieve full motor development. Doctors usually do not give any answer, telling that it can be different from case to case.

In this work I propose a model that tries to rescale the remaining development time according to the Vojta program of development and decrease in brain's neuroplasticity.

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Neuroplasticity describes the ability of neural system to create new signaling pathways [5]. This can be done in two ways: one is the synaptogenesis process, where new synaptic connections are established, the second is the pruning process, where synaptic connections are removed from the system [6].

The first process is easy to understand. The second works by improving the weights of neural connections by removal of synapses. If one removes an inhibitory synapse, the activation of a pathway become easier. In turn, if one removes an excitatory synapse, the possibility of activation for such a pathway becomes decreased [7].

The process of synaptogenesis and pruning act on different time scales. The process of synapse formation starts at the beginning of pregnancy (8th week in rhesus monkeys [8]) and stops almost completely in a two year old baby [6]. In turn the pruning process is much slower, and acts (roughly) across the whole life [6].

2. The graph of a newborn development

To estimate the remaining time of development in a delayed case, one first needs to know the expected unperturbed development time. This can be learned in many books devoted to the development of newborns, *i.e.* [1–4]. The data presented in these books is addressed to physicians, and has no readable graph structure being organized in form of prescriptions similar to "development stage–time $\pm\Delta t$" accompanied with partial informations on the development dependences between stages.

For the purpose of this research I have collected these data in form of a graph as shown in Fig. 1

As can be read in the literature, the development is accompanied by a growing uncertainty (the time after which a doctor needs to be consulted) [2]. One can observe that the uncertainty grows approximately at the rate of 1 week per 4 weeks (1 month) of development.

3. The model

It is possible to approximate the measured synaptic density growth curves presented in [6] with exponentials. I propose to fit these data by

$$f(t) = A\left[1.0 - \exp\left(-\frac{t+7}{T_{\mathrm{s}}}\right)\right]\left[(1-B)\exp\left(-\frac{t+7}{T_{\mathrm{p}}}\right) + B\right], \quad (1)$$

where: A — the proportionality constant — equals 19.3, B — the fraction of synapses that does not undergo pruning — equals 0.28, T_{s} — synaptogenesis time constant — equals to 12 months and T_{p} — the pruning time constant —

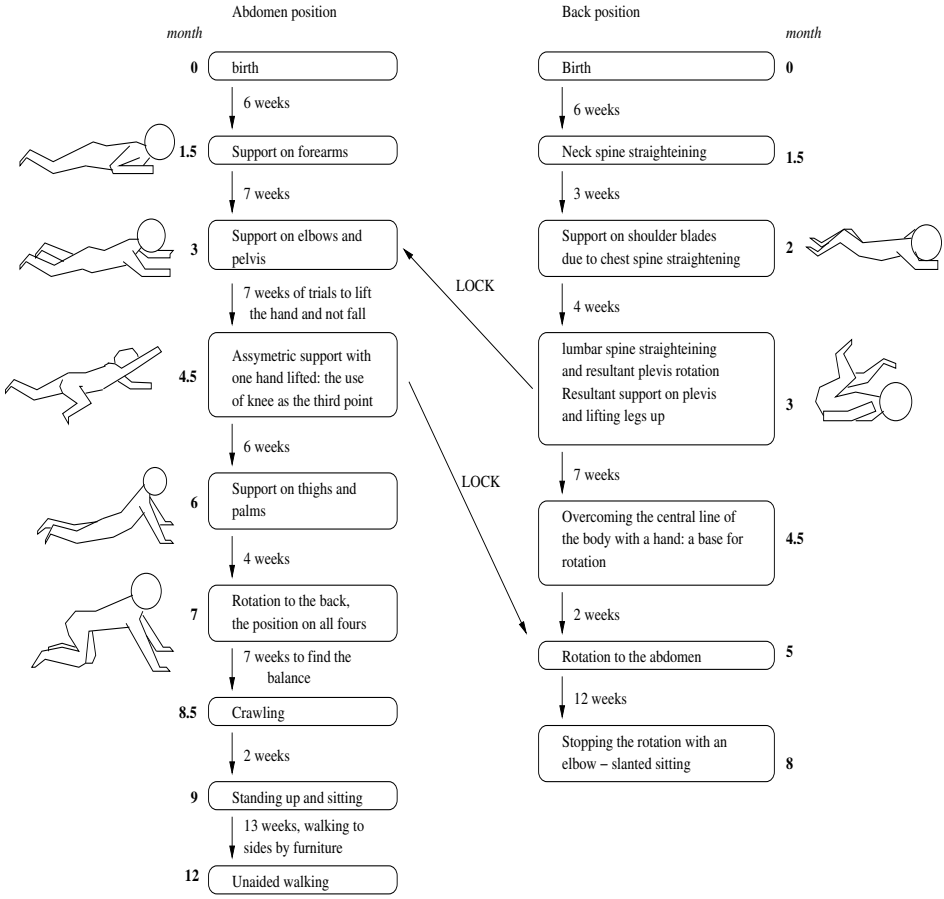

Fig. 1. The graph of newborn's development stages. For a premature newborn, one should use a *corrected age* in this graph, *i.e.* count the months since the predicted date of normal birth.

equals 18 years. This represents quite reasonably the competition between synapse creation and pruning and agrees with literature experimental data (Fig. 2).

Knowing this, I can tell that neuroplasticity is proportional to the *rate* of synaptogenesis plus the *rate* of pruning. Differentiating $f(t)$, we obtain the rate of change in synaptic density as:

$$f'(t) = \frac{A}{T_{\rm s}} \exp\left(-\frac{t+7}{T_{\rm s}}\right)\left[(1-B)\exp\left(-\frac{t+7}{T_{\rm p}}\right)+B\right] - \frac{A(1-B)}{T_{\rm p}} \exp\left(-\frac{t+7}{T_{\rm p}}\right)\left[1-\exp\left(-\frac{t+7}{T_{\rm s}}\right)\right]. \quad (2)$$

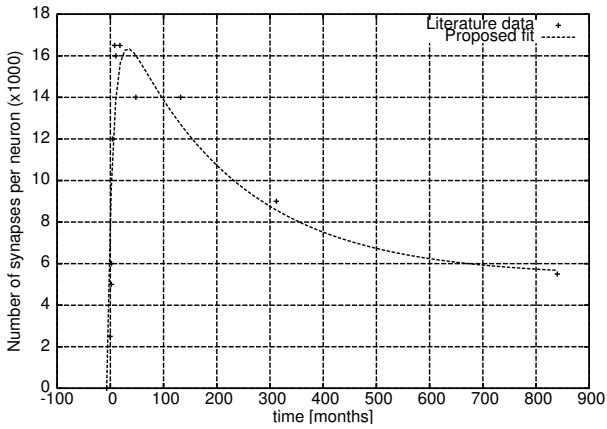

Fig. 2. The accuracy of synaptic density approximation by the proposed fit as compared to the data published in [6].

This derivative describes the two competitive processes: synapse creation and pruning. Both of these processes contribute equally to the neuroplasticity, as seen from the artificial neural network signaling theory, thus the plasticity P can be seen as the sum of these terms:

$$P(t) = \frac{A}{T_{\mathrm{s}}} \exp\left(-\frac{t+7}{T_{\mathrm{s}}}\right)\left[(1-B)\exp\left(-\frac{t+7}{T_{\mathrm{p}}}\right)+B\right] + \frac{A(1-B)}{T_{\mathrm{p}}} \exp\left(-\frac{t+7}{T_{\mathrm{p}}}\right)\left[1-\exp\left(-\frac{t+7}{T_{\mathrm{s}}}\right)\right]. \tag{3}$$

Then, the neuroplasticity is assumed to be directly proportional to the learning rate of a newborn. Thus, if neuroplasticity decreases twice, compared to the reference value, the newborn will need twice as much time to learn certain neural pattern.

Knowing this, I calculate the new time scale and sum up the infinitesimal time steps of development. The equation for the perturbed remaining development time τ (given initial development time τ_0 that corresponds at given stage to t_0 of attaining this stage in unperturbed development plus the delay δ, *i.e.* $\tau_0 = t_0 + \delta$) can be written as:

$$\tau = \int_{t_0}^{t_1} \frac{P(t)}{P(\tau+\tau_0)} dt \tag{4}$$

(note that τ in this integral is a function of t which causes difficulty in analytical treatment, but is fine for numerical calculations).

An example plot of $\tau - (t_1 - t_0)$ for a range of δ (delay in treatment), calculated for $t_0 = 0$ and $t_1 = 12$ months is shown in Fig. 3. This represents a situation where a newborn suffered a defect at birth, *i.e.* its development is fine compared to age, but does not develop further and requires therapy. The delay illustrates the waiting time before the therapy has been undertaken. The resultant time τ is accompanied by an uncertainty of $\sigma_\tau = \gamma\tau$, where $\gamma = 1/4$ [2].

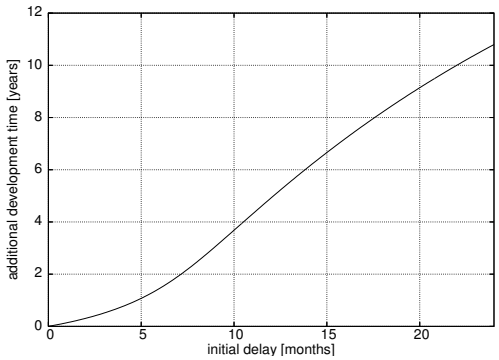

Fig. 3. The impact of initial delay in development (horizontal axis) on the remaining development time in case, when there is full 12 months of normal development remaining.

As can be seen, until six months of waiting, the development prolongs only by a year. The development is still dominated by the synaptogenesis. Then, the creation of new synaptic connections becomes more and more difficult in a short time period. The therapy becomes dominated by the slower pruning process and takes longer now, *i.e.* after 10 months it can reach almost 4 years. When dominated by pruning, the delay appears to be almost a straight line, since the pruning time constant $T_\mathrm{p} = 18$ years and causes very slow variation in the development time (seen not as an exponential but rather a straight line). After *c.a.* 14 years, the pruning resources become also limited and the waiting time blows to infinity (not shown in the figure).

The results can be qualitatively compared with the literature data [1], where the author investigated a group of 110 children, of which those, who started treatment at age of 4.33 months, needed in average 25.92 months of therapy (Fig. 3: 10.3 months), those who started at age of 1.73 needed in average 6.83 months of therapy (Fig. 3: 3.1 month), and those who started at age of 2.41 needed in average 5.73 months of therapy (Fig. 3: 4.7 months).

Comparing with my model, one must take into account many factors that complicate the approach, *i.e.* unknown time of the defect manifestation (not necessarily at birth as in my plot), possible alternative motion patterns

which deform the development procedure (very probable if one delays with the therapy), and also possibility of more severe deficiencies, which cannot be cured and prolong the average therapy time for given group beyond the case considered in this paper.

One should also take into account that abandonment of treatment in early age is difficult because the treated newborn should be assisted in achieving large development stages even when its development corresponds to its age far earlier. Also, the graph presented here, describes the *extra* time needed in development, compared to the time needed by a healthy child. This extra time and the natural time are not well separated in experimental data.

4. Concluding remarks

In this paper I try to give a tool to predict the development time of a newborn with neurological deficiencies. I try not to go into deep details to keep the idea simple and useful. The results seem to be reasonable, *i.e.* one can see the important threshold of 6 months after which it becomes significantly more difficult to fix the deficiencies (this is well known to doctors by their experience). A clear conclusion of this paper is therefore the need for *early* diagnostics in children. *Many of the children that suffer neuro-motor problems could have been cured in their infancy!*

I hope that this model (possibly further improved) will be of great use for the worried parents and will stimulate them to quick neurological consultations after noticing worrying symptoms.

I also hope that this paper, written in English, will build an interest in Vojta principle in English, as by now this method is used mainly in Europe with a main scientific center in Germany.

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