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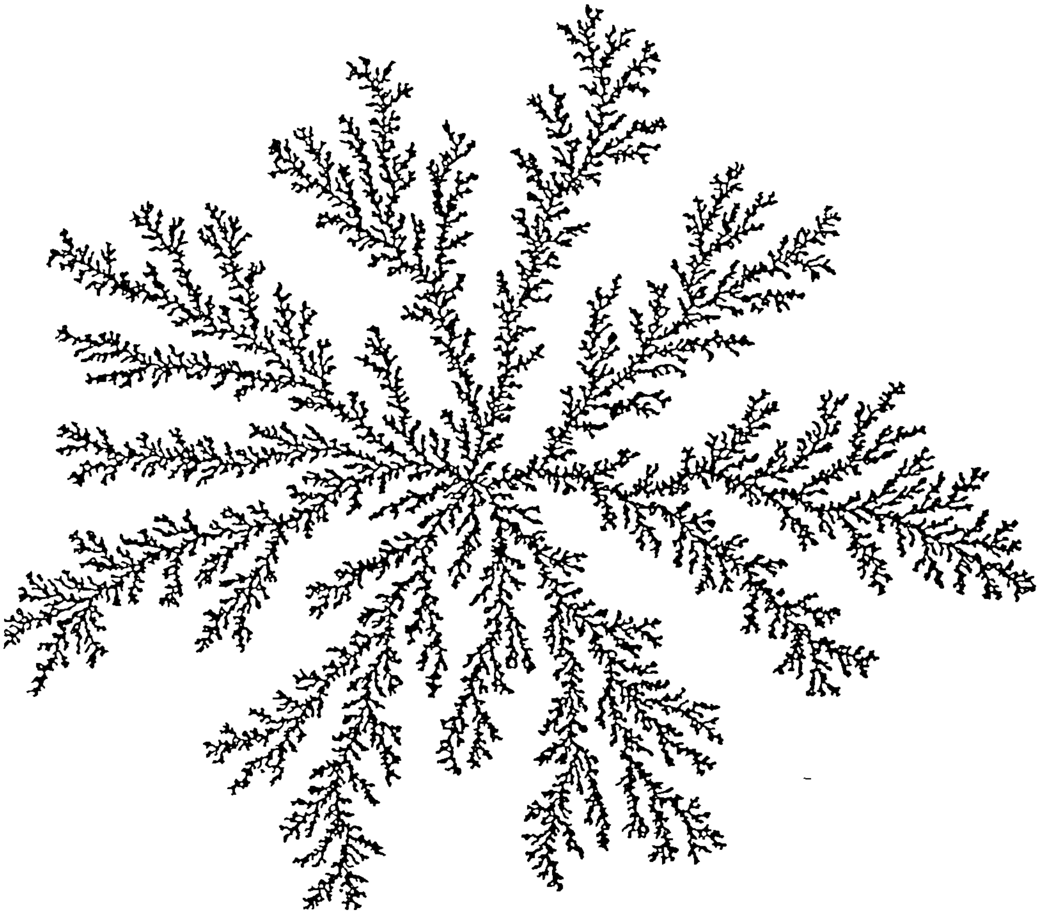

DLA fractal cluster
of 10^6 particles

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HOMINID CEREBRAL LATERALISATION AS SPONTANEOUS SYMMETRY BREAKING

Ágnes Holba and Béla Lukács

Á. Holba: Physicist, (b. Budapest, Hungary, 1943).
Address: Central Research Institute for Physics, Budapest 114, P.O.B. 49, H-1525 Hungary, E-mail: holba@rmk530.rmki.kfki.hu .
Fields of interest: Spurious scattering, computer graphics, data processing.
B. Lukács: Physicist, (b. Budapest, Hungary, 1947).
Address: Central Research Institute for Physics, Budapest 114, P.O.B. 49, H-1525 Hungary, E-mail: lukacs@rmk530.rmki.kfki.hu .
Fields of interest: Cosmology, general relativity, heavy ion physics, speech acoustics, economics.

Abstract: *The appearance of human cerebral lateralisation as a spontaneous symmetry breaking is investigated. The basic assumption is that an individual having speech center only on one side and spatial orientation one on the opposite possesses greater fitness than the symmetric individual. The reason may be anything, e.g., the dense packing and interference of the two centers on one side. The simplest possible model gives almost instantaneous lateralisation when the relative fitness passes the critical value 2.*

1. THE PROBLEM

Homo sapiens shows an expressed left-right functional asymmetry, especially in handedness, whose literal translation would be chirality. More than 90 per cent is right-handed, and a similar fraction has the cerebral speech center in the left half of the brain (Passingham, 1982). This fact is somewhat balanced by the similarly unilateral right position of the center for 3-dimensional geometric orientation. For chimpanzee such cerebral asymmetries are not known, and chimpanzee populations are not asymmetric for chirality (Prossinger, 1989). In this paper we try to interpret the human asymmetry as a manifestation of a spontaneous symmetry breaking. (For the idea of broken cerebral symmetry see (Prossinger, 1989).) In a model calculation we demonstrate that if some unequalities hold, then such a spontaneous symmetry breaking will propagate through the total population in some dozens of generations; the direction is random, but after the transients it remains unchanged.

2. ON TIME SCALES

Here we try to estimate the time scale of the process from the very scarce information available now.

1) Chimpanzee as species shows no chirality, hence the cerebral lateralisation is probably younger than 5-7 Mys (the age of man-(chimpanzee+gorilla) bi- or trifurcation (Sarich and Wilson, 1967).

2) Stone tools suggest human right-handedness from 1.5-2 Mys, and endocasts show an asymmetry greater than for apes (Toth, 1989).

3) The emergence of speech happened somewhere between 1.5 Mys and 35 kys (Passingham, 1982). For the latter bound, *Homo sapiens* (or *Homo sapiens sapiens*) goes back slightly before 35 kys (the horizon of C^{14} method). The speech ability of *Homo neanderthalis* is still open for discussion: cerebral capacity is quite sufficient, the form of the mandible is not too lucky, there are significant differences in the details of the vocal channel between us and the *Neanderthals*, while acoustic simulations result in a possibility of limited but substantial phoneme set (Lieberman, 1975).

3. THE MODEL

We use the simplest nontrivial model with a possibility for spontaneous symmetry breaking. The model is certainly oversimplified but works. It may anticipate the results of more elaborated later ones. *We assume that*:

1) cerebral asymmetry increases the fitness above a critical level of cerebral complexity (e.g., since the speech and spatial geometry centers are both space-consuming, or since in duplicate the copies could disturb each other (Passingham, 1982)), the relative gain is $v > 1$;

2) there is a single gene behind the lateralisation, with 3 alleles: wild 0, right-handed + and left-handed −, genotypes 00 and +− give a phenotype without lateralisation (i.e., both centers in both halves), 0+ and ++ lead to right-handed phenotype and 0− and −− to left-handed one (this being the simplest scheme);

3) + and − lateralisations give the same gain v (no preferred direction);

4) the mutation probabilities are

$$p_{0\to+} = p_{0\to-} = \epsilon < \delta = p_{+\to0} = p_{-\to0}\,;\; p_{+\to-} = p_{-\to+} = 0$$

5) the mating is random.

Hence one gets a recursive system of equations (see the Appendix), solvable for any given initial condition. In the actual calculations $\epsilon = 10^{-4}$, $\delta = 3\times10^{-4}$.

4. INITIAL CONDITIONS

Since gene 0 is the wild form, 3 types of initial conditions may have relevance.

1) *Symmetric*. We solve the evolution equations with $\nu = 1$ staring from the phenotypic concentration $c_{00} = 1$ for asymptotic times. The result is $c_{00} = 0.9996$, $c_{0+} = c_{0-} = 0.0002$, anything else is $\sim 10^{-8}$. This is the unperturbed wild population, and a symmetric initial condition.

2) *Minimally asymmetric*. Fluctuations (of order $1/\sqrt{N}$) are expected in any population. Since for any primordial population $0.0002N < 1$, the symmetric initial state cannot exist. We assume that by fluctuation $c_{00} = 0.9998$, $c_{0+} = 0.0002$, $c_{0-} = 0$. This minimal asymmetry is supposed to be right-handed, which is as probable as the opposite.

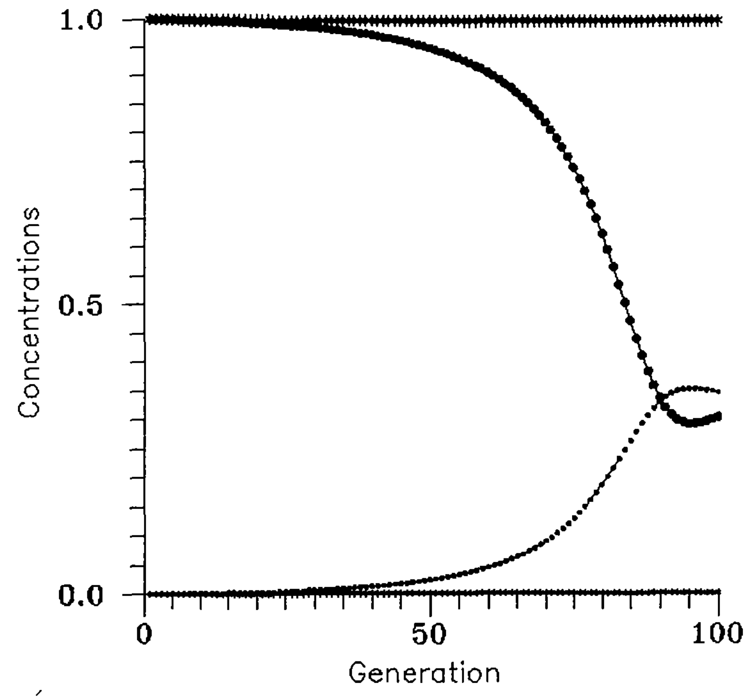

Figure 1: Evolution from the symmetric initial condition. Continous: wild; dotted: right-handed phenotypes. $\nu_{\text{thin}} = 1.9$; $\nu_{\text{bold}} = 2.1$

3) *Mixed.* After a contact of asymptotic homozygote populations. Initially only c_{00} and c_{++} are not 0.

5. RESULTS

Figure 1 is an evolution from the symmetric initial condition, for 100 generations; $\nu = 1.9$ and 2.1 respectively. Both the genotypes and the phenotypes remain symmetric. For $\nu = 1.9$ the final state very much resembles the initial one. On the other hand for $\nu = 2.1$ the wild gene almost completely eliminates, so the 0 phenotype is produced by + – genotype.

Figure 2 is the same for the minimally asymmetric initial condition. Again no serious change for $\nu = 1.9$. However, for $\nu = 2.1$ not only the wild 0 gene eliminates but the – one as well. Now observe that *(i)* the + and – genes are equivalent for function; *(ii)* fluctuations of order 10^{-4} always happen. So for $\nu > 2.1$ the total lateralisation of the population is inevitable in 100 generations.

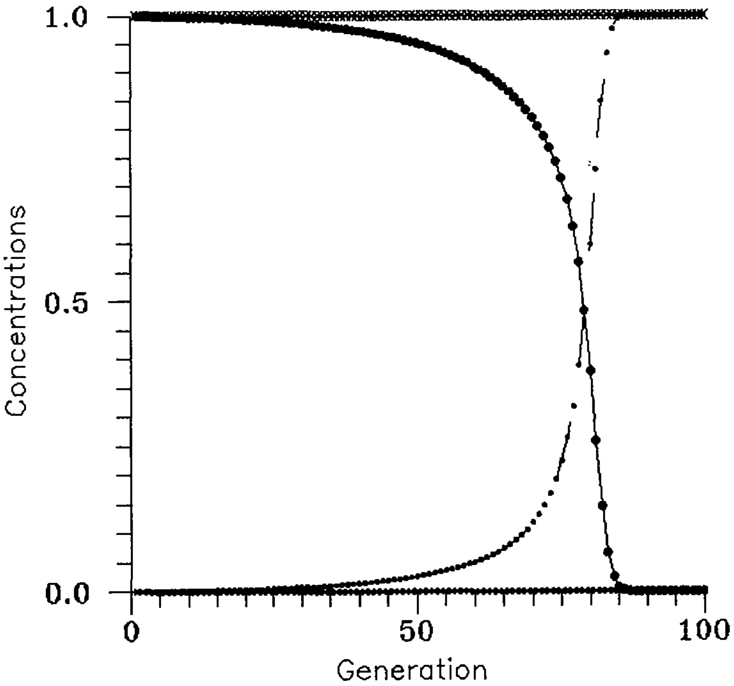

Figure 2: Like Figure 1, but for the minimally asymmetric initial conditions.

Figure 3 shows some phenotypic concentrations in the 100th generation from the minimally asymmetric initial condition, vs. v. As seen, except for a narrow range either nothing happens or the phenotype becomes almost purely +. Numerical experience suggests that the transitional range would simply need more generations.

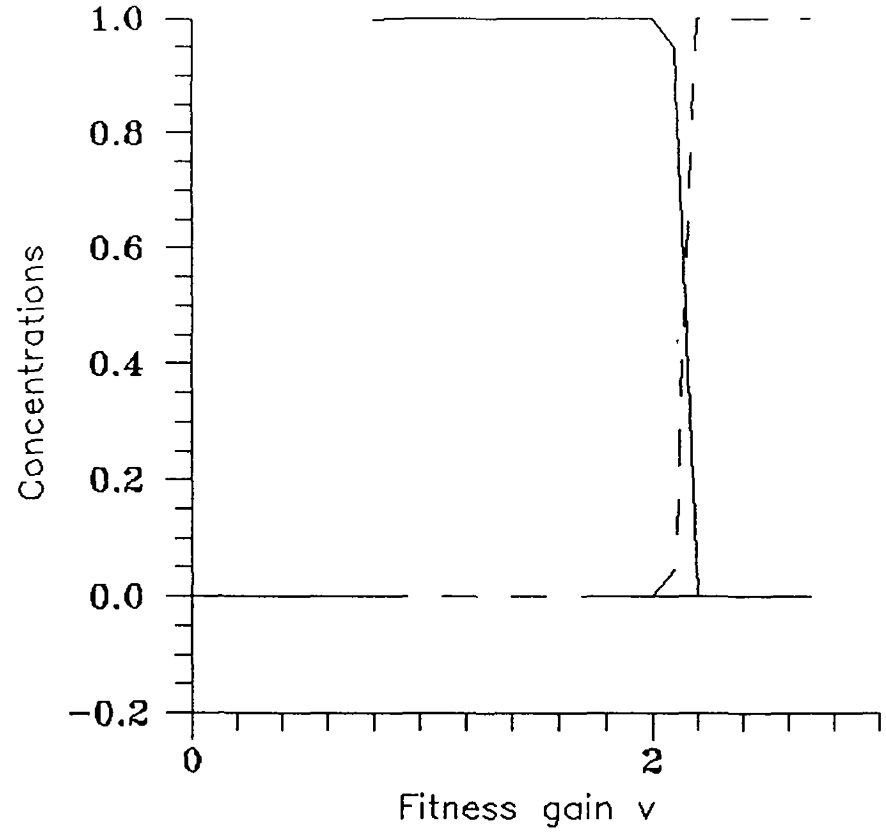

Figure 3: Concentrations after 100 generations, the minimally asymmetric initial condition. Continuous: unlateralised; dashed: right-handed phenotypes.

Figure 4 shows the critical line in mixed initial conditions. From above the line, c_{++} increases towards an asymptotic value very near 1; from below the state tends to almost purely the wild one. As seen, for $v > 2$ an arbitrarily small mixed right-handed injection can lateralise a wild population.

Finally, Figure 5 is the evolution from the special mixed initial condition $c_{00} = c_{++} = 0.5$ for $v = 1.01$. (The initial generation is not shown.) Even such a moderate difference eliminates the wild form in 20 generations.

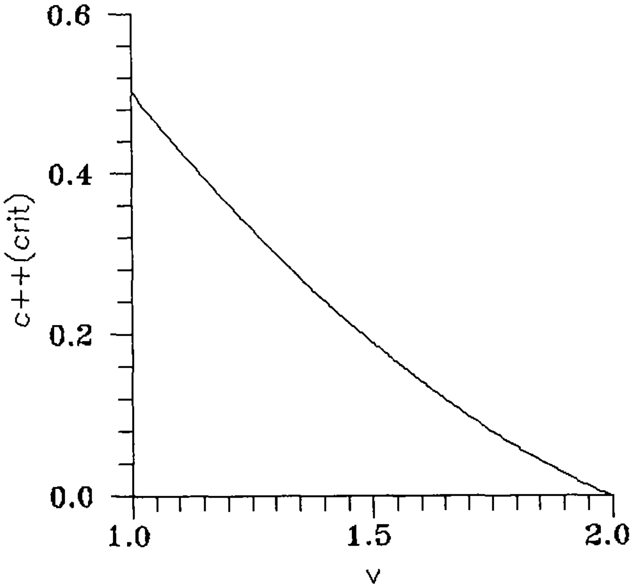

Figure 4: Mixed (++) and (00) initial condition. From above $c_{++\mathrm{crit}}(\nu)$ $c_{++} \to 1$.

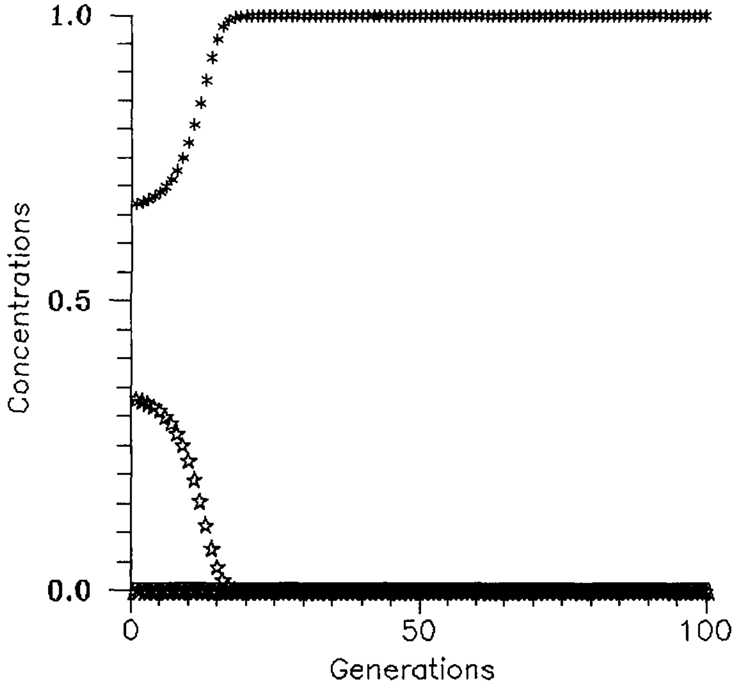

Figure 5: Evolution from mixed initial condition. $c_{++} = c_{00} = 0.5$, $\nu = 1.01$.
(stars): unlateralised; (*): right handed; (■): left-handed

6. CONCLUSIONS

The model system shows a spontaneous symmetry breaking for $\nu > 2$. It was assumed that the laws themselves are symmetric for the $(+-)$ exchange, therefore the symmetric state is a solution of the evolution equation, but it is unstable for $\nu > 2$. Any small initial fluctuation leads to a correspondingly asymmetric final state; since there is no preferred side for the fluctuations, the probability of final right and left-handed states is 50-50 per cent. The once lateralised population lateralises any further wild population in contact if $\nu > 2$.

The results of the present toy model suggest a simple coherent history. Since the overwhelming majority of *Primates* was always arboreal, the 3 dimensional geometric center must have been older than the speech center. Now consider a population with slowly developing speech ability, so with the increase of the corresponding center in the brain. Assume that after a stage the two centers disturb each other because too dense packing. Then the unlateralised individuals become more and more handicapped; relatively any lateralised one will have better speech ability, spatial orientation, manipulation, toolmaking or such, i.e., ν is increasing. Parallelly the + and − genes are accumulating. However for moderate ν's the dominant phenotype remains unlateralised (cf. Figs. 1-3). The situation, however, abruptly changes when ν exceeds the critical 2. From then the first small asymmetric fluctuation in gene concentrations led to rapid elimination of the mirror-reflected gene (cf. Figs. 2 and 3). This, of course happened in one local population (first exceeding $\nu = 2$ in the local circumstances). Then, if any neighbouring community moves into the local environment which prefers so much the ability $\nu > 2$ then a mixed initial condition with + surplus appears, which again rapidly eliminates the 0 and − genes (cf. Fig. 5). If the speech ability is younger than lateralisation (which is, as seen, quite possible), then the driving force must have been different. Then the simplest explanation is as follows. A speechless individual with doubly represented spatial geometry center would not have too clear feeling of the difference of left and right, which would be a handicap of toolmaking. Having eliminated one of the centers, a functional asymmetry occurs for manipulation. It would be hard to say anything more definite in this moment.

The present model is deliberately oversimplified. However even this model works and gives results conform with the very limited number of palaeonthologic facts known. E.g., this mechanism may lead to a history in which all populations are lateralised to the same sides; and right-handedness is characteristic for all present populations as well as for populations 1.5 Mys ago (according to the forms of the stone tools (Toth, 1989)). The critical value $\nu = 2$ seems rather high for first sight; it may be a consequence of oversimplifications, but such a value may have appeared in harsh primitive circumstances.

To be sure, we do not claim to have proven that the human lateralisation was a spontaneous symmetry breaking. Many vertebrate bodies are slightly asymmetric; see e.g., the asymmetric position of heart in mammals which may cause slight asymmetries in blood circulation, so in cerebral circumstances as well. Assuming $\nu_+ > \nu_-$ for any reason, a slow accumulation of the + gene would happen. However, the expected differences may be only very slight in the best case, resulting in a long timescale of transition. If the transition were found to be rapid, then the spontaneous symmetry breaking would remain as the only reasonable mechanism; on the other hand the spontaneous symmetry breaking is an existing mechanism in any case when the lateralisation in itself gives higher fitness, and it works any ways above a critical increase of v.

APPENDIX: THE EVOLUTION EQUATIONS FOR GENE CONCENTRATIONS

In general, the recursive equations for the gene concentrations can be built up similarly as in (Holba and Lukács, 1990). The whole system is rather complicated, so here we go step by step.

Let us start with the scheme

Genotype	Phenotype	Gain
0 0	0	1
0+	+	$v>1$
0−	−	v
++	+	v
+−	0	1
−−	−	v

Introduce concentrations in the population c. For genotype it is $c_{i\alpha\beta}$ where i labels the generation, and $\alpha = 0,+,-$. For phenotype we have $c_{i\alpha}$. Now introduce the gene concentrations $c_{i\alpha}$; obviously

$$c_{i\alpha\beta} = Q_i C_{i\alpha} C_{i\beta} \tag{A.1}$$

where Q_i is a normalizing factor.

For the phenotypes

$$\begin{aligned} c_{i0} &= c_{i00} + c_{i+-} \\ c_{i+} &= c_{i0+} + c_{i++} \end{aligned} \tag{A.2}$$

and similarly for −. The mating probabilities are enhanced by factors 1, v, v^2 according to the 0 or ± phenotypes of the individuals in the pairs. Finally, the meiotic scheme, with mutation leads to

$$\begin{array}{lcl} (0\,0) & \rightarrow & 2(1-2\epsilon)(0) + 2\epsilon(+) + 2\epsilon(-) \\ (0+) & \rightarrow & (1-2\epsilon+\delta)(0) + (1-\delta+\epsilon)(+) + \epsilon(-) \\ (++) & \rightarrow & 2\delta(0) + 2(1-\delta)(+) \\ (+-) & \rightarrow & 2\delta(0) + (1-\delta)(+) + (1-\delta)(-) \end{array} \tag{A.3}$$

+ mirror reflected two further relations. Hence

$$\begin{aligned} C_{i+10} &= 2(1-2\epsilon)\, c_{i00} + \nu(1-2\epsilon+\delta)\, c_{i0+} + \nu(1-2\epsilon+\delta)\, c_{i0-} + \\ &\quad + 2\nu\delta c_{i++} + 2\delta c_{i+-} + 2\nu\delta c_{i--} \end{aligned} \tag{A.4}$$

$$\begin{aligned} C_{i+1+} &= 2\epsilon\, c_{i00} + \nu(1+\epsilon-\delta)\, c_{i0+} + \nu\epsilon c_{i0\pm} 2\nu(1-\delta)\, c_{i++} + \\ &\quad + (1-\delta)\, c_{i+-} \end{aligned}$$

+ a mirror-symmetric equation.

Now the computation starts from some initial conditions for $c_{\alpha\beta}$ and proceeds according to

$$c_{i\alpha\beta} \rightarrow C_{i+1\alpha} \rightarrow c_{i+1\alpha\beta} \rightarrow \dots \tag{A.5}$$

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