# VACCINATION AND THE DYNAMICS OF CRITICAL DAYS

by Viera Scheibner, PhD ©2004

Nexus Magazine (Vol 12, No 6, Oct-Nov 2005)



**Scientific evidence shows that babies can have severe adverse reactions to vaccinations at critical intervals following their shots, and that vaccination is the more likely cause of cot death and shaken baby syndrome.**

## Dynamics of critical days as part of the dynamics of non-specific stress syndrome discovered during monitoring with Cotwatch breathing monitor

Recent editorials in the British Medical Journal (BMJ) by a number of authors have motivated me to publish the results of research into babies' breathing. This was research that the late Leif Karlsson (a Swedish biomedical electronics engineer living in Australia) and I conducted with the Cotwatch breathing monitor, developed by Leif in 1985-86 at my suggestion. Leif died in 1994 and the Cotwatch breathing monitor died with him: I had it delisted with the Therapeutic Goods Administration (TGA), and since 1994 it has not been distributed.

Cotwatch was a true breathing monitor, meaning its electronics separated heartbeat and breathing and only breathing delayed the alarm. The feedback on breathing from the standard home monitor was from alarms (figure 1), while the microprocessor-based unit provided computer printouts of the record of breathing in the form of histograms stacked up at an angle (figure 2) or vertical bars (figures 3,4), the length of which directly reflected the stress level as integrals of the weighted apnoea/hypopnoea density (WAHD).

The record of alarms in a baby over a period of five-and-a-half months, from October 1987 to March 1988 (figure 1), reveals that the stress-induced breathing pattern did not subside after 21 days following vaccine administration: it was still continuing on and off (following the critical days) two-and-a-half to three months later; and before really recovering from the first lot, the child was given the second injection of DPT and oral polio vaccines. Cotwatch recorded events in breathing: apnoeas (pauses in breathing) and hypopnoeas (low-volume breathing, i.e., below 5% of the volume of normal unstressed breathing). The events were logarithmically weighted (the figures on the vertical axis of the computer printouts are integrals of the WAHD).

The first two charts in figure 3 are computer printouts of the record of events in breathing in two babies: baby one, who was given the third DPT (diphtheria-pertussis-tetanus) vaccine and OPV (oral polio vaccine); and baby two, who was given the first DPT and OPV. The higher the vertical bar, the higher the stress level in breathing; figure 3 shows flare-ups of stress-induced breathing day by day from day 0 when the vaccines were administered and up to the 17th day.

It is obvious that even though baby one reacted much more than baby two, the flare-ups of stressed breathing followed the same pattern of critical days, the most important of these being day 2, after which day the stress level went down and started rising again between days 5 and 7, when the stress level subsided and started increasing again between days 14-16, subsided again and rose again between days 19-24, after which it subsided and rose again towards the 28th day and so on, following closely the pattern of alarms as recorded by a mother of one baby (figure 1). Days 10 or 11 also emerged as critical days in babies who reacted strongly, such as baby one. Needless to say, the increased intensity of reactions after the third DPT injection and OPV reflects the phenomenon of sensitisation (sensitisation in this context means increased deranged immunological response or anaphylaxis; and in the case of vaccines it also means increased susceptibility to the diseases that the vaccines are supposed to prevent and to a host of unrelated bacterial and viral infections (Parfentjev, 1955; Craighead, 1975; Daum et al., 1989).

The third chart in figure 3 is of 41 actual, randomly listed deaths after DPT and OPV; it can be seen that the distribution of deaths closely follows the dynamics of the flare-ups of stressed breathing of babies one and two after the administration of the DPT vaccine (Bernier et al., 1982, Walker et al., 1987, Coulter and Fisher, 1991).

Figure 4 illustrates that in our research every baby was its own control (the data measures the stress level in every baby before and after vaccination). For a number of days there was no stress level in breathing, then conies day 0 when the vaccine was administered and one can see how the babies reacted day by day. Figure 4 represents two babies (baby one and baby three) and one can see the individual differences in

response, since baby three reacted within the first 24 hours, and also that the highest stress level occurred for baby one on days 5-6, while for baby three it occurred on day 7, but this is to be expected since babies are individuals in their own right. One must also take into consideration that in statistics you always have a slight spread of a day or two before or after the critical days. One can also rephrase it that nature does not necessarily operate in a sudden, cut-off fashion but in a building-up and tapering-off way.

Figure 4 also illustrates the individuality of stress response after the 16th day: baby three had a significant delayed reaction towards the 24th day, while baby one had only a slightly increased stress level towards the 24th day.

**Underlying mechanisms of the cycle**

Immunological research unwittingly provided another explanation for the observed and recorded slight differences in the daily dynamics of maximum stress response. Takacs et al. (1997) studied the possible underlying mechanisms of the cyclic pattern of relapsing/remitting experimental allergic encephalomyelitis (EAE). Their approach was to conduct a longitudinal study correlating epitope recognition and cytokine production by draining lymph node cells, splenocytes and central nervous system (CNS) infiltrating cells with disease during relapsing and remitting EAE. Responses of lymph node cells and splenocytes were uni-formative with respect to epitope spread. However, there were interesting day-by-day dynamics as far as the time-course of T cell responses in lymph nodes was concerned.

EAE was induced with 200 micrograms of PLP (proeolipid protein) 139-151, PLP 178-191 or MBP (myelin basic protein) 87-106, emulsified in IF A supplemented with 200 micrograms of Mycobacterium tuberculosis and M. butyricum 8:1 and given subcutaneously (s.c.) on days 0 and 7. Immediately after this "immunisation" and 48 hours later, mice received 200 nanograms of Bordetella pertussis toxin (intraperitoneally) in PBS (protein baseline serum). Relapse was defined as a weight loss and clinical worsening was characterised by at

Figure 1: This record of alarms in a baby over a period of five-and-a-half months, from October 1987 to March 1988, reveals that the stress-induced breathing pattern did not subside after 21 days following vaccine administration.

Figure 2: Record of breathing in the form of histograms stacked up at an angle.

Figure 3: Record of breathing in the form of vertical bars.

least one full-grade clinical score after stable recovery, indicated by weight gain and at least one full clinical score.

Without going into great detail, strong proliferation to the PLP 178-191 peptide used to induce disease was detected as early as day 4 after "immunisation", reaching a peak by days 9-11. At the time of remission, days 15-16, a considerable decrease in proliferative capacity of lymph node cells was detected. IFN-gamma (interferon gamma) production followed the same pattern; some variability was observed between individual mice, but a relatively high concentration was measured during the first 11 days, decreasing thereafter. The highest concentration of IFN-gamma was measured at the time of disease onset, on day 11. The response to PLP 178-191 gradually waned and was lowest at day 17, which in almost all mice is a silent period of the disease. Days 22-25 were characterised by an increase in IFN-gamma production again: this is the time point which, in most mice, precedes detectable relapse.

Equally interesting are Takacs et al.'s immunological time dynamics from days 42 to 48, as established by our monitoring of stress response to vaccination in babies. These are the days with increased stress level in breathing and increased numbers of deaths after vaccination. The weight loss/weight gain dynamics accompanying the above immunological challenge is equally relevant to babies after vaccination. Leif's and my studies confirmed the validity of Hans Selye's concept of nonspecific (or general adaptation) stress syndrome as a characteristic but nonspecific response in mammals to any noxious substance or insult or injury of any kind (Selye, 1978). However, since our recording of breathing was done with a non-touch medical technology (Cotwatch had a sensor pad positioned under the mattress and nothing was attached to the body of the monitored person or an animal), we could record longitudinally for long periods of time (hour-by-hour or day-by-day recording of stress dynamics in breathing), while Selye studied the dynamics of adreno-cortical activity and had to perform invasive blood tests which limited the density of his record. His research only demonstrated the dynamics of stress response in very general terms as an alarm reaction (48 hours after the insult), a stage of resistance (an undetermined number of days after the first 48 hours) and a stage of exhaustion (another alarm-like reaction) following the stage of resistance (of undetermined duration) approximately corresponding to the 16th day. Our much more detailed recording of stress response established that the alarm reaction is biphasic and includes two flare-ups of stressed breathing, one on day 2 and another between days 5 and 7, then followed by about seven days corresponding to the stage of resistance, and the increased stress level around day 16 representing the stage of exhaustion.

**Vaccination and cot deaths**

Figure 4: Record of breathing in the form of vertical bars, showing the stress level in each baby before and after vaccination.

Figure 5: Tabulation of data on deaths after DPT and polio vaccination (Mitchell et al.).

Figure 6: Tabulation of data from four groups of babies who died after DPT and polio vaccinations (Griffin et al.).

Figure 5 represents tabulation of raw data on deaths after DPT and polio vaccination published by Mitchell et al. (1995). These New Zealand authors concluded that "there was a reduced chance of SIDS [sudden infant death syndrome] in the four-day period after immunisation" and hence that immunisation "may even lower the risk of SIDS" (though also saying that they cannot confidently state it as a certainty).

However, far from showing protection against cot death by vaccination, Mitchell et al.'s data show that all those babies they studied died as a direct consequence of their DPT and OPV vaccination, showing perfect clustering along the critical days. The "reduced" risk of SIDS in the "immunised" group is misleading, because only those who received vaccines on schedule were categorised as "immunised". Obviously this biases this group to be relatively healthier children because a, or the, major reason for vaccines not being given on time, and sometimes not ever again, is the child being unwell, at least when the shots are due, if not constantly. So, ironically, a child who suffered visible adverse effects from previous vaccines is likely to be in the "non-immunised" category in this study, even if he or she received further vaccines.

Generally speaking, the most fundamental error of judgement displayed by cot death researchers is that they do not look at what happened to the babies who succumbed to SIDS, days before they died, and instead they try to identify the elusive entity of "at risk" babies. The pneumographic studies are done without any regard to what happens to babies in the first six months and/or one year or 18 months of life when the initial DPT, Hib and polio vaccines and the first MMR and/or booster vaccines are given.

Vaccinations are mostly ignored in cot death studies. In our experience, the timing of pneumographic studies is determined by the availability of a bed in the overnight study unit rather than by looking at what happened to the baby just before it developed symptoms of stress or started causing alarms on its monitor.

The notion of false alarms, widely used by those who conduct monitoring of babies' breathing, has also delayed the understanding of the situation. Alarms which occurred when the monitored baby did not stop breathing but was breathing very shallowly are considered false alarms. Leif and I called them "warning alarms" because they sounded when the monitored babies started having longer and longer episodes of low-volume breathing, which is the true stress-induced breathing pattern. A baby who developed pneumonia experienced such "false alarms" for two weeks before going

down with typical symptoms of pneumonia. This happened about six weeks after the six-month vaccination with DPT and polio vaccines.

When reactions or deaths occur six weeks after vaccination, they would not be considered as being caused by vaccination. Yet our records of alarms with Cotwatch microprocessor computer printouts demonstrate increased stress level in breathing more than six weeks after vaccination.

**Deaths after vaccinations**

Griffin et al.'s (1988) data on deaths after vaccination are of interest as well. Even though the authors concluded that their data do not show the causal link, a proper tabulation of their own raw data (figure 6), looking at four groups of babies who died after DPT and polio vaccination, shows the following:

- Group 1 included babies aged 1.5-2.5 months (in the USA, vaccinations start at 6-8 weeks). The majority of these babies died within 8-14 days after the first dose.

- Group 2 included babies aged 2.5-4 months, who died after the second dose of DPT and OPV. The majority died between 15 and 30 days.

- Group 3 included babies aged 4-8 months, who died after the third dose. The majority died more than 31 days after vaccination.

- Group 4 included babies who died aged 8-12 months. These are the residue of delayed deaths after the third dose.

Far from showing no evidence of the causal link with the administration of DPT and OPV vaccines, the tabulated raw data by Griffin et al. show three important observed phenomena:

1. Younger babies died earlier than older, bigger babies who took longer to die.

2. Sensitisation: there was increased immunological reaction (anaphylaxis) after subsequent doses of vaccines.

3. Increased numbers of deaths with the increasing interval from vaccination: delayed reactions, which are a rule rather than an exception.

Interestingly, Torch (1982, 1986) independently also made the same observation as Leif Karlson and I: an increasing number of deaths with the increasing interval from the vaccine administration, increasing number of injections and increasing age. Torch (1982) wrote: "Preliminary data on the first 70 cases studied shows that 2/3 had been

immunized within 21 days prior to death... In the DPT SIDS group 6.5% died within 12 hours of inoculation, 13% within 24 hours, 26% within 3 days, and 37%, 61% and 70% within 1,2 and 3 weeks respectively. Significant SIDS clustering occurred within the first 2 to 3 weeks of DTP #1,2,3 or 4. The age range in the DTP group was 59 days to 3 years..."

One of many points I am making here is that the recipients of a vaccine such as DPT or OPV may react for more than 21 days after the vaccines are administered, this being additional information to that published by Innis (2004). Innis puts emphasis on the period of under 21 days from vaccination as a risk period for the onset of symptoms that can lead to allegations of child abuse, based on the 22 cases that he has analysed to date.

Vaccines, such as that for pertussis, are actually used to induce so-called experimental allergic encephalomyelitis (Levine et al., 1966; Levine and Sowinski, 1979; Steinman et al., 1982; and many others). Steinman et al. described an animal model for pertussis vaccination encephalopathy. They vaccinated mice with the heat-killed Bordetella pertussis-vaccine combined with bovine serum albumin (BSA). They concluded that neuropathology in their mouse model resembled that of human cases in which death occurred after DPT vaccination: diffuse vascular congestion and parenchymal haemorrhage in both the cortex and white matter. Cortical neurons showed ischaemic changes, and areas of hypercellularity were evident in the meninges.

B. pertussis has a wide range of physiological effects including increased IgE production, increased sensitivity to anaphylactic shock, lymphocytosis and hyperinsulinaemia. Its ability to induce increased vascular permeability may account for the tendency to produce haemorrhages. The relevance of the murine [i.e., mouse-related] model of pertussis vaccine encephalopathy is demonstrated by most babies being exposed to cow's milk (even in breast-fed babies) due to a pre-existing anti-BSA antibody. This sensitisation to BSA may lead to a similar chain of events following pertussis vaccination in genetically susceptible human babies.

When babies were only given four vaccines at one session (DPT and OPV), they developed the so-called minimal pathology: petechial (spot-like) bleeding into the thymus, pericardium, lungs and other organs and their deaths were classified as SIDS (which should stand for "sudden immunisation death syndrome"). Such pathology was considered insufficient to cause death, even though it was obviously sufficient, considering that tens of thousands of babies have died this way. According to Hess (1920) and many others, one of the symptoms of acute scurvy is petechial haemorrhage. Why consider scurvy in post-vaccinal death?

Vaccines are a cocktail of toxic substances such as formaldehyde (interestingly, when Selye discovered nonspecific stress syndrome, the first "noxious substance" he injected into his laboratory rats was formaldehyde), aluminium phosphate and aluminium hydroxide, mercury compounds (thimerosal, merthiolate, containing up to 49% mercury), phenol, coolant (propylene glycol), peanut oil, and of course foreign proteins (antigens), viruses and bacteria or their protein envelopes (such as pertussigen, an active toxic ingredient in all pertussis vaccines, whether whole cell or acellular), to mention just a few of the most common, standard ingredients in a variety of vaccines.

**Vaccination and shaken baby syndrome**

As Dr Innis repeatedly stated in his comments to a variety of BMJ articles on shaken baby syndrome (SBS), all of the SBS cases he studied were vaccinated within 21 days of the appearance of symptoms of SBS or death. I second this with a slightly qualified statement that among some 70-odd cases of SBS for which I have prepared a report, only two were cases of birth injury and were unvaccinated. Also, a few of the SBS babies died more than 21 days after their last vaccinations. Indeed, days 42 to 48 after vaccination represent important critical days with increasing numbers of deaths (as discussed above).

Most of those who have been involved in the study of SIDS or SBS, including those who have participated in the present and very much needed BMJ.com cathartic debate on SBS, have been rather shy or silent about the administered vaccines, even though those vaccine injections are, as a rule, the only documented facts. The act of shaking is undocumented and it is indeed (as Dr Innis correctly states) a little more than a figment of bizarre imagination by the accusing doctors, child protection agencies and police.

Some responders in this debate have questioned whether doctors are out to victimise innocent carers. The simple answer is that they are. As pointed out by Kirschner and Stein (1985): "...the treating physicians in the emergency room mistook life-threatening illness or postmortem artifacts for inflicted injury... Although the histories related by the parents in the emergency room were in all cases truthful and consistent with the results of physical examinations of the child, the involved physicians failed to make a correct diagnosis. Not only lack of experience with severe childhood illness and death but also an attitude of suspicion and/or hostility probably contributed to these misdiagnoses."

So what are the causes and mechanisms of what is considered the pathognomic triad of symptoms by the proponents of SBS, such as subdural and retinal haemorrhages and broken bones?

As I wrote in my previous papers on this subject (J ACNEM 2002; bmj.com Rapid Responses. 2 April 2004: and elsewhere), the whole idea of subdural haematomas and bizarre bone fractures as a result of child abuse was started by Caffey in 1946. He considered fractures in the long bones as a complication of the infantile subdural haematoma associated with the fractures of the cranium. Even though his own illustrations show what is generally considered typical scurvy fractures, he denied any "Roentgen signs of scurvy". Caffey (1946), without going into any more detail, concluded: "The fractures appear to be of traumatic origin but the traumatic episodes and the causal mechanism remain obscure." It is difficult to understand why such classical scurvy fractures as shown in his own photographs were misinterpreted. However, Caffey admitted in his 1965 article, "Significance of the history in the diagnosis of traumatic injury in children", that "it is still a wonder to me that Ross Golden welcomed me, a pediatrician without either formal or informal training or experience in radiology, into his department of traditionally and highly trained expert radiologists". Indeed, why?

The fact remains that Caffey made a mess of things which will take years to rectify. The sooner the rectification begins, the better not only for all those thousands of victims of Caffey's obvious ignorance and closed mindedness but also for those formally trained radiologists who blindly followed misinterpretations of formally untrained Caffey. Moreover, Silverman (1965) attested to Caffey's close mindedness when he wrote about Caffey: "A classic example of his attitude...occurred at the end of a hot discussion at an 11 o'clock conference at Babies Hospital...when he was heard to remark to someone with whom he had been debating a point, 'I wouldn't believe it even if you proved it to me[1]."

Killer (1972), a formally trained Australian radiologist, demonstrated that Caffey's bizarre fractures are in fact caused by scurvy, although he did not explain what actually caused scurvy in the affected babies.

It was Hess (1920) who pointed out in his elegant, almost 300-page tome on scurvy, which was much ahead of its time, the inadequacies of "antiscorbutic" vitamin (vitamin C) content of the usual infant food..

Later on, Pekarek and Rezabek (1959) demonstrated that the administration of DPT vaccine to rats caused them to develop acute scurvy which rectified itself within 24 hours.

However, human babies do not have the advantage the rats have of being able to produce their own vitamin C within their bodies; humans and other primates, fruit bats and guinea pigs, to mention the most important examples, do not produce their own

vitamin C and depend on their food having adequate content of this important, essential vitamin.

When human babies are given the same DPT vaccine as Pekarek and Rezabek's rats, they develop acute scurvy which does not rectify itself unless the babies are given sufficiently large amounts of vitamin C. This, of course, never happens because when babies with vaccine reactions are admitted to hospitals they are given antibiotics instead, further aggravating their vitamin C deficiency.

Scurvy affects all systems in the body. It causes depletion of collagen, resulting in vascular wall fragility, blood clotting and other haematological derangements resulting in bruising; it causes brain, retinal and other organ bleeding and many other malfunctions of all systems of the body, including derangement of the central control of temperature, blood pressure, etc.

Injecting foreign antigens (and other proteins) directly into the bloodstream causes immunological derangements—among others, the reversal of T4 and T8 cells ratio (Jefferys, 2001), which results in the whole cascade of untoward events resulting in death. I am surprised that any babies survive the intense vaccination programs they are subjected to these days. Others have mentioned haemophagocytic lymphohistiocytosis (HLH) as the syndrome which is accompanied by the same symptoms as SBS, without going into details as to what actually causes HLH.

**New diseases for old**

Medicine is known for repeatedly introducing new names for old diseases. It is probably due to the well-known failure of medical researchers to study medical literature (yes, I've heard American medicos bragging in court that they don't study "that stuff", meaning medical research, and in particular the foreign Journals; as a matter of interest, they considered BMJ not worthy of their scientific curiosity). This situation is relevant to the study of subdural and retinal haemorrhages of SBS.

Sparacio et al. (1971) described acute subdural haematoma in infancy. They described six cases in infants aged three months, 10 months, one year, 10 months, six months and nine months, of which two had a documented fall while the rest did not.

Hart and Earle (1975) described haemorrhagic and perivenous encephalitis in a clinical-pathological review of 38 cases. They wrote that haemorrhagic leucoencephalitis (AHL) and post-infectious perivenous encephalitis (PVS) associated with childhood mumps, measles, chickenpox and vaccination are important diseases of the central nervous system.

Graham et al. (1979) described acute haemorrhagic (also known as necrotising) leucoencephalitis as a complication of the generalised Schwartzman reaction which may occur after sensitisation (anaphylaxis) to drugs, such as sulphonamides and para-amino salicylic acid, and which has also followed pertussis vaccination and the administration of the antitetanus serum.

Levin et al. (1983) described haemorrhagic shock and encephalopathy as a new syndrome with a high mortality in young children. Interestingly, the children from whom polio virus was isolated had all been recently vaccinated. This means that other cases could have been vaccinated longer than a few days before developing symptoms of haemorrhagic shock.

In the 1970's and 1980's, a number of authors described so-called haemophagocytic syndrome or lymphohistiocytosis syndrome. The symptoms in haemorrhagic shock and encephalopathy and HLH are very similar; general feeling of malaise, fever, listlessness and vomiting, pallor, tachycardia, tachypnoea, convulsions, low blood pressure, glove and sock syndrome (hot body and cold extremities), distended abdomen, enlarged liver, tense fontanelle, hypotonia, watery, blood-stained diarrhoea, haematemesis, liquid unclotting blood (bleeding from venipuncture sites), deranged coagulation with deranged prothrombin and thromboplastin time, very low fibrinogen and fibrin degradation products very elevated, indicating severe disseminated intravascular coagulation. Other characteristic findings are severe metabolic acidosis (pH less than 7.35 or even less than 7), low bicarbonate, base deficit with compensatory respiratory alkalosis, impaired renal function, raised plasma urea and creatinine and especially hyperglycaemia, indicating central diabetes insipidus, cerebral oedema and internal haemorrhaging into the brain, retina, lungs and other organs, and diffuse macular cutaneous haemorrhages. All organs may be infiltrated with lymphocytes and histiocytes. At necropsy, the brain is oedematous, soft and virtually liquid. More severe cases have meningeal and perivascular infiltration of lymphoid cells in the brain.

Akima and Sumi (1984) described a number of cases of babies aged six months, four months, four-and-a-half months (readmitted at six-and-a-half months and died 11 days after admission), five months (readmitted at eight months and died two months later), six weeks of age with recurrence of symptoms at four-and-a-half months of age (died at five-and-a-half months) and seven weeks (died four days after admission to hospital). All cases clearly developed their symptoms after vaccination, based on their ages at the first admission and the time of readmission.

Some authors have called HLH a familial disease; however, this definition was a reflection of the familial habit of vaccinating all children, rather than some special

familial genetic predisposition other then predisposition to react violently to vaccines (Renter and Blinder, 1991).

Liao and Thompson (1997) described retinal haemorrhages as ophthalmic manifestations of virus-induced haemophagocytic syndrome.

Renter and Blinder (1992) described cerebromeningeal haemophagocytic lymphohistiocytosis as an immunological disorder, and Sperling (1997) described it as a "runaway" immune system.

Rosen (1997) quoted a number of vaccines (vaccinia, polio, measles and BCG) as the causal agents in HLH, and described the disease as a severe, combined immunodeficiency.

Comans-Bitter et al. (1997) described immunotyping of blood lymphocytes in childhood to be used as a yardstick in the diagnosis of haematological and immunological disorders.

Bonilla and Oettgen (1997) analysed the above article and wrote that T cells, B cells and natural killer (NK) cells interact with each other and with L a diverse array of "accessory cells", such as monocyte-derived cells, to generate an immune response. T cells may be identified by the CDS marker associated with the antigen receptor and are further divided into two populations: CD4+ and CD8+.

CD4+ T cells (also known as "cytotoxic" or "suppressor" cells) execute important effector functions such as the lysis of infected host cells (part of the cellular immune response). After interaction with CD4+ T cells, B cells give rise to plasma cells which produce antibodies (the humoral immune response). The NK cells are important in the early phases of immune responses to viruses and malignancy.

Since vaccines derange these elements of the immune system, it is not difficult to understand why they are implicated as causal agents in all those modern ills of children, such as asthma and allergies, a number of cancers, gastrointestinal problems, autism and other behavioural problems to mention just a few so-called new diseases.

In summary, there is a wealth of scientific data to demonstrate that vaccines cause serious derangements of all systems of the body, which result in serious injuries, including deaths—and in babies in particular—being misinterpreted as being caused by inflicted trauma. °°

**About the Author:**

Viera Scheibner, PhD, is a retired principal research scientist with a doctorate in natural sciences. During her distinguished career she has written three books and had over 90 papers published in refereed scientific journals. She has been researching vaccines and vaccinations since the early 1980s and is the author of Vaccination: 100 Years of Orthodox Research Shows that Vaccines Represent a Medical Assault on the System (1993; reviewed in NEXUS 2/16) and Behavioural Problems in Childhood (2000; reviewed in 7/05).

Dr Scheibner's article on the dynamics of critical days was first published in the Journal of the Australasian College of Nutritional & Environmental Medicine (J ACNEM) 23(3):1-5, December 2004.

Previous articles by Dr Scheibner on vaccines and vaccinations have been published in NEXUS: "Adverse Effects of Adjuvants in Vaccines" (NEXUS 8/01-02), "Shaken Baby Syndrome" (5/05); "Brain-eating Bugs" (3/03), and (with Leif Karlsson) "Cot Deaths Linked to Vaccinations" (2/05).

Dr Scheibner is often asked by lawyers to provide expert reports for vaccine-damage court cases, and she regularly conducts lectures. She was a speaker at the 2005 NEXUS Conference in Brisbane in September.

Dr Scheibner can be contacted by mail at 178 Govetts Leap Road, Blackheath, NSW 2785, Australia, by telephone on +61 (0)2 4787 8203 or by fax on +61 (0)2 4787 8988. She is happy to provide additional references for this article as well as accompanying diagrams on request.

**References**

(in order of appearance in text)

• Parventjev IA (1959). Bacterial allergy increases susceptibility to influenza virus in mice. Proc Soc Biol Med 90:373-375.

• Craighead JE (1975). Report of a workshop: disease accentuation after immunization with inactivated microbial vaccines. J Infect Dis 131(6):749-54.

• Daum RS, Sood SK, Osterhholm MT et al. (1989). Decline in serum antibody to the capsule of Haemophilus influenzae type B in the immediate post-immunization period. J Pediatrics 114:742-747.

• Takacs K, Chandler P and Altmann DM (1997). Relapsing and remitting experimental allergic encephalomyelitis: a focused response to the encephalitogenic peptide rather than epitope spread. Eur J Immunology 27:2927-2934.

• Selye H (1978). The Stress of Life. McGill University Press, Montreal.

• Mitchell EA, Stewart AW, Clements M et al. (1995). Immunisation and the sudden infant death syndrome: New Zealand Cot Death Study Group. Arch Dis Child 73:498-501.

• Scheibner V (1991). Evidence of the association between non-specific stress syndrome, DPT injections and cot death. Proc Second National Immunisation Conference, Canberra, 27-29 May 1991, pp. 90-91.

• Bernier RH, Frank JA, Dondero T and Nolan TF(I982). Diphtheria-tetanus-pertussis vaccination and sudden infant deaths in Tennessee. J Pediatrics 105(5):419-421.

• Walker AM, Jick H, Perera DR, Thompson RS, Knauss TA (1987). Diphtheria-tetanus-pertussis immunization and sudden infant death syndrome. Am J Pub Health 77:945-951.

• Coulter HL and Fisher BL (1991). A Shot in the Dark. Avery Publishing Group Inc., NY(246pp).

• Levine S, Wenk EJ, Devlin HB et al. (1966). Hyperacute allergic encephalomyelitis: adjuvant effect of pertussis vaccine and extracts. J Immunology 97(3):363-368.

• Levine S and Sowinski R (1973). Hyperacute allergic encephalomyelitis. Am J Pathology 73:247-260.

• Steinman L, Sriram S, Adelman NE et al. (1982). Murine model for pertussis vaccine encephalopathy: linkage to H-2. Nature 299:738-740.

• Munoz JJ, Arai H, Bergman RK et al. (1981). Biological activities of crystalline pertussigen from Bordetella pertussis. Infection and Immunity 33(3):820-826.

• Hess AF (1920). Scurvy, Past and Present, JB Lippincott Company (279pp).

• Kirschner RH and Stein RJ (1985). The mistaken diagnosis of child abuse. A form of medical abuse? Arch Dis Child 139:873-875.

- Caffey J (1946). Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. Am J Roentgenol & Radium Therapy 56(2):163-173.

- Caffey J (1965). Significance of the history in the diagnosis of traumatic injury in children. / Pediatr 67(5)pt2:1008-1014.

- Silverman FN (1965). Presentation of the John Howland Medal and Award of the American Pediatric Society to Dr John Caffey. J Pediatr 67(5)pt2:1000-1007.

- Hiller HG( 1972). Battered or not - a reappraisal of metaphyseal fragility. Am J Roentgenol & Radium Therapy & Nuclear Med 114(2):241-246.

- Pekarek J and Rezabek K (1959). An endocrinological test for innocuity of the pertussis vaccine. JHyg Epidemiol Microbiol Immunol 3:79-84.

- Jefferys R (2001). T cells and vaccination. Lancet 357:1451.

- Sparacio RR, Khatib R and Cook W (1971). Acute subdural hematoma in infancy. NY State J Med Janl5:212-213.

- Hart MN and Earle KM (1975). Haemorrhagic and perivenous encephalitis: a clinical-pathological review of 38 cases. J Neurol Neurosurg and Psychiatry 38:585-591.

- Graham DI, Behan PO and More IAR (1979). Brain damage complicating septic shock. / Neurol Neurosurg and Psychiatry 42:19-28.

- Levin M, Kay JDS, Gould JD et al. (1983). Haemorrhagic shock and encephalopathy: a new syndrome with a high mortality in young children. Lancet ii:64-67.

- Akima M and Sumi SM (1984). Neuropathology of familial erythrophagocytic lymphohistiocytosis. Six cases and review of literature. Hum Pathology 15:161-168.

- Henter JI and Blinder G (1991). Familial haemophagocytic lymphohistiocytosis. Clinical review based on the findings in seven children. Acta Paediatr Scand 80:269-277.

- Liao PM and Thompson JT (1997). Ophthalmic manifestations of virus-associated hemophagocytic syndrome. Arch Ophthalmol 109:777.

- Henter JI and Blinder G (1992). Cerebromeningeal haemophagocytic lymphohistiocytosis. Lancet 339:104-107.

• Sperling MA (1997). Hemophagocytic lymphohistiocytosis: a lethal disorder of immune regulation. J Pediatr 130(3):337-338.

• Rosen FS (1997). Severe combined immunodeficiency: a pediatric emergency. J Pediatr 130(3):345-356.

• Comans-Bitter WM, de Groot B, van den Beemd R et al. (1997). Immunotyping of blood lymphocytes in childhood. J Pediatr
130:388-393.

• Bonilla FAS and Oettgen HC (1997). Normal ranges for lymphocyte subsets in children. J Pediatr 130(3):347-349.

• Kieslich M, Vecchi M, Laverda AM et al. (2001). Acute encephalopathy as a primary manifestation of haemophagocytic lymphohistiocytosis. Developmental Medicine & Child Neurology 43:555-558.