The Other Side of India’s Polio Eradication Story

- Vratesh Srivastava (November 18th, 2023)

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Summary

Following the 1988 World Health Assembly declaration to eradicate polio by the year 2000, to which India was a signatory, India ran intensive pulse polio immunization campaigns since 1995 [1]. After 19 years, in 2014, polio was declared officially eradicated in India. India was formally acknowledged by WHO as being free of polio [2].

However, an in depth analysis of the India's polio eradication campaign reveals facts that contradict this narrative and call the ethics of the entire vaccination campaign into question. Some of the facts are highlighted below

1. **Using a consistent definition, there's no evidence polio was eradicated:** Using a consistent definition of a polio case, there is no evidence that polio got eradicated. In fact, if one were to go by the traditional way where all acute flaccid paralysis cases used to classify as polio, polio cases actually skyrocketed as the vaccination programs intensified. However, India opted for WHO's criteria for diagnosing polio since 1997, which led to a change in definition of polio and consequently a drastic drop in cases of polio, despite a sustained increase in cases of acute flaccid paralysis which pre-1997 used to be classified as polio. [1, 3, 4]
2. **The entire immunization programme was an exercise in eliminating wild polio viruses from stool specimens, not reducing incidence of acute flaccid paralysis**: While the mainstream narrative indicated that the vaccine was reducing incidence of paralysis from polio, Dr. T. Jacob John, chairman of the polio eradication committee, made it clear that the aim was to only eradicate wild viruses from stool specimens, not the incidence of acute flaccid paralysis. [5]
3. **Deliberate Violation of informed consent**: It was known by members of the polio eradication committee that the vaccine itself can cause polio - a condition called VAPP or vaccine associated paralytic poliomyelitis. This known side effect was deliberately hidden from the public and parents of the vaccinated children, with full knowledge of WHO and UNICEF. Worse, polio induced by the vaccine was paradoxically classified as non-polio. [**6**, **7**, **1**, **8**, **9**]
4. **Coercion to vaccinate:**There were reports of coercion where families were threatened with power cuts and no ration if they refused to vaccinate their child. [**7**]
5. **Several red flags about vaccine failure**:A disproportionately high number of polio cases were vaccinated. There was no effort made to study in detail the cause of vaccine failure and vaccine induced polio [**7**, **4**, **10**]. A study by Dr. Pulliyel et al.actually indicated a strong association between rise in cases of acute flaccid paralysis and the polio campaign. [**11**]. Similar observation was made in another study of 9 AFP cases, where children had received upto an astonishing 25 doses. [**12**]
6. **Indiscriminate repeated vaccination with no safety studies:** While the original dosage recommendation for the polio vaccine had been 3 doses, in India's case it steadily increased to 7 and then to theoretically 10 doses [**13**, **9**]. It was reported that 70 million children received 10 doses a year [**14]**. Many children received upto 25 doses, prompting concerns of safety from the Indian Medical Association which was ignored by the government. [**15**]
7. **Poor surveillance of side effects implies that there was likely an incalculable number of serious adverse events**:India has had poor post marketing surveillance of adverse events and it is difficult to gauge the entire spectrum of adverse events from vaccinations [**17**]. As recently as 2018, a former member of NTAGI (National Technical Advisory Group on Immunization) acknowledged that AEFI (Adverse Events Following Immunization) in India was less than

adequate and adverse events underreported as the AEFI committee sat only 4 times a year and analyzed 100 cases at a time, a very small number given the thousands of adverse events reported annually [**16**]. There were reports of children getting brain stroke, going blind and even dying post the vaccination. [**18**, **19**, **20**]

8. **Lack of justice for vaccine victims:** While adverse effects like vaccine induced polio were deliberately hidden, there was no compensation scheme in place. In matters where victims went to court and were able to successfully fight for compensation, the settlement in some of such cases took 10 to 25 years. [**21**, **22**]

In short, what actually happened on the ground seems to completely contradict what has been disseminated and publicized through mainstream media. One can argue with the above facts that the entire polio vaccination programme is mired in controversy and unscientific principles that actually led to incalculable suffering and misery.

1. Using a consistent definition, there's no evidence polio was eradicated

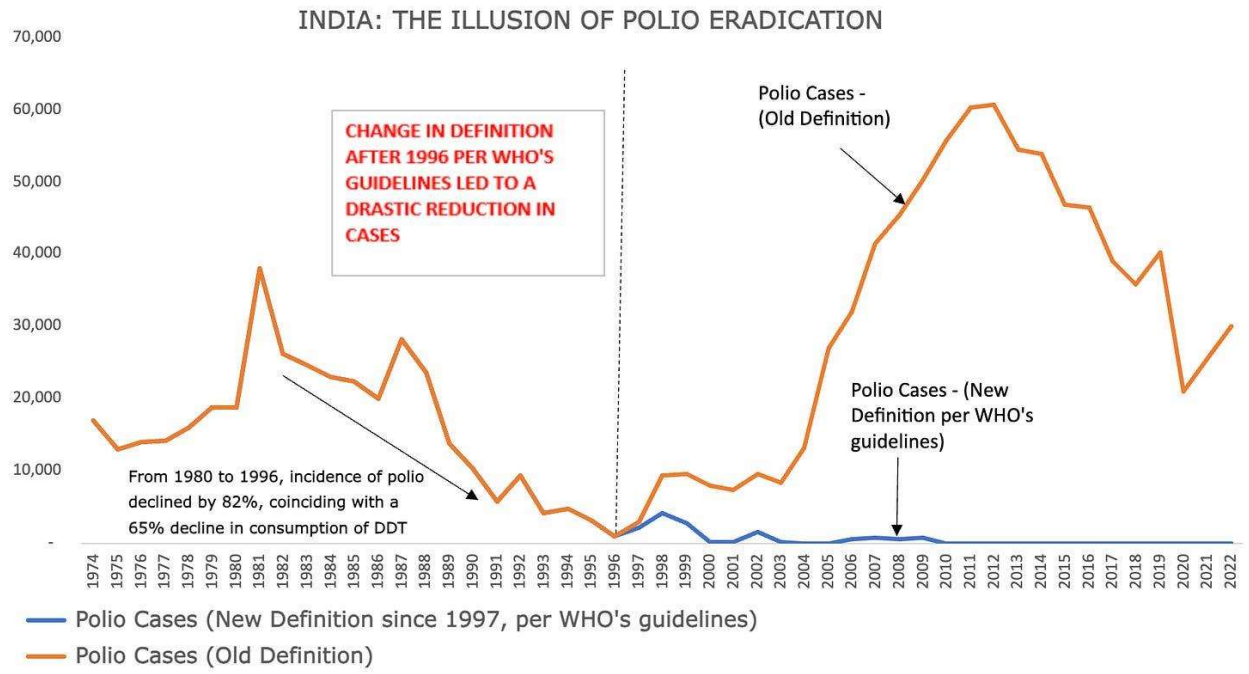
1.1 Comparison of Polio Incidence using old and new definitions

Using a consistent definition of a polio case, there is no evidence that polio got eradicated. In fact, if one were to go by the traditional way where all acute flaccid paralysis cases were classified as polio, polio cases actually skyrocketed as the vaccination programs intensified. However, India opted for WHO's criteria for diagnosing polio since 1997, which led to a change in definition of polio and consequently a drastic drop in cases of polio, despite a sustained increase in cases of acute flaccid paralysis (AFP) which pre-1997 used to be classified as polio. [1, 3, 4]

*"**Upto 1996 all reported cases of acute flaccid paralysis (AFP) were labelled as polio cases**" - Dr. Yash Paul, former member of India's Polio Eradication Committee [1]*

*"**All polio cases reported before 1997 were confirmed by attending physicians with no standard case definition.**" - CDC [23]*

As can be seen in the chart below, polio was actually on the decline before the introduction of pulse polio immunizations in 1995. This decline correlated well with the decline in use of DDT (*details in section 1.3)*. Subsequently, the recorded cases of acute flaccid paralysis (or polio per the pre-1997 classification) skyrocketed, a strong signal raising the question if the vaccination programme was responsible for much of this rise. [26, 27, 1, 28, 29]



Polio Cases in India - Old Definition and New Definition

Based on published research, it can be argued that many of the case of acute flaccid paralysis were triggered by the vaccine itself. Oral Polio vaccine can trigger polio itself (called vaccine associated paralytic poliomyelitis or VAPP) [3] and other so called "non-polio acute flaccid paralysis" cases such as Guillain-Barré syndrome (GBS), transverse myelitis and facial paralysis. Supplements 1,2 and 3 highlight literature from the early to mid 20th century, primarily before the widespread use of vaccines, about the classification of GBS, transverse myelitis and Bell's palsy as different types of poliomyelitis.

"Molecular characterization of polio virus isolated from paralysis cases of GBS, transverse myelitis and facial paralysis have confirmed the vaccine origin of the strain and demonstrated mutation known to increase neurovirulence. ***This suggests that the Sabin vaccine–derived poliovirus strains could also trigger such diseases****" - Journal of Indian Pediatrics (2006) [24]*

""The evidence favors acceptance of a ***causal relation between OPV (oral polio vaccine) and GBS.****" - Vaccine Safety Committee, Institute of Medicine, US [25]*

Pre-1997, 2 things that stand out as far as incidence of poliomyelitis is concerned are - (1) intramuscular injections and (2) DDT consumption

1.2 Pre-1997, 67% of poliomyelitis cases were triggered by intramuscular injections

Going through the literature of poliomyelitis outbreaks before the change in definition, it is clear that close to 67% of the cases were triggered by intramuscular injections. Since, 1997, it appears that many such cases were labeled "non-polio acute flaccid paralysis" (NPAFP) under various headings such as *"traumatic neuritis", "sciatic nerve mononeuropathy", "Post injection Palsy" etc.,* especially where stool specimens were unavailable, inadequate or tested negative.

- "*In India, where poliovirus infection is still widespread, our findings suggest that three-quarters of cases of paralytic poliomyelitis in the past decade were caused or made more severe by unnecessary injections.*" (Provocation Paralysis writeup in Lancet, 1993)[*39*]
- "*Provocation of poliomyelitis occurred in 66% of children and usually followed intragluteal injections associated with treatment of non-specific fevers*" (study of over 12,800 cases of polio in Pondicherry, India, 1989) [*40*]
- "*Two thirds of the affected children had received injections in one or both sides of the gluteal region.*" (1987 study of children admitted to a hospital in Chennai, Tamil Nadu) [*41*]
- "*all those with paralysis gave a history of injections into the gluteal region or thigh during the pre-paralyticstage*." (a 1993 study from Rohtak, Haryana) [*42*]
- "*Intramuscular injection was given in 70% (of cases) within one month of onset of paralysis*" (1989 study from Chennai, Tamil Nadu) [*43*]
- 5 out of 7 children with paralytic poliomyelitis in this study about outbreak of polio in an orphanage in Delhi in 1992, suffered the condition following DPT injections. [44]
- "*Onset of paralysis was noticed in 73.5% of the children following administration of intramuscular injection on the same limb*" (1994 study of poliomyelitis study in Andhra Pradesh) [*45*]
- "*60% had a history of intramuscular injections preceding paralysis*" (1991 study of 37 cases in Haryana) [*46*]

- "*The relative risk of giving injection during the prodromal stage in provoking paralysis was found to be 18, 10 and 13, by matched and triplet analysis respectively*" (1994 field study from Chennai, India) [*47]*

Since 1997, below are snippets from journals/writeups that indicate that paralysis following injections were classified as non-polio AFP

- *27 of the 45 AFP cases investigated in Chhatisgarh per this study in 2003 were classified as "post injection palsy [48]*
- Per this study in 2003 from New Delhi 12-14% of cases of AFP were *"traumatic sciatic neuritis following intra gluteal injections" [49]*
- Per this 1998 writeup ***"post-injection sciatic nerve mononeuropathies"*** was one of the 3 most common causes of Acute Flaccid Paralysis [50]
- Per this 2005 writeup - *"It is essential that cases of polio are not misdiagnosed as traumatic neuritis" [51]*

1.3 The DDT polio connection

"Some evidence exists that ***pesticides*** can cause deformities of limbs and visual disabilities in humans even when taken in low doses". [68]

References to the connection and possible causal association between pesticides like DDT and polio can be found here[62] and here [61]

"in the works of Ralph Scobey, MD,"…. "from ancient times to the early 20th century, the symptoms and physiology of paralytic poliomyelitis were often described as the results of poisoning. It wasn't until the mid-19th century that the word "poliomyelitis" became the designation for the paralytic effects of both severe poisoning and polio-like diseases assumed to be germ-caused"

"McCormick , Scobey, and Goddard, in detailed studies, have all pointed out that factors other than infective agents are certainly involved in the etiology of polio, varying from nutritional defects to a variety of poisons which affect the nervous system." [105]

With reference to India, it is noticeable that while DDT consumption was steady in the 70s and 80s, the use of DDT in agriculture peaked at 4,700 tones in 1978 and then declined to 0 in the 90s, following ban of its use on agriculture in 1989. Overall use of DDT, which includes indoor spraying, declined from 12,500 tonnes in 1980 to 4,400 tonnes in 1996. It is important to note that the major environmental impact of DDT is in agriculture. It is estimated that DDT spraying has only 0.04% of the environmental impact compared to use of DDT in agriculture. This accentuates the ban of DDT in agriculture in 1989 and a plausible hypothesis can be made that ban of DDT in agriculture accelerated the decline of polio incidence in India in the early 1990s. The decline in polio cases in India from 1980 to 1996 of about 82% correlates well with overall decline of about 65% in DDT usage but more so with the decline in agricultural use (from a peak of 4,700 tons in 1978 to 0 in 1996). [63, 64, 65, 66, 67]

Type of use	1960	1966	1970	1975	1976	1977	1978	1979	1980	1984	1994**	1996**
Public health	21.0	2.7	6.2	7.3	7.3	9.0	6.8	6.5	8.5	12.0	4.3	4.4
Agriculture	0.6	2.4	2.4	2.5	1.3	2.5	4.7	4.2	4.0	2.0	0	0

DDT Consumption in India (***1994 and 1996 are production numbers, however it's reasonable to infer that India's consumption was close to its production numbers as by 1994 India was producing almost all the pesticides it consumed. DDT consumption in agriculture was banned in 1989)*

2. Polio eradication was not about reducing incidence of acute flaccid paralysis

2.1 The inadequacy of virological tests in diagnosing poliomyelitis

While the mainstream narrative indicated that the vaccine was reducing incidence of paralysis from polio, Dr. T. Jacob John, chairman of the polio eradication committee, made it clear that the aim was to only eradicate wild viruses from stool specimens, not the incidence of acute flaccid paralysis.

*""**Indeed it is not AFP (acute flaccid paralysis) that is under eradication, but wild polioviruses. Thus the criterion of eradication is the absence of wild polioviruses in stools**" - Dr. T. Jacob John, Former Chaiman, Polio Eradication Committee, Indian Academy of Pediatrics [30]*

What should one make of a statement like the above? There are tens of thousands of acute flaccid paralysis cases every year, but just because wild polioviruses are not detected in stools, such cases are discarded as non-polio? This was not the case pre-1997. Clinical diagnosis took precedence over virological examination and AFP cases were classified as polio per Dr. Yash Paul.

Moreover, it's important to note that virological examination is alone does not negate a clinical diagnosis. This observation was made in the 4th international poliomyelitis conference in 1958. [31]

*"**Failure to isolate the virus does not necessarily negate a clinical diagnosis**" - Dr. Edwin Lennette as quoted in his paper "Problems of Viral Diagnostic Laboratory with Respect to Poliomyelitis"*

*"**Failure to isolate polio virus from a patient exhibiting a clinical syndrome compatible with poliomyelitis does not rule out the diagnosis**" Dr. Albert Sabin, credited as inventor of the oral polio vaccine, as quoted from his paper "Discussion - Isolation of Viruses"*

Below are a few more examples that highlight why simply testing stool specimens can significantly understate case counts of paralytic polio.

- Per a study in Texas, only 26% of suspected cases were confirmed polio cases through laboratory stool test. Historically, clinical diagnosis would've meant all such cases be diagnosed as polio, but with the new definition "confirming diagnosis", only 26% of such cases would count as polio. [32]

TABLE 3.—*Comparative Value of Stool Culture and Serum Complement Fixation Test in Confirmation of Diagnosis of Poliomyelitis.*

No. of Cases Studied by Both Techniques	Poliovirus Type on Basis of Stool Culture			Poliovirus Type on Basis of Complement Fixation Test		
	I	II	III	I	II	III
227	53	2	3	56	3	4

- Only 14.8% of diagnosed polio or polio like illnesses were laboratory confirmed as polio per this study in Washington state in 1956 [33]

Table 3.

Final Diagnosis	Total	Poliovirus Recovered		Non-poliovirus Recovered		No Virus Recovered	
		Cases	%	Cases	%	Cases	%
Non-Paralytic Polio	8	8	100.0	0	0	0	0
Paralytic Polio	54	29	53.7	2	3.7	23	42.6
Aseptic Meningitis	160	0	—	83	51.9	77	48.1
Other Diseases	28	0	—	3	10.8	25	89.2
Totals	250	37	14.8	88	35.2	125	50.0

- Only 39 out of 458 cases were confirmed polio cases in 1957 per a Michigan study in 1957. [34]

than three doses.[8] Furthermore, specimens from many cases reported as poliomyelitis, especially nonparalytic cases, have been subjected to laboratory examination with the result that poliomyelitis was ruled out either by failure to isolate the agent or by the actual isolation of other causative organisms. As an example, stool specimens from 458 cases diagnosed clinically in Michigan during 1957 have been processed in the Virus Laboratory of the School of Public Health and poliomyelitis was confirmed in only thirty-nine.

- In Nepal, the AFP (acute flaccid paralysis) rate went up more than 3 fold in 1999 compared to 1998, still the poliomyelities cases dropped by 40%. How was this achieved? Simply by

increasing and testing the number of stool specimens. In 1999, stool specimens of 79% of the AFP cases were collected, while in 1998 only 35% of the stool specimens were collected. Higher the % of stool specimens collected, lower the number of laboratory confirmed cases of poliomyelitis. [**35**]

Table 1

Note: To print large tables and graphs users may have to change their printer settings to landscape and use a small font size.

TABLE 1. Performance indicators for acute flaccid paralysis (AFP) surveillance -- Nepal, 1996-1999

Indicator	Target	1996	1997	1998	1999*
Total AFP rate†	--	0.18	0.40	0.74	2.41
Nonpolio AFP rate§	>=1	0.2	0.26	0.41	1.60
Two stool specimens¶	>=80%	7%	33%	35%	79%
60-day follow-up	>=80%	47%	75%	100%	88%
Total poliomyelitis cases**	--	9	12	31	18
Wild poliovirus	--	1	1	0	0

* Annualized as of September 15, 1999.
† Total poliomyelitis cases + nonpolio AFP cases + cases pending classification per 100,000 children aged <15 years.
§ Number of nonpolio AFP cases per 100,000 children aged <15 years.
¶ Two stool samples collected within 14 days of paralysis onset.
** Nepal uses the World Health Organization clinical classification system.

In Nepal, the AFP (acute flaccid paralysis) rate went up more than 3 fold in 1999 compared to 1998, still the poliomyelities cases dropped by 40%. How was this achieved? Simply by increasing and testing the number of stool specimens. In 1999, stool specimens of 79% of the AFP cases were collected, while in 1998 only 35% of the stool specimens were collected

- In the paper titled "Problems of the Viral Diagnostic Laboratory with Respect to Poliomyelitis" during the 4th International Poliomyelitis Conference in 1958, Dr. Edwin H. Lennette observed that polio viruses were isolated from stool specimens from only 36% of the cases clinically diagnosed as polio. No viruses (polio viruses or other enteroviruses) were detected in 47% of the specimens. It also observed that a "restricted diagnostic service" was introduced in May 1955, a month after Salk's vaccine was licensed **[31]**

TABLE 130. VIRUS ISOLATIONS FROM PATIENTS WITH CLINICAL DIAGNOSIS OF POLIOMYELITIS

VIRUS RECOVERED	PROPORTION VIRUS RECOVERIES FROM PATIENTS DIAGNOSED AS: PARALYTIC POLIO		NONPARALYTIC POLIO		TOTAL PATIENTS	
	NUMBER	PER CENT	NUMBER	PER CENT	NUMBER	PER CENT
Polio Virus						
Type 1	173	49%	47	13%	220	31%
Type 2	4	1%	4	1%	8	1%
Type 3	29	8%	3	1%	32	4%
Types 1 and 2 and Coxsackie B-5 mixed*	1	+%	—	—	1	+%
Other Virus						
Orphan	18	5%	102	28%	120	17%
Negative	187	37%	212	57%	339	47%
Totals	352	100%	368	100%	720	100%

Table includes all patients on whom laboratory work was complete as of March 15, 1957.
* All 3 agents recovered from single stool specimen.

2.2 Viruses the sole causative agents of poliomyelitis?

An article in 1937 in the **Journal of the American Osteopathic Association raised skepticism about the virus origin theory of poliomyelitis** *(the theory on the basis of which the first polio vaccines of Kolmer and Broie had been made available in 1935) [*59*]*

Snippets:

"Contrary to what is commonly believed, ***poliomyelitis is rather infrequent in crowded districts and among children who frequent crowded places*** *such as schools, churches, theaters, etc."*

"Despite the tremendous amount of research aimed at the understanding of the causative factors underlying anterior poliomyelitis, we are today still in a quandary as to the true etiology. ***Even the most widely accepted cause has not been universally acknowledged. The virus theory is the basis for most official investigations, but it is subject to much questioning.****"*

"Most investigators agree thus far that the agent is a filtrable virus, but ***controversy wages as to 2 how the virus enters the human body and why.****"*

*"****The exact manner of the spread of the disease is not known****,"*

*"****the disease has spread despite the most rigid isolation****."*

As Scobey observed in one of his writeups - ***"There is evidence that poliomyelitis has many causes rather than a single cause"*** [60]

Based on the evidence presented in this section, the question needs to be asked if viruses are the sole cause of poliomyelitis, or even if they are a necessary condition for the manifestation of disease we know as poliomyelitis. If it is, why was the virus detected in such a small % of stool specimens? Why does poliomyelitis incidence correlate so strongly with DDT usage? Pre-1997, why did provocation by injection comprise almost 67% of the poliomyelitis cases?

It is clear that absence of wild polio viruses from stool specimens does not negate a diagnosis of polio given symptoms of acute flaccid paralysis, but this fact has been ignored and people were misled to believe that eradication of polio is the same as eradication of acute flaccid paralysis, whereas in reality the acute flaccid paralysis cases continue to number in thousands every year. What has been reported from surveillance information is that wild polioviruses are no longer detected in stool specimens. However this mere fact means little given the thousands of AFP cases, which pre-1997 were labelled polio, continue to be reported every year.

3. Violation of informed consent and coercion to vaccinate

Polio is a side effect of the polio vaccine. This condition is called Vaccine Associated Paralytic Poliomyelitis or VAPP. Not just the recipient but close contacts of the recipient are at risk of contracting polio. This fact was deliberately hidden from the masses per Dr. Yash Paul - former member of Polio Eradication Committee of Indian Academy of Pediatrics. [1, 3]

Snippets:

"In a publication entitled 'Together we make India polio free' produced jointly by the Indian Academy of Pediatrics and UNICEF following a workshop held in New Delhi on May 20-21, 2000 on page 14 under Issues and Concerns it was stated: (i) Public discussion of VAPP may cause serious damage to credibility of the polio eradication strategy (ii) In the present scheme ***VAPP is discarded as non-polio, although they are the unwanted product of polio programme****, (iii) Epidemiological, clinical and laboratory investigation of VAPP is not carried out. Thus* ***not only information regarding possible harm to a child by OPV was being held back, but facts regarding the occurrence of VAPP were being suppressed.****"*

*"****It was known that OPV can cause VAPP, but during pulse polio immunization program people were told that OPV is absolutely safe, which was not the truth****."*

"People were also told through media that OPV is highly effective, although children were developing polio despite taking large number of polio doses"

"No studies regarding the incidence of VAPP cases occurring in India were available"

*"This" (VAPP) "was considered a 'price' to be paid for polio eradication and t****he information was guarded as a secret from the public****, because doctors had been advised to restriction the discussion regarding VAPP to academic circles only, so that the pulse polio immunisation may not be affected"*

Snippets from the article "Some ethical issues arising from the polio eradication programmes in India" make the below amply clear [6]

- Parents were deliberately not informed of risks of VAPP
- The lie that OPV is safe was deliberate to induce maximum participation
- WHO's guidelines made no mention of informed consent, only a one sided view of "benefits"

Snippets:

"advocates for the programme present a one-sided promotion of OPV vaccination"

"This approach is in line with WHO documents about polio which focus on the importance of 'advocacy' or the promotion of vaccination. ***They make no mention of informed consent****"*

*"****Regarding the message that OPV is absolutely safe, we do know that it is not****. However, I will not suggest that the public be alarmed by the very small risk of vaccine associated polio, a price we have accepted to pay for the control and eradication of wild polioviruses"*

"Dr. Vipin M. Vashishtha, co-convener of the Polio Eradication Committee of the Indian Academy of Pediatrics added that 'we can dare to disclose the true figures of VAPP only if we have an alternate strategy in place to implement without delay.'"

*"The question to be asked is whether this **sacrifice of parental autonomy**, due to the absence of informed consent, is worth making for the greater good?"*

*"if it is indeed to be accepted that the benefits of polio eradication outweigh the **withholding of information about the risks of harm**, then, at the very least, an adequate compensation scheme needs to be in place for those that are harmed as a result of the programme. Children (through their parents) should be eligible for compensation if they develop VAPP, as either a recipient or contact case, or if they develop polio and are handicapped or die despite being fully vaccinated"*

The below statement is from 2005, in an interaction between 2 members of the polio eradication committee,10 years after pulse polio immunization started, and decades after OPV was made available in India. What should one make of a question like this coming from a member/former member of the polio eradication committee? Implicit in this question is that parents were knowingly not informed of the risks of VAPP Also read the convoluted response (which acknowledges that parents have not been informed about the risk of VAPP) [36]

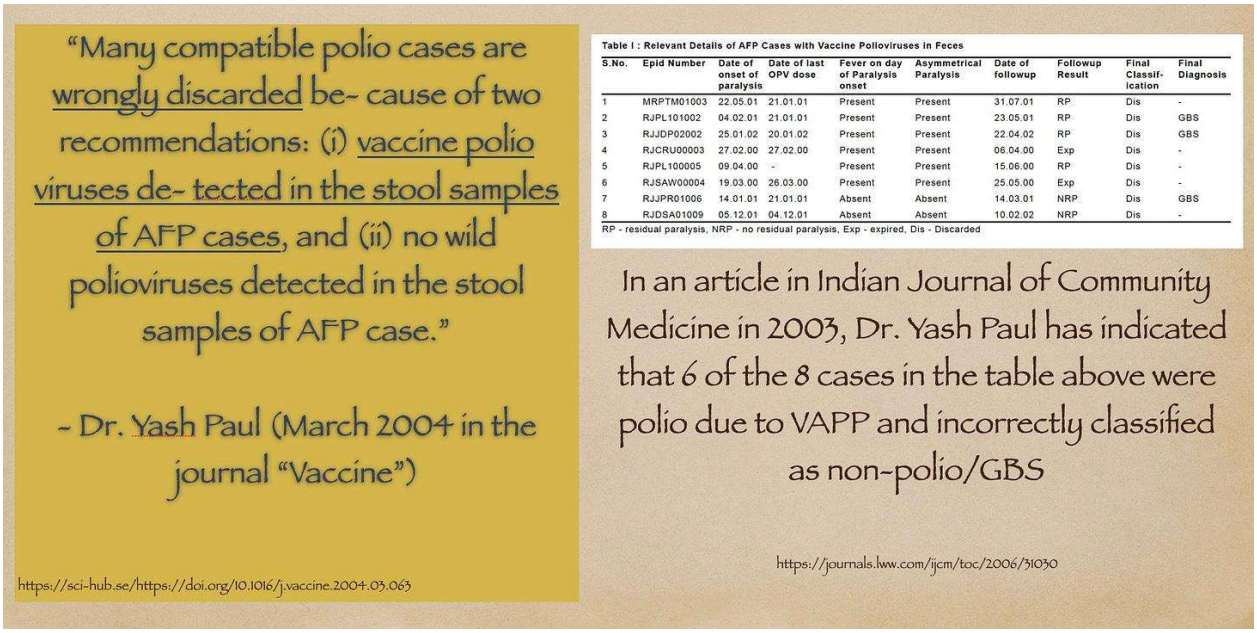
*"**Should the parents be told the truth if a child happens to develop VAPP**?"*

*"In the West, vaccine-associated paralytic poliomyelitis (VAPP) in **vaccine-recipients and contacts** had been identified"*

*"**ethical problem** that had been **ignored by the silent majority**"* [9]

The above statement was made by T Jacob John, chairman of polio eradication committee. He uses the term **"silent majority"** clearly implying majority of those involved with designing the polio immunization strategy knew polio vaccines can cause polio, and this known side effect was deliberately hidden i.e they were silent about it.

The illogical WHO backed classification of VAPP as "non-polio" was contested by pediatrician Dr. Yash Paul in 2 separate writeups. [37, 38]



Despite the glaringly obvious violation of informed consent, what added fuel to the fire was coercion to vaccinate. There were reports of punitive measures being employed against parents who refused vaccination. Per an article in Hindustan Times, power supply was cut and ration withheld after a parent refused vaccination. [3]

"A news item published in Hindustan Times, New Delhi edition dated August 14, 2007 on page 9 under caption 'Refuse polio drops, lose power and ration cards' stated that "sub-divisional magistrate Raghuvir Yadav ***ordered immediate disconnection of power supply to the house of one Hafiz in Manechha village as well as cancellation of his ration card. Hafiz had refused to let his children be immunized on August 10. The same punishment was meted out to Ayub of Sabarhad****. Other people were warned of similar action"*

4. Several red flags about vaccine failure ignored?

A disproportionately high number of polio cases were vaccinated. There was no effort made to study in detail the cause of vaccine failure and vaccine induced polio [**7**, **4**, **10**].

In 2007 to 2009, when coverage of 3 doses or more was 67%-73%, an astonishing 96% of the reported polio cases had received 4 or more doses of the vaccine. In 2007, an unbelievable 85% of the cases had received more than 7 doses.

OPV Doses	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009
0 Dose	15%	14%	14%	9%	16%	14%	4%	0%	3%	1%	1%	1%
1 - 3 doses	47%	45%	28%	31%	41%	35%	11%	11%	10%	3%	3%	4%
4 - 7 doses	32%	34%	35%	41%	33%	34%	41%	44%	22%	12%	18%	18%
> 7 dose	7%	8%	23%	18%	11%	17%	44%	45%	65%	85%	78%	77%

Table 1: Number of OPV doses received by polio cases, 1998-2009.

A study by Dr. Pulliyel et al.actually indicated a strong association between rise in cases of acute flaccid paralysis and the polio campaign. [**11**].

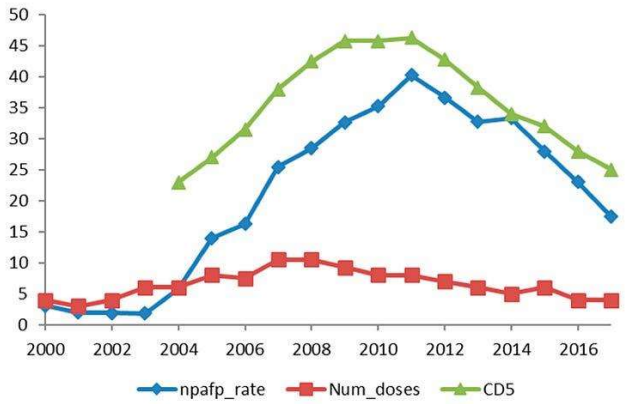


Figure 2. Non-polio AFP over the years in the state of Bihar alongside the 5-year cumulative doses of OPV. npafp_rate: Non-polio acute flaccid paralysis rate; Num_doses: Number of pulse polio rounds; CD5: Cumulative doses in the past 5 years.

Similar observation was made in another study of 9 AFP cases, where children had received upto an astonishing 25 doses. [**12**]

Table 2 Characteristics of non-polio AFP cases in the study

From: Support for children identified with acute flaccid paralysis under the global polio eradication programme in Uttar Pradesh, India: a qualitative study

	Case Number								
	1	2	3	4	5	6	7	8	9
Gender	F	M	M	M	M	F	M	M	M
Age at interview	2 yr.	1.5 yr.	1.9 yr.	4 yr.	8 m	4.5 yr.	3 yr	5 yr.	6 yr.
Age at onset	1.5 yr.	1.9 yr.	6.3 yr.	3.5 yr.	5 m	4 yr.	3 yr.	4.3 yr.	6 yr.
Residual paralysis	No	Yes	No	No	No	No	No	Yes	No
OPV doses received before	12	15	25	21	NA	20	15	25	20
Religion	Muslim	Muslim	Muslim	Muslim	Muslim	Muslim	Hindu	Hindu	Hindu
Occupational of parents	Furniture shop	tailor	driver	labourer	labourer	labourer	Sugar mill employee	labourer	labourer
Literacy of parents	literate	illiterate	illiterate	literate	illiterate	illiterate	literate	illiterate	illiterate

* Data retrieved from medical records of each patient
** NA: not available

Was the below statement by Dr. T. Jacob John, chairman of polio eradication committee, a veiled acknowledgement that the vaccination campaign was a failure?

"*All those who ignored ethics and scientific evidence in the past …**have realised their mistakes when it is too late to correct them***." [9]

5. Indiscriminate repeated vaccination with no safety studies

While the original dosage recommendation for the polio vaccine had been 3 doses, in India's case it steadily increased to 7 and then to theoretically 10 doses [**13**, **9**]. It was reported that 70 million children received 10 doses a year [**14]**. Many children received upto 25 doses, prompting concerns of safety from the Indian Medical Association which was ignored by the government. [**15**]

"*An earlier estimate of **seven doses of OPV may be necessary,** with high coverage to achieve eradication*." - Study, ICMR centre of virology

"*If the question is whether wild viruses can be eradicated by the tactical use of OPV, the answer is 'yes'. To achieve that, **near 100 per cent coverage with an average of 10 doses per child will be required.** Where the vaccination infrastructure is weak, this is best achieved through repeated pulse vaccination campaigns*"

It is hard to find any safety studies associated with this recommendation for increased dosage. It appears as if the sole purpose was to boost the efficacy through repeated revaccination, with little knowledge of unintended consequences.

6. An incalculable number of serious adverse events? Justice elusive for vaccine victims?

India has had poor post marketing surveillance of adverse events and it is difficult to gauge the entire spectrum of adverse events from vaccinations [**17**]. As recently as 2018, a former member of NTAGI acknowledged that AEFI in India was less than adequate and adverse events underreported as the AEFI committee sat only 4 times a year and analyzed 100 cases at a time, a very small number given the thousands of adverse events reported annually [**16**]. There were reports of children getting brain stroke, going blind and even dying post the vaccination. [**18**, **19**, **20**]

INDIA

Baby dies after polio immunisation in TN

A seven-month old girl child died hours after being administered oral polio drops but health department officials said the death was not due to the vaccination.

Media video report of child losing eyesight following polio vaccination (2019, West Bengal)
https://rumble.com/v3wl3c4-child-lost-eyesight-following-polio-vaccination-with-english-subtitles.html

Media video report of a father looking to sell his kidney to raise funds for treatment of his child who suffered brain stroke following polio vaccination (2019, Assam)

https://rumble.com/v3wl3ly-father-looking-to-sell-kidney-to-raise-funds-for-vaccine-injured-child-with.html

Media report of a child suffering acute flaccid paralysis following polio vaccination (2023, Bihar)

https://rumble.com/v3wl427-child-suffering-acute-flaccid-paralysis-following-polio-vaccination-with-en.html

While adverse effects like vaccine induced polio were deliberately hidden, there was no compensation scheme in place. In matters where victims went to court and were able to successfully fight for compensation, the settlement of some of such cases took 10 to 25 years. [**21**, **22**] (screenshots below)

Rajgarh: He became disabled after drinking polio medicine, after 25 years compensation of Rs 42 lakh was ordered.

10 yrs after side effects of polio drop kills infant,court orders govt to pay Rs 2 lakh to family

Written by Utkarsh Anand | New Delhi | January 5, 2009 2:22 am

7. Is IPV the answer?

Should oral polio vaccine (OPV) be replaced with the injectable polio vaccine (IPV)? The answer is **No**. For 3 reasons - scientific, financial and ethical.

The scientific case against IPV: There was enough information available in the late 1950s, early 60s itself to suggest that IPV (also called Salk vaccine) was ineffective or had low effectiveness, did not prevent transmission, and could potentially cause paralysis. In a Congressional hearing in the US, it was recommended that oral polio vaccines be administered regardless of number of injectable polio vaccines received. So while the oral polio vaccine was recommended back then due to limitations of the Salk vaccine (IPV), things have come back a full circle, with IPV being recommended due to limitations of the oral polio vaccine! [53, 54, 55] IPV is used in much of the developed world today as it is perceived to be a "safer" vaccine, but again its usage is questionable given its history.

"The extensive use of the Salk inactivated vaccine forpoliomyelitis control during the last few years has not eliminated the danger of paralytic forms of the disease developing in triply vaccinated children and has had no effect on the circulation of the virus among vaccinated children. The limited duration of post-vaccinal immunity has made re-immunization necessary after completion of the schedule of three vaccinations with the inactivated vaccine." (Bulletin of the World Health Organization, 1955)

"Once a live, oral vaccine is fully approved, it will be more effective than the killed Salk vaccine. Because of the doubt about the potency and effectiveness of the Salk vaccine in the past, a full course of the new vaccine will undoubtedly be recommended for everyone, regardless of how many Salk shots each individual has had." [52]

It is also important to note that, contrary to popular belief that polio vaccines were introduced in the 1950s, the first polio vaccines made available were in the 1930s. In the mid-1930s, 2 separate polio vaccines were made available in US, one by Dr. Maurice Brodie (killed virus) and the other by Dr. John Kolmer (attenuated). Both vaccines were discontinued following reports of 12 cases of paralysis following vaccination, including 6 deaths. [56, 57, 58]

The ethical case against IPV: Many of the entities recommending IPV are the very same entities who encouraged adoption of OPV and deliberately hid information that can be construed as violation of informed consent. Given the incalculable harm caused by OPV over close to 3 decades, why should these entities be entrusted with anything to do with public health?

The financial case against IPV: The OPV programme between 1994-2012 cost 2.5 billion USD per Dr. Pulliyel. The terrible health tragedy notwithstanding, this sum also represents an enormous waste of the country's financial resources. Would it be justified to continue spending on this programme, given everything highlighted in this paper with regards to the failure of the oral polio vaccination programme, as well as the ethical and scientific case against IPV?

Conclusion

What actually happened on the ground seems to completely contradict what has been disseminated and publicized through mainstream media. One can argue based on the facts presented in this article that the entire polio vaccination programme is mired in controversy and unscientific principles that actually led to incalculable suffering and misery. The only conclusion one can arrive at, therefore is to end all polio vaccination programmes at a minimum, with a roadmap for justice for those harmed by this programme.

Supplement 1 - Bell's palsy and Polio

In an article in the Journal of American Medical Association in 1941, poliomyelitis was classified into 8 types, of which **type VI was labeled as facial paralysis typical of "Bell's Palsy findings". [**69**]**

TABLE 2.—*Differential Signs and Symptoms*

	Type	Signs and Symptoms Plus Positive Spinal Fluid Findings
I	Abortive and preparalytic	Any one or more of the following: headache, fever 2-5 days lasting 4 days or more, stiff neck, pain along the spine, nausea, vomiting, gastrointestinal signs, diarrhea, constipation, pain and cramps, restlessness, sore throat (not common)
II	Peripheral neuritis	Typical symptoms of peripheral neuritis over the skin, chiefly paresthesias (not to be confused with segmental pain of central origin occurring in muscles)
III	Meningeal	All symptoms of type I, abortive and preparalytic, plus meningeal signs (mentioned in text), positive Kernig, etc.
IV	Spinal	All signs of type I, abortive and preparalytic, and perhaps type III, meningeal; occasional convulsion, delirium, plus segmental pain in the muscles and tendons (not skin hyperesthesia), sweating, irritability, signs of a lower motor neuron lesion, athetoid movements, froglike position in bed; if areas from the second, third and fourth cervical segments are involved, there will be signs of difficult breathing, and suffocation may follow; there may be difficulty in urination and in defecation
V	Bulbar	All signs of type I, abortive and preparalytic, perhaps type III, meningeal, and usually type IV, spinal, plus dysarthria, dysphagia, loss of the gag reflex, fast pulse (late), inability to expectorate mucus, somnolence (late), occasional coma; perhaps paralysis of muscles supplied by the ninth, tenth, eleventh and twelfth nerves, less often the seventh and infrequently the third and sixth nerves—the latter usually in the matter of accommodation; individual cranial nerve palsies may occur
VI	Facial	All signs of type I, abortive and preparalytic, and occasionally type III, meningeal (typical Bell's palsy findings)
VII	Hemiplegic	All signs of type I, abortive and preparalytic, and possibly type III, meningeal, and signs of an upper motor neuron on one side (see text)
VIII	Cortical	All signs of type I, abortive and preparalytic, possibly type III, meningeal, plus signs of general

"Facial paralysis is the most frequent manifestation of acute bulbar-pontine type of poliomyelitis" (pg. 155) [70]

CHAPTER VI.

Symptomatology of Special Types of Acute Poliomyelitis.

RECLASSIFICATION OF TYPES OF POLIOMYELITIS.[1]

I. *The Arrested Type.* (Most frequent. Abortive: old classification.)

II. *The Spinal Myelitic Type.* (Flaccid paralysis.)

III. *Acute Ascending or Descending Spinal Paralysis.* (Landry's. Separation from Type II arbitrary. Both spinal origin.)

IV. *Acute Bulbar.* (Cranial nerve paralysis: facial, auditory, hypoglossal, oculomotor.)

V. *Encephalic Type.*—Polioencephalitis superior: Rolandic cortex—spastic hemiplegias. Frontal area—mental defectives. Occipital area—blindness with normal eye-grounds.

Polioencephalitis inferior: Acute bulbar-pontine type: see above.

Encephalitis cerebelli. The predominant *acute ataxia type.*

Encephalitis of midbrain and connections. *Acute tremor.*

Thalamic encephalitis. Spastic para- or hemi- plegias associated with athetoid and choreic movements and tetany.

VI. *Meningitic Type.*—With or without paralysis.

[1] Modified from Wickmann and Reginald Miller, to whom we are indebted for much recent knowledge of the disease.

(128)

VII. *Neural Type.*—Acute multiple neuritis. Sciatica. Herpes zoster. Chorea.

VIII. *Rapidly Fatal Institutional Disease Type.*

There may be no marked distinction between the types of poliomyelitis; the cerebral type may present symptoms

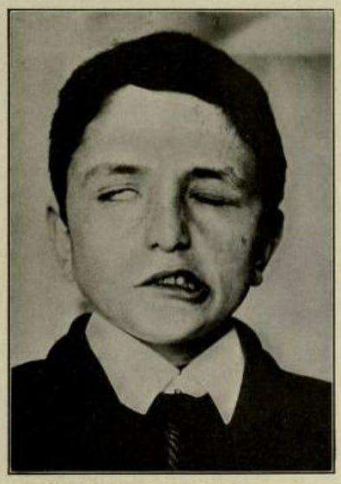


Fig. 35.—Paralysis of facial nerve.

of spinal involvement and *vice versâ;* the localization of this disease may exhibit every caprice of selection.

The symptom relationship between all types is shown most clearly during the earliest stages of the disease, for all types present a similar onset. This symptom group of onset is also the clinical expression of the most numerous class of cases, the arrested, formerly called the abortive, type of this disease. Next in frequency appears the typical

9

In another book on infantile paralysis, one of the forms of poliomyelitis was classified as ***"Acute Bulbar paralysis - Cranial nerve is attacked, facial nerve is the one most frequently paralyzed, generally on one side".*** [71]

(*d*) *Bulbar and Pontine Types.*—Medin was the first to call attention to the frequency with which the cranial nerves were attacked. It is rare to see them involved alone; usually the spinal centres are affected at the same time. The facial nerve is the one most frequently paralysed, generally on one side only and in its whole distribution. With it is associated sometimes the hypoglossal (figs. 4 and 5).

FIG. 4.

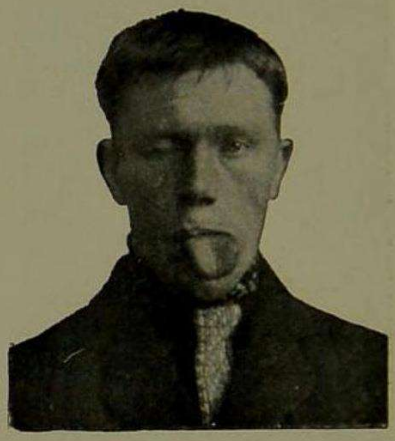


FIG. 5.

"Bell's palsy is another name for facial paralysis" [72]

Bell's Palsy.—Bell's palsy is another name for facial paralysis. The name is derived from Sir Charles Bell, who first described it. It is a paralysis of the seventh cranial nerve and results in a more or less complete paralysis of one side of the face. The cheek is flattened; the muscles are drawn to the opposite side; the patient cannot whistle and is unable to control the lips of the

***"Peripheral facial palsy (Bell's palsy) is the common type of facial paralysis, making up over 80 per cent, of cases**. It is of infra-nuclear (peripheral) or nuclear origin.*

Etiology. — The typical cases of this disease are due to exposure and so-called "rheumatic influences" with infection. After this the most frequent causes are ear disease, trauma, syphilis and tumors. It may complicate poliomyelitis and encephalitis. Males are oftener affected, and the common age is between twenty and forty. It is more frequent in the winter and in temperate chmates. It is not hereditary, but it may be congenital. A neuropathic tendency predisposes to it. Meningeal syphilis sometimes causes an

isolated facial palsy, although it is apt to leave this nerve alone. Facial palsj' may occur in multiple neuritis, when it is often bilateral. Non-typical and accidental cases of peripheral facial palsy are due to injuries, fracture of the petrous bone, or ear disease. Forceps pressure in difficult labor causes some cases. The ordinary cases are mostly due to microbic infections similar to those of "colds" and influenza." [73]

FACIAL PALSIES

The paralyses of the facial nerve may be due to lesions that are cerebral and supra-nuclear, or nuclear and peripheral.

Facial palsy of *supra-nuclear origin* is almost invariably an accompaniment of hemiplegia and is due to hemorrhage, softening, inflammation, or tumor of the brain. The lower two branches of the facial are chiefly involved.

Facial palsy of *nuclear origin* is very rare and is an accompaniment of glosso-labial palsy, of diphtheritic palsy, or of gross lesions of the pons.

A congenital facial palsy may occur. It is *apt* to be associated with other anomalies of development and function, such as misshapen ear and temporal bone defect. It may be familial.

Peripheral facial palsy (*Bell's palsy*) is the common type of facial paralysis, making up over 80 per cent. of cases. It is of infra-nuclear (peripheral) or nuclear origin.

Etiology.—The typical cases of this disease are due to exposure and so-called "rheumatic influences" with infection. After this the most frequent causes are ear disease, trauma, syphilis and tumors. It may complicate poliomyelitis and encephalitis. Males are oftener affected, and the common age is between twenty and forty. It is more frequent in the winter and in temperate climates. It is not hereditary, but it may be congenital. A neuropathic tendency predisposes to it. Meningeal syphilis sometimes causes an isolated facial palsy, although it is apt to leave this nerve alone. Facial palsy may occur in multiple neuritis, when it is often bilateral. Non-typical and accidental cases of peripheral facial palsy are due to injuries, fracture of the petrous bone, or ear disease. Forceps pressure in difficult labor causes some cases. The ordinary cases are mostly due to microbic infections similar to those of "colds" and influenza.

Symptoms.—The disease comes on rather suddenly, and reaches its height within a few hours or, at most, two or three days. Preceding and accompanying the onset there may be some pain about the ears, and a little swelling is sometimes seen.

The patient feels a subjective discomfort on the paralyzed side of the face. He finds that he cannot completely shut the eye and if he

"Bell's palsy accounts for 72% of facial palsies. Isolated facial palsy usually manifests as Bell's palsy" *(Journal of Medical Case Reports, 2011)* [74]

*"Patients with a Bell's Palsy will present with varying severity of painless unilateral **lower motor neuron (LMN) weakness of the facial muscles"** (Teach Me Series)* [75]

In a study of 614 cases of poliomyelitis admitted to a hospital in Chennai between January 1988 and September 1989, **3.6% of the poliomyelitis cases were diagnosed as isolated facial palsy. About 18.9% presented with cranial nerve paralysis**. [76]

TABLE II—*Acute Poliomyelitis: Symptoms and Signs at Presentation*

Symptoms	(n = 614) %	Signs	(n = 614) %
Fever	94.3	Respiratory distress	4.7
Diarrhea	43.0	Altered sensorium	2.3
Loss of head control	22.2	Anxiety	1.3
Pain limbs	21.8	Phantom hernia	28.0
Feeble cry	21.5	Cranial nerve paralysis	18.9
Vomiting	9.9	Diaphragmatic paralysis	4.1
		Intercostal paralysis	2.3

Spinal polio was the commonest clinical type in our study, similar to other reports(3,8,9). Isolated facial palsy was observed in 3.6% of the cases.

Study of 400 hospital admissions in an outbreak of poliomyelitis in Telangana in 1992, Three children had lower motor neurone type of facial nerve paralysis - which fits with the definition of Bell's palsy [77]

Clinical pattern of paralysis showed spinal form of polio with involvement of one, two or more limbs in 82.9% of the cases. Bulbar or bulbospinal form was noticed in 13.5%. Weakness of neck muscles with head drop was the only presenting manifestation in 3.5% of the cases. Three children had lower motor neurone type of facial nerve paralysis, two of them having associated bulbospinal paralysis.

In a polio outbreak in Israel in 1951 ->*"In 22 cases the only manifestation of poliomyelitis was facial paralysis."* [78]

OBSERVATIONS ON THE 1950 POLIOMYELITIS EPIDEMIC IN ISRAEL. W. FOLK, Harefuah **21**:7, 1951.

During 1951, a total of 1,600 cases of poliomyelitis was seen in Israel. The height of the epidemic was in June. Only 1% of the patients were Arabs. The disease was severest in adults who had immigrated from the Netherlands and the United States. Most of the patients were from the North; 60% were males; 93% were 5 years of age or under; 56% were 2 years of age or under, and 30, or 7.4%, were reported to be below 6 months of age. Nineteen were 5 months of age or less. One case was that of an infant, 17 days old, whose mother had the disease at the same time. No difference of susceptibility was observed between infants who were breast fed and those who were artificially fed. There was no difference between children who were well nourished and those with malnutrition. No instances of a second attack were reported. Only two sibling pairs were affected in this series. In three cases the infection apparently took place while the child was in the hospital. No case of paralysis appeared in a limb which had recently been vaccinated. Eighty-seven patients had no paralysis. Prodromal symptoms, usually lasting two or three days, were noted in two-thirds of the cases. Paralysis was found twice as frequently in the lower limbs as in the upper limbs. The deltoid was the muscle most frequently involved in the upper limbs. In the lower limbs the muscles involved most frequently were the tibialis anterior, the peroneus, and the quadriceps. In 22 cases the only manifestation of poliomyelitis was facial paralysis. In 34 cases facial paralysis was present with bulbar or spinal paralysis. No changes were found in the electrocardiogram in any of the cases, although autopsy revealed myocardial involvement. In 9% of the cases the spinal fluid contained less than 10 cells per cubic millimeter; in 40%, 20 to 100 cells; in 1%, over 1,000 cells. The highest cell count was 2,515. The total protein of the spinal fluid was higher in cases of severe paralysis. The blood cell count was normal in all cases. In 10 to 15% of the patients admitted with the diagnosis of poliomyelitis, some other disease simulating it was found. Of 51 deaths, 26, or 13.2%, occurred in the first 24 hours. The greatest mortality was in infants. Forty-two of the patients had to be put in respirators; 67% of these died. The mortality in the cases of bulbar involvement was 67%. The author notes that quarantine did not seem to diminish the number of cases or protect persons from contracting the disease. None of the medical or nursing personnel became ill with poliomyelitis. The author stated his disappointment with the use of drugs for reduction of spasms. He prefers the application of heat. He warns against the use of tubes in patients who cannot swallow. He prefers prolonged intravenous feedings, avoiding in this way the danger of vomiting and aspiration. Tracheotomy was performed in eight patients, three of whom recovered.

In a reported outbreak of poliomyelitis in an orphanage in 1939, Bell's palsy was diagnosed in one of the 6 reported cases, and the diagnosis of poliomyelitis was confirmed with laboratory tests. It was also reported that 2 other cases of Bell's palsy has been observed following acute poliomyelitis infection. Bell's palsy was inferred as a "sequel to polio." [79]

The 6th case lends further uncommon features. She was 21 months old, had been admitted to the orphanage on March 14, having been brought to the city 2 weeks before from a village 30 miles distant. Because she was found to have gonococcus vaginitis she had been transferred from the orphanage March 25. When she was seen on April 2 at the City Hospital, she was found to have a Bell's palsy. Painstaking questioning failed to reveal any record of fever or illness between March 14 and April 2. Nurses, attendants, and welfare agents had failed to note anything wrong with the child's face, but the attempt to examine her on April 2 caused her to cry, and gave evidence of the paralysis. When the poliomyelitis incidence at the orphanage came to light the writer wondered whether this child, too, represented a case of the disease, having seen 2 cases of poliomyelitis that showed Bell's palsy as the sequel to the acute attack.

TABLE 1

Cases of Poliomyelitis in Institutional Outbreak, Syracuse, 1939

Case No.	Name	Age	Sex	Time in Institution: Entered	Time in Institution: Left	Illness: Date of Onset	Illness: Paralysis or Weakness and Date Noted	Laboratory Investigation: Spinal Fluid: Date	Spinal Fluid: Pleocytosis	Spinal Fluid: Globulin	Virus Studies: Date	Virus Studies: Lab.[1]	Virus Studies: Stool	Virus Studies: Nasal Washings	Neutralization for St. Louis and Equine Virus Date
1	E.S.	19 yr.	F	2/ 1/39	3/20	3/19 3/25	Right quadriceps and iliopsoas 3/31	4/18	Neg.	Pos.	5/2	Yale	Neg.		Serum contaminated 5/2
2	J.E.W.	14 mo.	M	2/ 8/38	4/18 to CH[2]	4/9	Right leg 4/18	4/18	Neg.	Pos.	4/29 4/30	N.Y. Yale	Pos Neg.		Serum contaminated 5/6
3	A.M. McD.	17 mo.	F	11/24/37	4/18 to CH	4/9	Left leg 4/18	4/18	Neg.	Pos.	4/29 4/30	N.Y. Yale	Pos. Neg.		Serum contaminated 5/6
4	N.W.	7 mo.	M	3/27/39	4/18 to CH	4/14	Both legs 4/18	4/18	Pos.	Pos.	4/29 4/30	N.Y. Yale	Pos. Neg.		Serum contaminated 5/6
5	J.J.	24 yr.	F	2/ 4/39	4/19 to CH	4/17	None	4/19	Pos.	Pos.	4/22 4/29 5/2	N.I. N.Y. Yale	Pos. Neg. Neg	Neg.	Neg. 5/2
6	S.C.	21 mo.	F	3/14/39	3/25	Undetermined	Right facial 4/2	4/30	Neg.	Pos.	4/30 5/11	Yale N.Y.	Neg. Pos.		Neg. 5/5

[1] Yale: James D. Trask and associates; New York: George Y. McClure, Div. of Lab. and Research, New York State Dept. of Health; N.I.: Surgeons Charles Armstrong and R. D. Lillie, Natl. Institute of Health.
[2] CH—City Hospital.

As polio vaccines were being researched since 1930s, so did the widespread acknowledgement that polio is caused by "viruses", and accordingly, per an article in Illinois medical journal in 1950, a list of 58 diseases that were "*confused*" as poliomyelitis until then was listed, with Bell's Palsy listed as #49 in the list [80]

TABLE 2

DISEASES WHICH HAVE BEEN CONFUSED WITH POLIOMYELITIS

1. Nasopharyngitis
2. Follicular tonsillitis
3. Sinusitis, Acute
4. Mastoiditis, Acute
5. Otitis media
6. Influenza
7. Pneumonia (Viral, Bacterial)
8. Measles
9. Mumps
10. Scarlet fever
11. Whooping cough
12. Endocarditis (Sub-acute and Acute)
13. Rheumatic fever
14. Typhoid fever
15. Bacillary dysentery
16. Gastro-enteritis, Acute
17. Syphilis, congenital
18. Pyelonephritis
19. Nephritis, Acute
20. Trichinosis
21. Osteomyelitis
22. Torticollis
23. Exanthem subitum
24. Tuberculosis, Acute miliary
25. Cerebral Thrombosis
26. Abscess
    a) Retropharyngeal
    b) Cervical
    c) Intra-spinal
    d) Brain
    e) Inguinal
27. Epilepsy
28. Herpes Zoster
29. Tetanus
30. Infectious Mononucleosis
31. Rabies
32. Malaria, cerebral
33. Serum sickness
34. Vaccination — Post (Encephalitis)
35. Appendicitis, Acute
36. Scurvy
37. Rickets
38. Fractures
39. Suppurative Arthritis
40. Traumatic Arthritis
41. Leukemia
42. Post-Diphtheritic Neuritis
43. Lead poisoning
44. Acute multiple sclerosis
45. Acute myositis
46. Amyotrophic lateral sclerosis
47. Hysteria
48. Bronchial Asthma
49. Bell's Palsy
50. Transverse Myelitis
51. Encephalitis
    a) Post-infectious Measles; Mumps; Pertussis
    b) Von-Economo
    c) St. Louis
    d) Japanese B
52. Lymphocytic Chorio-meningitis
53. Infectious Polyneuritis
54. Meningidites
    Tuberculous
    Meningococcic
    Influenzal, etc.
55. Brain tumor
56. Birth injuries
57. Pre-tibial Fever (Fort Bragg fever)
58. Coxsackie Viral Infections

In an analysis of polio outbreak in New York in 1935, it was observed that Bell's palsy simulates poliomyelitis and hence during epidemics many patients with Bell's Palsy are "mistakenly" diagnosed as poliomyelitis. Recall that before the vaccines, clinical diagnosis was used to affirm and record a polio case." [81]

and postvaccinal encephalitis, tetany, sinusitis, cervical adenitis, rheumatic fever involving the muscles of the neck and dysentery with cerebral symptoms. In the third group were patients with conditions simulating bulbar or paralytic poliomyelitis, such as Bell's palsy, conditions caused by cerebrovascular accidents, osteomyelitis, arthritis, epiphysitis and injuries to joints. Thus, it is seen that as in previous epidemics a large number of different diseases presented a clinical picture easily mistaken for some phase of poliomyelitis.

"Of the 446 referred cases of poliomyelitis in a hospital in Chicago over the period 1928-1931, 2 were diagnosed as Bell's palsy." [82]

TABLE 2.—*Differential Diagnosis of 446 Cases Referred as Poliomyelitis (Herman Kiefer Hospital 1928-1931)*

Disease	Number of Cases	Percentage of Total
Poliomyelitis	224	50.2
Nasopharyngitis	66	14.8
Meningococcus meningitis	16	3.6
Otitis media, acute	13	2.9
Tuberculous meningitis	13	2.9
Gastro-enteritis, acute	13	2.9
Tonsillitis, acute follicular	11	2.5
Acute rheumatic fever	9	2.0
Lobar pneumonia	8	1.8
Bronchopneumonia	6	1.4
Appendicitis	5	1.1
Encephalitis	4	0.9
Osteomyelitis	3	0.7
Serum sickness	3	0.7
Scarlet fever	3	0.7
Psychoneurosis	2	0.4
No disease	2	0.4
Influenza	2	0.4
Peripheral neuritis—Bell's palsy	2	0.4
Typhoid fever	2	0.4
Streptococcus meningitis	2	0.4
Pyelitis	2	0.4
Acute generalized miliary tuberculosis	2	0.4
Spastic paraplegia old, acute nasopharyngitis	2	0.4
Exanthem subitum	2	0.4
Traumatic injury of knee	1	0.2
Traumatic injury of hip	1	0.2
Cerebral hemorrhage, hemiplegia	1	0.2
Septicemia	1	0.2
Acute catarrhal jaundice	1	0.2
Poliomyelitis and whooping cough	1	0.2
Acute suppurative mastoiditis	1	0.2
Acute epiphysitis	1	0.2
Whooping cough	1	0.2
Tetanus	1	0.2
Erysipelas	1	0.2
Intracranial hemorrhage of the new-born	1	0.2
Migraine	1	0.2
Malnutrition	1	0.2
Status epilepticus	1	0.2
Bronchial asthma	1	0.2
Syphilitic aortitis	1	0.2
Mucous colitis	1	0.2
Congenital heart disease	1	0.2
Rickets	1	0.2
Acute sinusitis	1	0.2
Pfeiffer bacillus meningitis	1	0.2
Cerebrospinal syphilis	1	0.2
Paratyphoid fever B	1	0.2
Pneumococcus peritonitis	1	0.2
Subacute bacterial endocarditis	1	0.2
Abscess of cervical lymph node	1	0.2
Fracture of skull	1	0.2
Lymphatic leukemia	1	0.2

"From a number of sources the following diseases are obtained as at times ***simulating poliomyelitis****. In the* ***preparalytic stage*** *the exanthematous diseases, as measles and scarlet fever;* ***tonsillitis, diphtheria, croup, influenza, bronchopneumonia, gastro-enteritis, appendicitis, enterocolitis, typhoid fever, intussusception, ptomaine poisoning, nephritis with uremia, eclampsia, rheumatism, scurvy, dentition,***

trichinosis, acidosis, tetanus, rabies, the meningitides and meningismus, chorea, lethargic encephalitis, and subarachnoid hemorrhage.

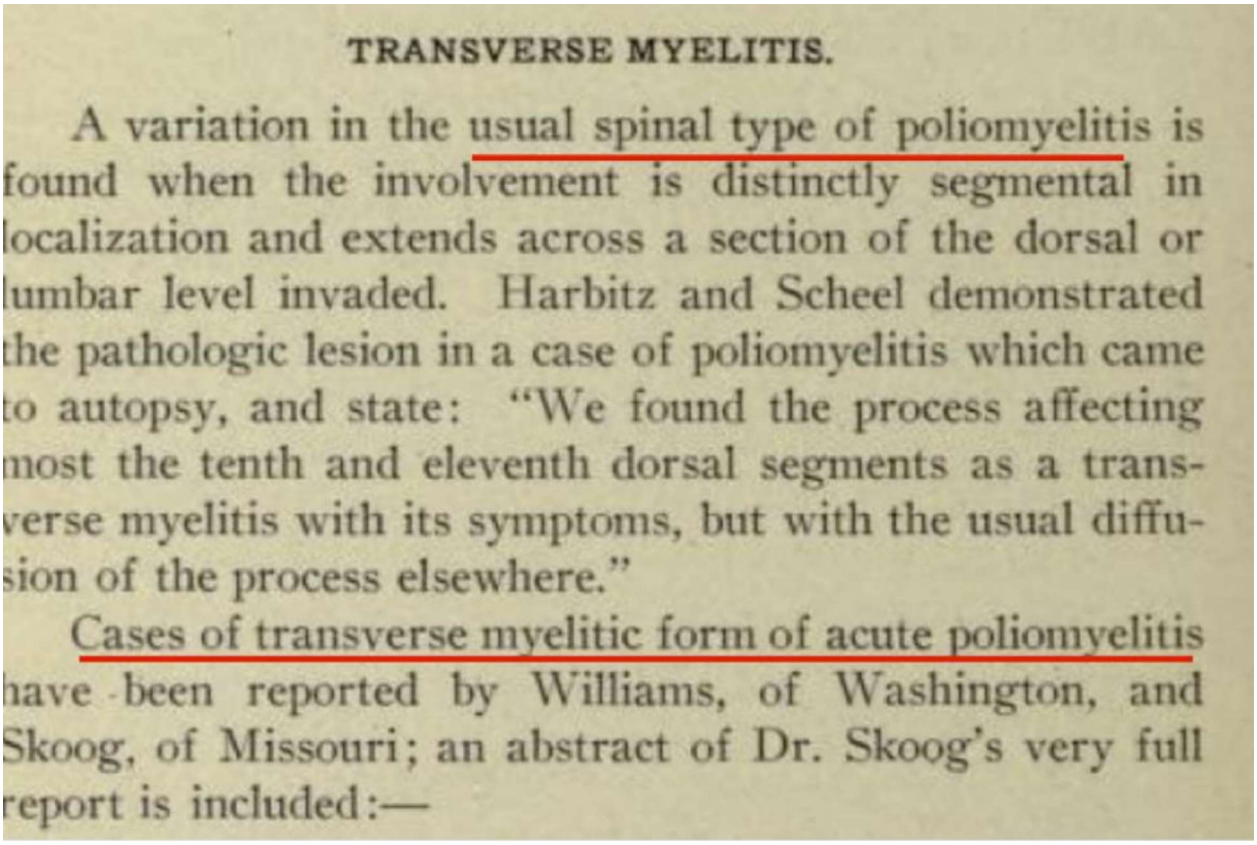
*In the **paralytic stage** at times a differential diagnosis must be made from the pseudoparalysis of scurvy, hysteria, injuries and disease of joints, especially tuberculosis, cerebral arterial thrombosis, sinus thrombosis, amaurotic family idiocy, the cerebral palsies of childhood, polioencephalitis, disseminated encephalomyelitis, cerebral tumor, abscess, transverse myelitis, hematomyelia, syringomyelia, Friedreich's ataxia, Pott's disease, traumatic paralysis, spina bifida occulta, progressive muscular atrophy, muscular dystrophy, infectious neuronitis, multiple neuritis, **Bell's palsy,** birth palsy, diphtheritic palatal palsy, and myatonia congenita."* [83]

From a number of sources the following diseases are obtained as at times simulating poliomyelitis. In the preparalytic stage the exanthematous diseases, as measles and scarlet fever; tonsillitis, diphtheria, croup, influenza, bronchopneumonia, gastro-enteritis, appendicitis, enterocolitis, typhoid fever, intussusception, ptomaine poisoning, nephritis with uremia, eclampsia, rheumatism, scurvy, dentition, trichinosis, acidosis, tetanus, rabies, the meningitides and meningismus, chorea, lethargic encephalitis, and subarachnoid hemorrhage.

In the paralytic stage at times a differential diagnosis must be made from the pseudoparalysis of scurvy, hysteria, injuries and disease of joints, especially tuberculosis, cerebral arterial thrombosis, sinus thrombosis, amaurotic family idiocy, the cerebral palsies of childhood, polioencephalitis, disseminated encephalomyelitis, cerebral tumor, abscess, transverse myelitis, hematomyelia, syringomyelia, Friedreich's ataxia, Pott's disease, traumatic paralysis, spina bifida occulta, progressive muscular atrophy, muscular dystrophy, infectious neuronitis, multiple neuritis, Bell's palsy, birth palsy, diphtheritic palatal palsy, and myatonia congenita.

Supplement 2: Transverse Myelitis and Polio

According to this 1914 book titled "A Manual of Infantile Paralysis" ***transverse myelitis was described as variation in the usual spinal type of poliomyelitis"*** [84]



TRANSVERSE MYELITIS.

A variation in the usual spinal type of poliomyelitis is found when the involvement is distinctly segmental in localization and extends across a section of the dorsal or lumbar level invaded. Harbitz and Scheel demonstrated the pathologic lesion in a case of poliomyelitis which came to autopsy, and state: "We found the process affecting most the tenth and eleventh dorsal segments as a transverse myelitis with its symptoms, but with the usual diffusion of the process elsewhere."

Cases of transverse myelitic form of acute poliomyelitis have been reported by Williams, of Washington, and Skoog, of Missouri; an abstract of Dr. Skoog's very full report is included:—

Per a writeup in Lancet in 1918, there were 8 classifications of Heine-Medin disease (another name for poliomyelitis) of which Type-3 was transverse myelitis. [85]

Type	Epidemic Heine-Medin disease.	"Sporadic" analogues.	Present epidemic.
1	Abortive.	"Convulsions," "constipation," "ptomaine poisoning," "acidosis," &c.	Slight cases of various types, meningeal and encephalitic.
2	Spinal, of sporadic type.	Infantile paralysis.	Not recognised (*cf.* text).
3	"Spinal" of transverse and ascending type.	Some cases (*a*) of transverse, (*b*) of "Landry" type.	Transverse myelitis cases, "ascending myelitis" cases, &c.
4	Meningitic cases.	C.-s. meningitis of unproved bacteriological origin.	Meningitic cases.
5	Encephalitic cases.	Polio-encephalitis of Strümpell.	Most of the present cases, including "epidemic stupor."
6	Ponto-bulbar cases.	Polio-encephalitis superior and inferior.	
7	Atactic cases and	Certain cases of toxic	Cases with sensory and
8	Polyneuritic	polyneuritis. ? Cere-	atactic manifestations

Botulism ?

A 1921 writeup by Association for Research in Nervous and Mental Disease quoted ***4 reported cases of polio that presented symptoms of transverse myelitis*** [86]

Netter and Levaditi (*Bull. et mém. Soc. méd. d. hôp. de Par.*, 1914, Ser. 3, xxxvii, 570) reported 4 cases presenting symptoms of transverse myelitis which they attribute to the virus of acute anterior poliomyelitis. These authors were able to show that the blood of a patient who had recovered, possessed the property of neutralizing the virus of acute anterior poliomyelitis.

Association for Research in Nervous and Mental Disease, and Columbia University Libraries. Acute Epidemic Encephalitis (Lethargic Encephalitis) : An Investigation. Internet Archive, New York, : P.B. Hoeber, 1921

In a writeup in the journal “Neurology” in 1956, there were reports of multiple cases of transverse myelitis that were diagnosed as poliomyelitis or attributed to the “virus of poliomyelitis” [87]

Sensory Loss With Poliomyelitis

Fred Plum, M.D.

PAIN AND CUTANEOUS HYPERALGESIA are common in acute poliomyelitis, while loss of sensation is rare. Levaditi, Pignot, and Leoneano,[1] however, reported the autopsy findings on a 34 year old man whom they diagnosed as having poliomyelitis. He had died on the fourth day of a febrile illness with ascending paralysis and loss of sensation in the legs. The anterior and posterior horns of the spinal cord were infiltrated with hemorrhagic and inflammatory foci, and nerve cells were equally degenerated in both anterior and posterior horns. There were hemorrhagic foci in the cord white matter and neuronolysis in the dorsal root ganglia. Inoculation of a filtrate of spinal cord into the abdomen of a Macacus sinicus monkey was not followed by disease in the monkey, although the animal was later proved susceptible to poliomyelitis virus. Sachs[2] and Lloyd[3] reported individual patients with acute motor and sensory transverse myelitides attributed to poliomyelitis virus. Evidence for poliomyelitis in Sachs' patient consisted of a single positive serum poliomyelitis neutralization test, while Lloyd's case was considered to be poliomyelitic principally on the grounds that it occurred during a poliomyelitis epidemic. Strümpell[4] observed a 19 year old male six months after an attack of acute poliomyelitis. In addition to residual atrophic paralysis of the right and left arms and left hip, the boy had reduced sensation to pain and temperature below segment T_3 on the left and from T_3 to T_6 on the right. Strümpell postulated that poliomyelitis had caused lesions in the posterior gray columns resulting in this syrinx-like clinical picture. Wickman[5] cited one case of poliomyelitis from his extensive series who had “distinct diminution” of pain and temperature sensation in the legs, the sensory changes persisting for over a year. He refers to patients with similar changes observed by Krause[6] and Müller.[7] Pathologic observations are lacking in all of these cases. Little clinical detail was given by Schaller[8] in describing a patient thought to have acute poliomyelitis who suffered “questionable” hypesthesia transiently in the thighs associated with paralysis of both legs. Hüuerman[9] described a 14 year old boy with a complete thoracic transverse myelitis. Because of fever and 26 cells in the spinal fluid the diagnosis of poliomyelitis was made. The child recovered, apparently without residua, in three months. Bell[10]

From the Northwest Respirator Center of the division of neurology, University of Washington School of Medicine, Seattle.
Aided by an annual grant from The National Foundation for Infantile Paralysis, Inc.

In a 1949 article that appeared in the ‘Bulletin of the Mahoning County Medical Society’, **transverse myelitis was referred to as an “atypical form of poliomyelitis” [**88]

In addition to the abortive or non-paralytic type of poliomyelitis, there also occur atypical or unusual forms generally seen during a severe epidemic. These are representations of the disease which in part or whole do not correspond to the usual clinical picture and which present serious problems in diagnosis. Of the numerous atypical forms which have been described, only a few need be mentioned. Occasionally transverse myelitis may occur as a result of acute poliomyelitis and this is said to be due to an "overflow" of the inflammatory process from the gray to the white matter with involvement of the dorsal horns. In such instances, the paralysis is spastic after the stage of spinal shock has subsided.

In a 1952 writeup in the American Journal of Diseases for Children, transverse myelitis was referred to as one of the clinical forms of poliomyelitis [89]

CLINICAL FORMS OF POLIOMYELITIS. C. ARCE, Acta pediat. españ. **9**:1225 (Oct.) 1951.

In a previous article Dr. Arce discussed the spinal type of poliomyelitis, which accounts for 80% of all cases. In the present article he deals with other clinical forms, including abortive, meningeal, the preparalytic, the neuritic or polyneuritic, and the bulbar.

The diagnosis of the bulbar type, which depends upon the nucleus affected, is elaborated in detail. The facial nerve nucleus is the most commonly affected. The progressive ascending form, commonly called Landry's paralysis, is very serious, often fulminating and fatal. The type called encephalitic, the form which affects the pyramidal tracts, the cerebellar form, and the transverse myelitis forms are also described. The disease in all forms is rare among children under 6 months, but occasionally may be seen. In the cases studied by the author in an epidemic in Santander, there were 63 cases, with 2 in children under 6 months. Forty of the cases were spinal. The list is itemized according to site of the paralysis.

Laboratory diagnosis must confirm the clinical findings. The differential diagnosis must distinguish the disease from many mild upper respiratory and gastrointestinal infections. Even in the period of early paralysis, the differential diagnosis is not simple unless there is an epidemic which causes early suspicion.

In a discussion during the 1949 International Poliomyelitis Congress, according to Nielson, *as many as 50 different diseases were called poliomyelitis during epidemics in the Los Angeles area, and symptoms of transverse myelitis simulated polio.* [91]

As polio vaccines were being researched since 1930s, so did the widespread acknowledgement that polio is caused by "viruses", and accordingly, per an article in Illinois medical journal in 1950, a list of 58

diseases that were “confused” as poliomyelitis until then was listed, with **Transverse Myelitis listed as #50 on the list** [80]

TABLE 2

DISEASES WHICH HAVE BEEN CONFUSED WITH POLIOMYELITIS

1. Nasopharyngitis
2. Follicular tonsillitis
3. *Sinusitis, Acute*
4. Mastoiditis, Acute
5. *Otitis media*
6. Influenza
7. *Pneumonia (Viral, Bacterial)*
8. Measles
9. Mumps
10. Scarlet fever
11. Whooping cough
12. Endocarditis (Sub-acute and Acute)
13. Rheumatic fever
14. Typhoid fever
15. Bacillary dysentery
16. Gastro-enteritis, Acute
17. Syphilis, congenital
18. Pyelonephritis
19. Nephritis, Acute
20. Trichinosis
21. Osteomyelitis
22. Torticollis
23. Exanthem subitum
24. Tuberculosis, Acute miliary
25. Cerebral Thrombosis
26. Abscess
    a) Retropharyngeal
    b) Cervical
    c) Intra-spinal
    d) Brain
    e) Inguinal
27. Epilepsy
28. Herpes Zoster
29. Tetanus
30. Infectious Mononucleosis
31. Rabies
32. Malaria, cerebral
33. *Serum sickness*
34. Vaccination — Post (Encephalitis)
35. *Appendicitis, Acute*
36. Scurvy
37. *Rickets*
38. Fractures
39. Suppurative Arthritis
40. Traumatic Arthritis
41. Leukemia
42. Post-Diphtheritic Neuritis
43. Lead poisoning
44. Acute multiple sclerosis
45. Acute myositis
46. Amyotrophic lateral sclerosis
47. Hysteria
48. Bronchial Asthma
49. Bell's Palsy
50. Transverse Myelitis
51. Encephalitis
    a) Post-infectious Measles; Mumps; Pertussis
    b) Von-Economo
    c) St. Louis
    d) Japanese B
52. Lymphocytic Chorio-meningitis
53. Infectious Polyneuritis
54. Meningidites
    Tuberculous
    Meningococcic
    Influenzal, etc.
55. Brain tumor
56. Birth injuries
57. Pre-tibial Fever (Fort Bragg fever)
58. Coxsackie Viral Infections

Before 1997, all cases of acute flaccid paralysis (AFP) were classified as polio. While the definition changed in 1997, what’s important to note is that **one of the 3 most common causes of AFP per an editorial in Indian Journal of Pediatrics was Transverse Myelitis.** [24 , 1]

TABLE I

Other Causes of Acute Flaccid Paralysis in Children

Cornman
1. Transverse myelitis
2. Gullian-Barre syndrome including " Axonal" variety
3. Post-injection sciatic nerve mononeuropatheis
Uncommon
1. Botulism
2. Porphyria
3. Diphtheric polyneuropathy
4. Toxins
5. Human rabies and post-rabies vaccine paralysis

Decline in Polio Incidence

The number of polio cases in India was 13,000-38,000 per annum during the 1980s; currently, polio cases have dramatically declined. This reduction is incorrectly being attributed to polio vaccination only; however, the reasons for the reduction in the incidence of polio include the following: (1) Change in the diagnostic criteria: (a) Up to 1996 all reported cases of acute flaccid paralysis (AFP) were labelled as polio cases, but no follow up was done. (b) From 1997 onwards, an AFP case has been labelled as polio in the presence of one or more of the following: (i) wild polio virus detected in stool sample, (ii) residual paralysis observed after a period of 60 days of onset of paralysis, (iii) the patient has died, or (iv) the patient is lost to follow up. For example there were 10,408 and

Supplement 3: Guillain-Barré syndrome (GBS) and polio

Before 1997, all cases of acute flaccid paralysis (AFP) were classified as polio. While the definition changed in 1997, what's important to note is that one of the 3 most common causes of AFP per an editorial in Indian Journal of Pediatrics was Guillain-Barré Syndrome (GBS). [24 , 1]

GBS (Guillain-Barré syndrome (GBS) is a documented side effect of the Oral Polio vaccine and accounts for close to half of the AFP (acute flaccid paralysis) cases.

Among the non-polio AFP cases, GBS is the most frequently occurring one. Studies indicate GBS accounts for over half of the AFP cases, and a weighted average of the below 4 studies indicate 45% of the the AFP cases were GBS

- Over 55% AFP were GBS per this study of 53 patients at a tertiary care hospital in Mumbai [91]
- Over 32% of the 187 AFP cases at a tertiary hospital in Delhi were GBS [92]
- Over 41% of 130 AFP cases in a hospital in Rajasthan were GBS [93]
- 78% of the 50 AFP cases in a hospital in Mumbai were GBS [94]

Is GBS a side effect of OPV (oral polio vaccine)? According to US Vaccine Safety commission it is?

"The evidence favors acceptance of a causal relation between OPV (oral polio vaccine) and GBS." - Vaccine Safety Committee, Institute of Medicine, US [25]

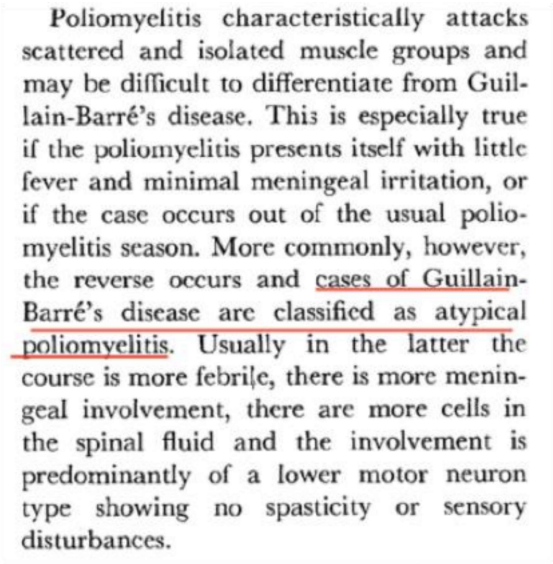
According to this study, **94% of the GBS cases had received 3 or more doses of the polio vaccine**, a disproportionately high number given that estimated vaccination coverage was 57%-75%. [95, 4]

Table 1

Clinical and demographic characteristics of 79 children with GBS in India.

Characteristic	Value
Female (% cases)	42
Mean age ± SD (range), y	6.6 ± 4.0 (4.0–14.5)
All limbs affected (%)	80
Mean time to maximal weakness (range) (days)	5.3 (0.5–30)
UE hypotonia (%)	62
LE hypotonia (%)	86
Hypo/areflexia (%)	88
Symmetrical weakness (%)	84
Ascending weakness (%)	78
Maximal degree of UE weakness (median, IQR)	3.4 (0–5)
Maximal degree of LE weakness (median, IQR)	2.1 (0–4)
Cranial nerve signs/symptoms (%)[a]	11
Bulbar signs/symptoms (%)	22
Respiratory involvement (%)	20
Babinski sign present (%)	3
Meningismus (%)	5
Fever present (%)	35
No. vaccinated with ≥3 doses of OPV (%)	94

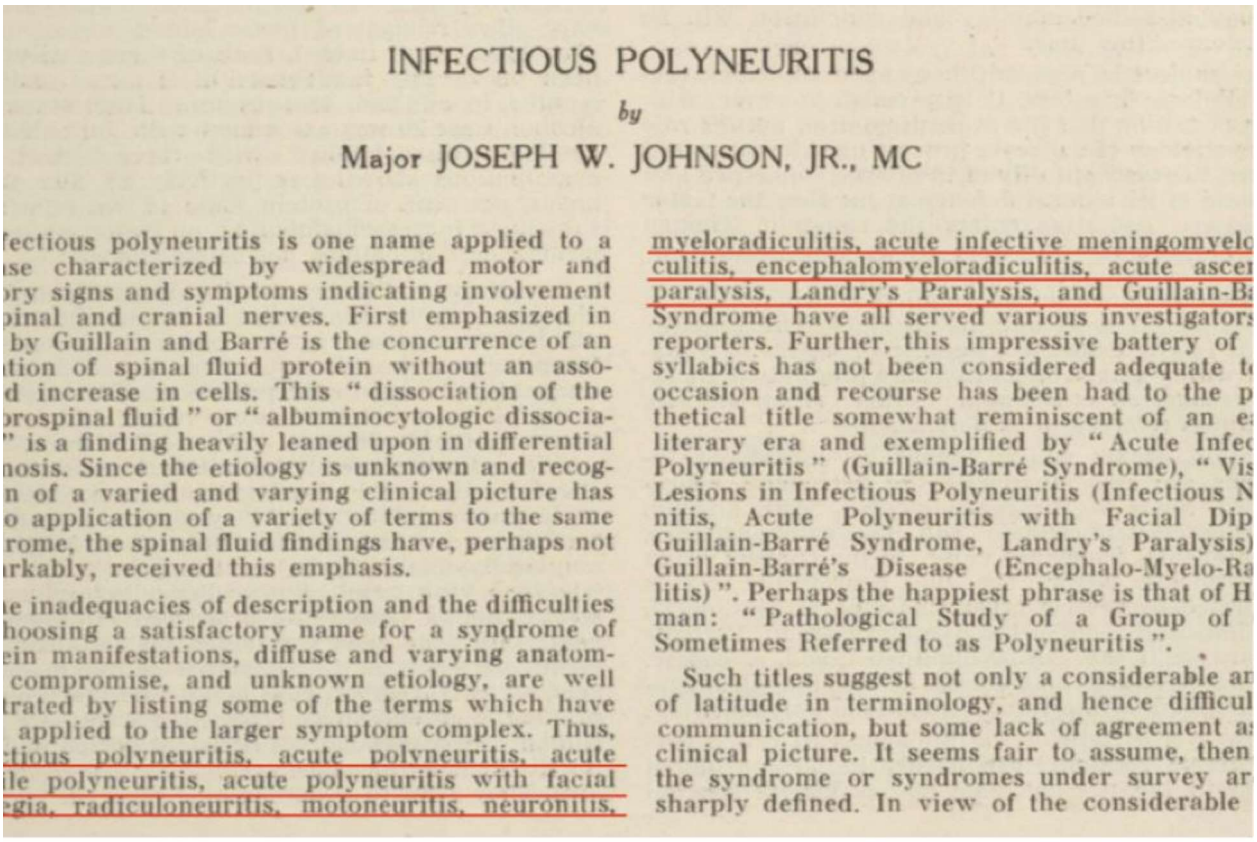
"cases of Guillain-Barré's disease are classified as atypical poliomyelitis" [96]



Poliomyelitis characteristically attacks scattered and isolated muscle groups and may be difficult to differentiate from Guillain-Barré's disease. This is especially true if the poliomyelitis presents itself with little fever and minimal meningeal irritation, or if the case occurs out of the usual poliomyelitis season. More commonly, however, the reverse occurs and cases of Guillain-Barré's disease are classified as atypical poliomyelitis. Usually in the latter the course is more febrile, there is more meningeal involvement, there are more cells in the spinal fluid and the involvement is predominantly of a lower motor neuron type showing no spasticity or sensory disturbances.

Brown, J. R., & Baker, A. B. (1947). The diagnosis of Guillain-Barré's disease. The American Journal of Medicine, 2(1), 45–52. doi:10.1016/0002-9343(47)90007-7

GBS synonyms include: infectious polyneuritis, acute polyneuritis, acute febrile polyneuritis, acute polyneuritis with facial diplegia, radiculoneuritis, motoneuritis, neuronitis, myeloradiculitis, acute infective meningomyeloradiculitis, encephalomyeloradiculitis, acute ascending paralysis, Landry's Paralysis. [97]



INFECTIOUS POLYNEURITIS

by

Major JOSEPH W. JOHNSON, JR., MC

fectious polyneuritis is one name applied to a se characterized by widespread motor and ry signs and symptoms indicating involvement inal and cranial nerves. First emphasized in by Guillain and Barré is the concurrence of an tion of spinal fluid protein without an asso- d increase in cells. This " dissociation of the rospinal fluid " or " albuminocytologic dissocia- " is a finding heavily leaned upon in differential nosis. Since the etiology is unknown and recog- n of a varied and varying clinical picture has o application of a variety of terms to the same rome, the spinal fluid findings have, perhaps not rkably, received this emphasis.

e inadequacies of description and the difficulties hoosing a satisfactory name for a syndrome of ein manifestations, diffuse and varying anatom- compromise, and unknown etiology, are well trated by listing some of the terms which have applied to the larger symptom complex. Thus, tious polyneuritis, acute polyneuritis, acute le polyneuritis, acute polyneuritis with facial gia, radiculoneuritis, moloneuritis, neuronitis, myeloradiculitis, acute infective meningomyelo culitis, encephalomyeloradiculitis, acute ascer paralysis, Landry's Paralysis, and Guillain-Ba Syndrome have all served various investigators reporters. Further, this impressive battery of syllabics has not been considered adequate t occasion and recourse has been had to the p thetical title somewhat reminiscent of an e literary era and exemplified by " Acute Infec Polyneuritis " (Guillain-Barré Syndrome), " Vis Lesions in Infectious Polyneuritis (Infectious N nitis, Acute Polyneuritis with Facial Dip Guillain-Barré Syndrome, Landry's Paralysis) Guillain-Barré's Disease (Encephalo-Myelo-Ra litis) ". Perhaps the happiest phrase is that of H man: " Pathological Study of a Group of Sometimes Referred to as Polyneuritis ".

Such titles suggest not only a considerable ar of latitude in terminology, and hence difficul communication, but some lack of agreement a clinical picture. It seems fair to assume, then the syndrome or syndromes under survey ar sharply defined. In view of the considerable

Conference of Army Physicians, Central Mediterranean Forces (1945 : Istituto superiore di sanità), et al. Infectious Polyneuritis, Proceedings of the Conference of Army Physicians, Central Mediterranean Forces : Held at the Institute Superiore Di Sanità : Rome, 29th January to 3rd February 1945. Internet Archive, Conference of Army Physicians, Central Mediterranean Forces (1945 : Istituto superiore di sanità), author, 1945, archive.org/details/23720340R.nlm.nih.gov/

Landry's case = acute poliomyelitis. Autopsy of a case diagnosed with Landry's paralysis revealed acute anterior poliomyelitis.

"Wechsler mentions a Landry type of poliomyelitis, and again a syndrome, usually without fever and probably toxico-infectious in character, in which recovery may occur. Jelliffe and White view it as belonging primarily to acute poliomyelitis" [98]

ACUTE ASCENDING PARALYSIS: LANDRY SYNDROME

PROLONGED USE OF ARTIFICIAL RESPIRATIONS; DEATH AND AUTOPSY

CHARLES F. READ, M.D.; THOMAS E. CONLEY, M.D., AND HENRY H. CONLEY, M.D., CHICAGO

Landry in 1859 described an acutely progressive paralysis with slight or no sensory involvements and negative microscopic observations, noting recovery in some mild cases. The Oppenheim textbooks of 1911 and 1923 accept the syndrome as such, emphasizing the rapid onset of a flaccid paralysis without pain or especial tenderness and usually without marked disturbances of sensibility, death usually resulting from bulbar paralysis but recovery occurring not infrequently. A close genetic relationship to acute anterior poliomyelitis and polyneuritis is recognized, as well as the fact that inflammatory and vascular changes occur almost always in the cord.

Purves-Stewart [1] treats the syndrome throughout as Landry's paralysis, a condition of the lower motor neuron attributable to various infective agents. Wechsler [2] mentions a Landry type of poliomyelitis, and again a syndrome, usually without fever and probably toxico-infectious in character, in which recovery may occur. Jelliffe and White [3] view it as belonging primarily to acute poliomyelitis, "though in certain forms it is a neuritis," and state that "rabies also has its Landry's type."

Knutti [4] has recently described the rabic form, and there are a number of references in the literature to the syndrome following antirabic inoculation. However, during the past ten years comparatively little has been written on the subject, when reports on diffuse myelitis are excluded. The differentiation from acute polyneuritis seems to be exceedingly difficult at times; in fact, it often seems to be quite impossible during life. Postmortem observations often indicate involvement of the entire lower neuron, together with degenerative and reactive changes in the cord elsewhere than in the anterior horns.

were free; the apexes of both lungs showed old scars and there was passive congestion of the lower right lobe. The abdomen and its contents were negative. The brain was wet, with some exudate of fibrin on the parietal lobes near the central fissures; no exudate was seen at the base. The spinal cord was normal on inspection.

Dr. G. B. Hassin reported the microscopic observations as follows: The spinal cord showed throughout perivascular infiltrations, which were especially marked in the gray matter. The infiltrating cells were mainly lymphocytes, with plasma cells and polyblasts.

The blood vessels were not only infiltrated but greatly engorged; in many places there were very large hemorrhages. The ganglion cells were swollen and showed phenomena of chromatolysis and neurophagia. Glia cells were proliferated throughout the gray as well as the white matter, and in many instances formed nodules, especially in the medulla.

The changes were alike throughout the cord. They were very prominent in the medulla, where, however, they were not so marked as in the spinal cord. The meninges were but slightly involved and the roots were also infiltrated by hematogenous elements; especially the perineural spaces were involved. The microscopic diagnosis was acute anterior poliomyelitis.

30 North Michigan Avenue

A diagnosis of Landry's type of ascending paralysis was evident. The absence of an epidemic, together with negative spinal fluid observations, inclined us against the idea of poliomyelitis, although the febrile reaction and high leukocyte count indicated a severe infection of some kind. The high sugar content of the spinal fluid, if reliable, was in line with similar observations in epidemic encephalitis, but there was nothing else in the picture to suggest this. Dextrose solution by hypodermoclysis was given freely, together with digifolin, ergot (Oppenheim), methenamine and, toward the end, oxygen. The patient did fairly well for several days, supervising the work of the artificial respiration crews and dozing a little at times. On the fourth day after admission—the sixth day of the disease—he seemed to swallow somewhat better but failed rapidly the following day and died of cardiorespiratory failure.

Per an article that appeared in the Journal "Brain" in 1916, 'Landry' or jump cases (i.e. GBS) was one of the features of poliomyelitis of ascending form

*"Chapter VI of Batten's monograph deals at length with the **clinical features of poliomyelitis**. Typically there is flaccid paralysis of one or more limbs. Sometimes this ascends progressively from the legs, and at an irregular pace, eventually affecting all muscles below the neck causing death from respiratory failure. **These are the 'Landry' or jump cases**, in some of whom temporary improvement seems to occur before the illness again progresses"* [99]

Chapter VI of Batten's monograph deals at length with the clinical features of poliomyelitis. Typically there is flaccid paralysis of one or more limbs. Sometimes this ascends progressively from the legs, and at an irregular pace, eventually affecting all muscles below the neck causing death from respiratory failure. These are the 'Landry' or jump cases, in some of whom temporary improvement seems to occur before the illness again progresses. Less common is a descending form of poliomyelitis. Children with isolated trunk weakness show marked scoliosis; and, in other cases, the differential weakness leads to abnormal posture and fixed deformities of the trunk or limbs (Fig. 3). Batten attributes ophthalmoplegia and blindness to poliomyelitis is some cases. Examples of transverse poliomyelitis, as described by Batten, with flaccid paralysis, loss of sphincter control and a sensory level developing brisk reflexes and clonus now seem inappropriately classified. And,

Compston, Alastair. "Acute Poliomyelitis.ByF. E. Batten MD Cantab FRCP Lond. The Lumleian Lectures for 1916 Delivered before the Royal College of Physicians.Brain1916; 39: 115–211." Brain, vol. 139, no. 5, 17 Apr. 2016, pp. 1615–1620, https://doi.org/10.1093/brain/aww073.

Per the below book on Infantile paralysis (another name for polio), Landry's paralysis (GBS) is the same as infantile paralysis (i.e. poliomyelitis) with a fatal tissue.

"Landry's paralysis was only " infantile paralysis " with a fatal issue; further, that fatal cases of infantile paralysis ran the same clinical course as cases of Landry's paralysis." [71]

of the limbs and rapidly progressed to a fatal issue by involving the medullary centres. The history of this disease furnishes a good example of the way in which an arbitrary and narrow classification may prevent the recognition of wide and important relationships between diseases. Observers overlooked the fact, which was clear from the identity of the anatomical findings, that Landry's paralysis was only "infantile paralysis" with a fatal issue; further, that fatal cases of infantile paralysis ran the same clinical course as cases of Landry's paralysis. As Wickman said, they

This 1876 writeup in the Journal of Nervous and Mental Disease establishes equivalence between Landry's paralysis and poliomyelitis

"the tendency to find a pathological anatomy for a group of symptoms resembling in some respects poliomyelitis has led to relatively frequent descriptions of the lesions of poliomyelitis under the heading of Landry's paralysis." [100]

Landry's Paralysis:—As has been suggested, the tendency to find a pathological anatomy for a group of symptoms resembling in some respects poliomyelitis has led to relatively frequent descriptions of the lesions of poliomyelitis under the heading of Landry's paralysis. Judging from the illustrations, as

Per a book on poliomyelitis (also called Heine-Medin's disease), it's impossible to distinguish between Landry's paralysis (GBS) and Heine-Medin's disease (poliomyelitis). [101]

Heine-Medin's disease may run a course resembling Landry's paralysis. I am of the opinion that a clinical differentiation of these conditions is impossible at present.

"There is a growing feeling among physicians that most of the cases of the so-called Landry's paralysis (GBS) are really poliomyelitis, and this is no doubt true". [102]

ing type are usually fatal. There is a growing feeling among physicians that most of the cases of the so-called Landry's paralysis are really poliomyelitis, and this is no doubt true, although it is possible there may be other diseases which cause the same picture. There can, however, be no doubt that the cases of ascending paralysis, as ordinarily met with, are true poliomyelitis, and this has been verified by studies of many observers. It is, unfortunately, a rather common form of the disease. It forms a characteristic group clinically, but should be grouped as of the bulbospinal type. There is otherwise no reason to separate it. In the cases which have been described as Landry's paralysis, apart from those in which the diagnosis of poliomyelitis was certain, there has been an ascending, seldom a descending, paralysis running, as a rule, a rather rapid course, generally not much fever, and this has been preceded by pain, tingling, numbness, a sense of fatigue, and heaviness in the arms and legs. The paralysis either continues to progress without intermission or it may develop step by step in progressive stages and corresponding roughly to the spinal innervation. When it

"in recent years the term Landry's paralysis has been applied to cases of poliomyelitis displaying a very acute severe generalized paralysis" [103]

"***Landry's paralysis (or GBS) usually, though not always, is a rapidly form of poliomyelitis*** ; *sometimes it is familial; sometimes it is due Acute to rabies, and occasionally it follows injections of vaccine*" [104]

Landry's paralysis usually, though not always, is a rapidly ascending form of poliomyelitis;[5] sometimes it is familial;[6] sometimes it is due to rabies, and occasionally it follows injections of vaccine.[7] Acute aseptic or serous meningitis[8] is very likely a nonparalytic form of poliomyelitis.

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