

**Summer Training at
Indian Institute of Public Health, Gandhinagar, Ahmedabad
(April - June, 2013)**

- 1) To better understand and learn the existing healthcare delivery system including public and private sectors.**
- 2) To observe the implementation of various National Health Programmes at State/District/Block levels.**
- 3) To acquire more knowledge regarding the roles and responsibilities of key players and stakeholders in health sector.**
- 4) To understand various functions of health system by interactions with key stakeholders, policy makers, programme managers, academicians and researchers.**
- 5) To work on the project/task assigned by summer training organization.**

**By
Rohan Nalinbhai Gheewala**

**Under the guidance of
Mr. Vijan Pratap Raghuvanshi**

**Post Graduate Diploma in Hospital and Health Management
2012-2014**



**Institute of Health Management Research,
Jaipur
2013**

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CERTIFICATE

This is to certify that, Rohan Nalinbhai Gheewala, student of Post Graduate Diploma in Hospital and Health management (PGDHM) 17th Batch from Institute of Health Management and Research, Jaipur has undergone summer training in Indian Institute of Public Health Gandhinagar, Ahmedabad from April 8th – June 8th, 2013.

The candidate has successfully carried out the studies assigned to him during summer training and his approach to the studies has been sincere, scientific and analytical.

I wish him success in all his future endeavors.



Mr. Vijay Pratap Raghuvanshi

Faculty and Mentor

IIHMR, Jaipur



Dr. Ashok Kaushik

Dean, Academic and Student Affairs

IIHMR, Jaipur

Introduction

Summer internship is an integral part of the PGDHM course curriculum. As a part of the course; the students completing the first year of their studies are required to undergo summer placement (training) at any reputed organization to get an in-depth exposure of the various departments of the organization and to get a better understanding of the health care delivery system and its functions.

The major objectives of the summer training programme are as follows:

- 1) To better understand and learn the existing healthcare delivery system including public and private sectors.
- 2) To observe the implementation of various National Health Programmes at State/District/Block levels.
- 3) To acquire more knowledge regarding the roles and responsibilities of key players and stakeholders in health sector.
- 4) To understand various functions of health system by interactions with key stakeholders, policy makers, programme managers, academicians and researchers.
- 5) To work on the project/task assigned by summer training organization.

During the internship period, I had worked on many different assignments,

They are as follows-

- 1) AFP(Acute Flaccid Paralysis) project for detection of residual cases of polio
- 2) In Diabetes stall in Health Expo camp on April 12, 13, 14 with Swasthya Diabetes Care, Ahmedabad.
- 3) Study on alcohol consumption and alcohol related crimes in Gujarat.
- 4) 2 days on field trip to Mehsana for data collection for conquer diabetes project in 8 PHCs.
- 5) Clicking pictures which were a part of 'Wall of Shame' in TAPS (violation of tobacco advertising, promotion and sponsorship ban) from across India- 3 pictures clicked by me got selected in the Wall of Shame.
- 6) Translations of multiple articles from Gujarati into English and vice Versa.

Acknowledgements

I extend my sincere gratitude to Professor Dr. Dileep Mavalankar, Director, Indian Institute of Public Health, Gandhinagar (IIPH-G), for giving me the opportunity to have this summer training experience and undergoing the process of learning at IIPH-G. I thank him for all the trust and faith that he posed in me and I only hope that I have been able to live up to his expectations. His guidance and support was helpful in enhancing my work.

I extend my sincere gratitude to Mr. Mayur Trivedi (Assistant Professor) Indian Institute of Public Health, Gandhinagar (IIPH-G), who has been my mentor for the summer internship program & has been instrumental in helping me shape this study in a desirable way.

I would also like to thank Dr. Deepak Saxena (Assistant Professor), and Mr. Gaurav Kumar (Research Associate), at IIPH-G for supporting my work all along.

Last but not the least; I would like to thank the entire staff of Indian Institute of Public Health Gandhinagar, for their support, co-operation and guidance.

I would also like to express my sincere thanks to Dr. S.D. Gupta, Director, IIHMR, Dr. P.R. Sodani, Dean (Academics), my mentor Mr. Vijay Pratap Raghuvanshi, IIHMR, as well Mr. Dalbir Singh, Senior Placement Officer, for the proper guidance and cooperation extended by him. I am also extremely grateful to my parents, my family and my friends for giving me moral support.

However, I accept the sole responsibility for any possible error of omission and would be extremely grateful to the readers of this Internship project report if they bring such mistakes to my notice.

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SECTION A

1. About PHFI (Public Health Foundation of India)

The Public Health Foundation of India (PHFI) is a public private initiative that has collaboratively evolved through consultations with multiple constituencies including Indian and international academia, state and central governments, multi & bi-lateral agencies and civil society groups. PHFI is a response to redress the limited institutional capacity in India for strengthening training, research and policy development in the area of Public Health.

Structured as an independent foundation, PHFI adopts a broad, integrative approach to public health, tailoring its endeavours to Indian conditions and bearing relevance to countries facing similar challenges and concerns. The PHFI focuses on broad dimensions of public health that encompass promotive, preventive and therapeutic services, many of which are frequently lost sight of in policy planning as well as in popular understanding.

The Prime Minister of India, Dr. Manmohan Singh, launched PHFI on March 28, 2006 at New Delhi. PHFI recognizes the fact that meeting the shortfall of health professionals is imperative to a sustained and holistic response to the public health concerns in the country which in turn requires health care to be addressed not only from the scientific perspective of what works, but also from the social perspective of, who needs it the most.

1.1 Charter

The PHFI is working towards building public health capacity in India by:

- Establishing 5 -7 new institutes of public health;
- **Assisting the growth of existing public health training institutions/ Departments** and facilitating their evolution into major institutes of public health;
- **Establishing a strong national research network of public health and allied institutions** which would undertake policy and programme relevant research that will advance public health goals in prioritized areas - **with suitable international partnerships where useful and appropriate;**
- Engaging public health expertise to collectively undertake analytical work **for generating policy recommendations** related to public health action, in not only the health sector but also in all other sectors which impact upon health of people, **and developing a vigorous advocacy platform** to effectively communicate these recommendations to policy makers and other relevant stake holder groups (including civil society organizations which represent the interests of people's health);
- Facilitating in establishing an independent accreditation body for degrees in public health which are awarded by training institutions across India.

1.2 Activities

- **Health Communication**
Communication that is engaging and empowering, and provides individuals and populations with evidence-based options for positive action is critical to enhancing health literacy in society, thereby enabling its movement towards better public health outcomes.



- Health System Support
The purpose of establishing a Health Systems Support Unit (HSSU) at PHFI is to leverage and learn from research, education, advocacy and training, and provide a platform for offering technical support for strengthening initiatives across the country.

Mandate

HSSU has the following mandate:

- Establishing strategic partnerships with central and state governments and other relevant stake holders in the area of Health System strengthening
- Initiating programmatic and research activities in the arena of health system strengthening including emergency and disaster preparedness in hospitals and communities.
- Carrying out operational research, operational improvement and quality improvement activities with private and public agencies on themes of public health relevance.
- Hand holding and partnering with non-governmental organisations (NGOs) to work on health services.

- Promoting alignment of educational programmes of PHFI with present and future National Health Programme needs
- Engaging in setting up of mentoring, monitoring and management frameworks in public health systems.

2. About IIPH (Indian Institute of Public Health), Gandhinagar

The Indian Institute of Public Health – Gandhinagar (IIPH-G) started on World Health Day – 7th April 2008.



The institute's activities received funding support from NRHM/ Ministry of Health and Family Welfare, and Medical Council of India, CSIR, NABARD, Karoliska Institute, NRDC, IPH Bangalore, etc. IIPH G has been providing research based policy advice to Government of Gujarat in its health policy making process. Institute faculty are on various government committees, NGO boards and international advisory committees. The faculty also helps in teaching programs and advisory committees. The faculty also helps in teaching programs and conducting workshops at other IIPHs and other academic institutions.

SECTION B – DIFFERENT PROJECTS

1) Finding cases of Residual Paralysis in Acute Flaccid Paralysis cases

Introduction

As we know, **Poliomyelitis**, often called **polio** or **infantile paralysis**, is an acute, viral, infectious disease spread from person to person, primarily via the faceco-oral route, caused by Polio virus. It invades the nervous system, and can cause total paralysis in a matter of hours. The virus enters the body through the mouth and multiplies in the intestine. A severe infection can extend into the brainstem and even higher structures, resulting in *polioencephalitis*, producing apnea that requires mechanical assistance such as an Iron lung. Although approximately 90% of polio infections cause no symptoms at all, affected individuals can exhibit a range of symptoms if the virus enters the blood stream.

In about 1% of cases, the virus enters the central nervous system, preferentially infecting and destroying motor neurons, leading to muscle weakness and acute flaccid paralysis.

Different types of paralysis may occur, depending on the nerves involved. Spinal polio is the most common form, characterized by asymmetric paralysis that most often involves the legs. Bulbar polio leads to weakness of muscles innervated by cranial nerves. Bulbospinal polio is a combination of bulbar and spinal paralysis .One in 200 infections leads to irreversible paralysis (usually in the legs). Among those paralysed, 5% to 10% die when their breathing muscles become immobilized.

Polio cases have decreased by over 99% since 1988, from an estimated 350 000 cases in more than 125 endemic countries then, to 223 reported cases in 2012. In 2013, only parts of three countries in the world remain endemic for the disease–the smallest geographic area in history–and case numbers of wild poliovirus type 3 are down to lowest-ever levels.

Background and History

Poliomyelitis was first recognized as a distinct condition by Jakob Heine in 1840. Its causative agent, poliovirus, was identified in 1908 by Karl Landsteiner. Although major polio epidemics were unknown before the late 19th century, polio was one of the most dreaded childhood diseases of the 20th century. Polio epidemics have crippled thousands of people, mostly young children; the disease has caused paralysis and death for much of human history. Polio had existed for thousands of years quietly as an

endemic pathogen until the 1880s, when major epidemics began to occur in Europe; soon after, widespread epidemics appeared in the United States.

By 1910, much of the world experienced a dramatic increase in polio cases and epidemics became regular events, primarily in cities during the summer months. These epidemics, which left thousands of children and adults paralyzed, provided the impetus for a "Great Race" towards the development of a vaccine. Developed in the 1950s, polio vaccines have reduced the global number of polio cases per year from many hundreds of thousands to under a thousand today. Enhanced vaccination efforts led by Rotary International, the World Health Organization, and UNICEF should result in global eradication of the disease.

Classification

Outcomes of poliovirus infection	
Outcome	Proportion of cases
Asymptomatic	90–95%
Minor illness	4–8%
Non-paralytic aseptic meningitis	1–2%
Paralytic poliomyelitis	0.1–0.5%
— Spinal polio	79% of paralytic cases
— Bulbospinal polio	19% of paralytic cases
— Bulbar polio	2% of paralytic cases

The term "poliomyelitis" is used to identify the disease caused by any of the three serotypes of poliovirus. Two basic patterns of polio infection are described: a minor illness which does not involve the central nervous system (CNS), sometimes called abortive poliomyelitis, and a major illness involving the CNS, which may be paralytic or non-paralytic. In most people with a normal immune system, a poliovirus infection is asymptomatic. Rarely, the infection produces minor symptoms; these may include upper respiratory tract infection (sore throat and fever), gastrointestinal disturbances (nausea, vomiting, abdominal pain, constipation or, rarely, diarrhoea), and influenza-like illness. Initial symptoms are fever, fatigue, headache, and stiffness in the neck and pain in the limbs

The virus enters the central nervous system in about 3% of infections. Most patients with CNS involvement develop non-paralytic aseptic meningitis, with symptoms of headache, neck, back, abdominal and extremity pain, fever, vomiting, lethargy, and irritability. About one to five in 1000 cases progress to paralytic disease, in which the muscles become weak, floppy and poorly controlled, and, finally, completely paralyzed; this condition is known as acute flaccid paralysis. Depending on the site of paralysis, paralytic poliomyelitis is classified as spinal, bulbar, or bulbospinal. Encephalitis, an infection of the brain tissue itself, can occur in rare cases, and is usually restricted to infants. It is characterized by confusion, changes in mental status, headaches, fever, and, less commonly, seizures and spastic paralysis.

Cause

Poliomyelitis is caused by infection with a member of the genus *Enterovirus* known as poliovirus (PV). This group of RNA viruses colonize the gastrointestinal tract — specifically the oropharynx and the intestine. The incubation time (to the first signs and symptoms) ranges from three to 35 days, with a more common span of six to 20 days. PV infects and causes disease in humans alone. Its structure is very simple, composed of a single (+) sense RNA genome enclosed in a protein shell called a capsid. In addition to protecting the virus's genetic material, the capsid proteins enable poliovirus to infect certain types of cells. Three serotypes of poliovirus have been identified—poliovirus type 1 (PV1), type 2 (PV2), and type 3 (PV3)—each with a slightly different capsid protein. All three are extremely virulent and produce the same disease symptoms. PV1 is the most commonly encountered form, and the one most closely associated with paralysis.

Individuals who are exposed to the virus, either through infection or by immunization with polio vaccine, develop immunity. In immune individuals, IgA antibodies against poliovirus are present in the tonsils and gastrointestinal tract, and are able to block virus replication; IgG and IgM antibodies against PV can prevent the spread of the virus to motor neurons of the central nervous system. Infection or vaccination with one serotype of poliovirus does not provide immunity against the other serotypes, and full immunity requires exposure to each serotype.

A rare condition with a similar presentation, nonpoliovirus poliomyelitis, may result from infections with nonpoliovirus enteroviruses.

Transmission

Poliomyelitis is highly contagious via the oral-oral (oropharyngeal source) and fecal-oral (intestinal source) routes. In endemic areas, wild polioviruses can infect virtually the entire human population. It is seasonal in temperate climates, with peak transmission occurring in summer and autumn. These seasonal differences are far less pronounced in tropical areas. The time between first exposure and first symptoms, known as the incubation period, is usually six to 20 days, with a maximum range of three to 35 days. Virus particles are excreted in the faeces for several weeks following initial infection. The disease is transmitted primarily via the fecal-oral route, by ingesting contaminated food or water. It is occasionally transmitted via the oral-oral route, a mode especially visible in areas with good sanitation and hygiene. Polio is most infectious between seven and 10 days before and after the appearance of symptoms, but transmission is possible as long as the virus remains in the saliva or faeces.

Factors that increase the risk of polio infection or affect the severity of the disease include immune deficiency, malnutrition, tonsillectomy, and physical activity immediately following the onset of paralysis, skeletal muscle injury due to injection of vaccines or therapeutic agents, and pregnancy. Although the virus can cross the maternal-foetal barrier during pregnancy, the foetus does not appear to be affected by either maternal infection or polio vaccination. Maternal antibodies also cross the placenta, providing passive immunity that protects the infant from polio infection during the first few months of life.

As a precaution against infection, public swimming pools were often closed in affected areas during poliomyelitis epidemics.

Pathophysiology

Poliovirus enters the body through the mouth, infecting the first cells with which it comes in contact — the pharynx and intestinal mucosa. It gains entry by binding to an immunoglobulin-like receptor, known as the poliovirus receptor or CD155, on the cell membrane. The virus then hijacks the host cell's own machinery, and begins to replicate. Poliovirus divides within gastrointestinal cells for about a week, from where it spreads to the tonsils (specifically the follicular dendritic cells residing within the tonsillar germinal centers), the intestinal lymphoid tissue including the M cells of Peyer's patches, and the deep cervical and mesenteric lymph nodes, where it multiplies abundantly. The virus is subsequently absorbed into the bloodstream.

Known as viremia, the presence of virus in the bloodstream enables it to be widely distributed throughout the body. Poliovirus can survive and multiply within the blood and lymphatics for long periods of time, sometimes as long as 17 weeks. In a small percentage of cases, it can spread and replicate in other sites, such as brown fat, the reticuloendothelial tissues, and muscle. This sustained replication causes a major viremia, and leads to the development of minor influenza-like symptoms. Rarely, this may progress and the virus may invade the central nervous system, provoking a local inflammatory response. In most cases, this causes a self-limiting inflammation of the meninges, the layers of tissue surrounding the brain, which is known as nonparalytic aseptic meningitis. Penetration of the CNS provides no known benefit to the virus, and is quite possibly an incidental deviation of a normal gastrointestinal infection. The mechanisms by which poliovirus spreads to the CNS are poorly understood, but it appears to be primarily a chance event—largely independent of the age, gender, or socioeconomic position of the individual.

Paralytic Polio

In around 1% of infections, poliovirus spreads along certain nerve fiber pathways, preferentially replicating in and destroying motor neurons within the spinal cord, brain stem, or motor cortex. This leads to the development of paralytic poliomyelitis, the various forms of which (spinal, bulbar, and bulbospinal) vary only with the amount of neuronal damage and inflammation that occurs, and the region of the CNS affected.

The destruction of neuronal cells produces lesions within the spinal ganglia; these may also occur in the reticular formation, vestibular nuclei, cerebellar vermis, and deep cerebellar nuclei. Inflammation associated with nerve cell destruction often alters the colour and appearance of the gray matter in the spinal column, causing it to appear reddish and swollen. Other destructive changes associated with paralytic disease occur in the forebrain region, specifically the hypothalamus and thalamus. The molecular mechanisms by which poliovirus causes paralytic disease are poorly understood.

Early symptoms of paralytic polio include high fever, headache, stiffness in the back and neck, asymmetrical weakness of various muscles, sensitivity to touch, difficulty swallowing, muscle pain, loss of superficial and deep reflexes, paresthesia (pins and needles), irritability, constipation, or difficulty urinating. Paralysis generally develops one to ten days after early symptoms begin, progresses for two to three days, and is usually complete by the time the fever breaks.

The likelihood of developing paralytic polio increases with age, as does the extent of paralysis. In children, nonparalytic meningitis is the most likely consequence of CNS involvement, and paralysis occurs in only one in 1000

cases. In adults, paralysis occurs in one in 75 cases. In children under five years of age, paralysis of one leg is most common; in adults, extensive paralysis of the chest and abdomen also affecting all four limbs — quadriplegia — is more likely. Paralysis rates also vary depending on the serotype of the infecting poliovirus; the highest rates of paralysis (one in 200) are associated with poliovirus type 1, the lowest rates (one in 2,000) are associated with type 2.

History of the vaccine

Two types of vaccine are used throughout the world to combat polio. Both types induce immunity to polio, efficiently blocking person-to-person transmission of wild poliovirus, thereby protecting both individual vaccine recipients and the wider community (so-called herd immunity).

The first candidate polio vaccine, based on one serotype of a live but attenuated (weakened) virus, was developed by the virologist Hilary Koprowski. Koprowski's prototype vaccine was given to an eight-year-old boy on February 27, 1950. Koprowski continued to work on the vaccine throughout the 1950s, leading to large-scale trials in the then Belgian Congo and the vaccination of seven million children in Poland against serotypes PV1 and PV3 between 1958 and 1960.

The second inactivated virus vaccine was developed in 1952 by Jonas Salk at the University of Pittsburgh, and announced to the world on April 12, 1955. The Salk vaccine, or inactivated poliovirus vaccine (IPV), is based on poliovirus grown in a type of monkey kidney tissue culture (vero cell line), which is chemically inactivated with formalin. After two doses of IPV (given by injection), 90% or more of individuals develop protective antibody to all three serotypes of poliovirus, and at least 99% are immune to poliovirus following three doses.

Subsequently, Albert Sabin developed another live, oral polio vaccine (OPV). It was produced by the repeated passage of the virus through nonhuman cells at subphysiological temperatures. The attenuated poliovirus in the Sabin vaccine replicates very efficiently in the gut, the primary site of wild poliovirus infection and replication, but the vaccine strain is unable to replicate efficiently within nervous system tissue. A single dose of Sabin's oral polio vaccine produces immunity to all three poliovirus serotypes in about 50% of recipients. Three doses of live-attenuated OPV produce protective antibody to all three poliovirus types in more than 95% of recipients. Human trials of Sabin's vaccine began in 1957, and in 1958 it was selected, in competition with the live vaccines of Koprowski and other researchers, by the US National Institutes of Health. Licensed in 1962, it rapidly became the only polio vaccine used worldwide.

Because OPV is inexpensive, easy to administer, and produces excellent immunity in the intestine (which helps prevent infection with wild virus in areas where it is endemic), it has been the vaccine of choice for controlling poliomyelitis in many countries. On very rare occasions (about one case per 750,000 vaccine recipients), the attenuated virus in OPV reverts into a form that can paralyze. Most industrialized countries have switched to IPV, which cannot revert, either as the sole vaccine against poliomyelitis or in combination with oral polio vaccine.

The Global Polio Eradication Initiative

Launch

In 1988, the forty-first World Health Assembly adopted a resolution for the worldwide eradication of polio. It marked the launch of the Global Polio Eradication Initiative (GPEI), spearheaded by national governments, WHO, Rotary International, the US Centers for Disease Control and Prevention (CDC), UNICEF, and supported by key partners including the Bill and Melinda Gates Foundation. This followed the certification of the eradication of smallpox in 1980, progress during the 1980s towards elimination of the poliovirus in the Americas, and Rotary International's commitment to raise funds to protect all children from the disease.

Progress

Overall, since the GPEI was launched, the number of cases has fallen by over 99%. In 2013, only three countries in the world remain polio-endemic: Nigeria, Pakistan and Afghanistan.

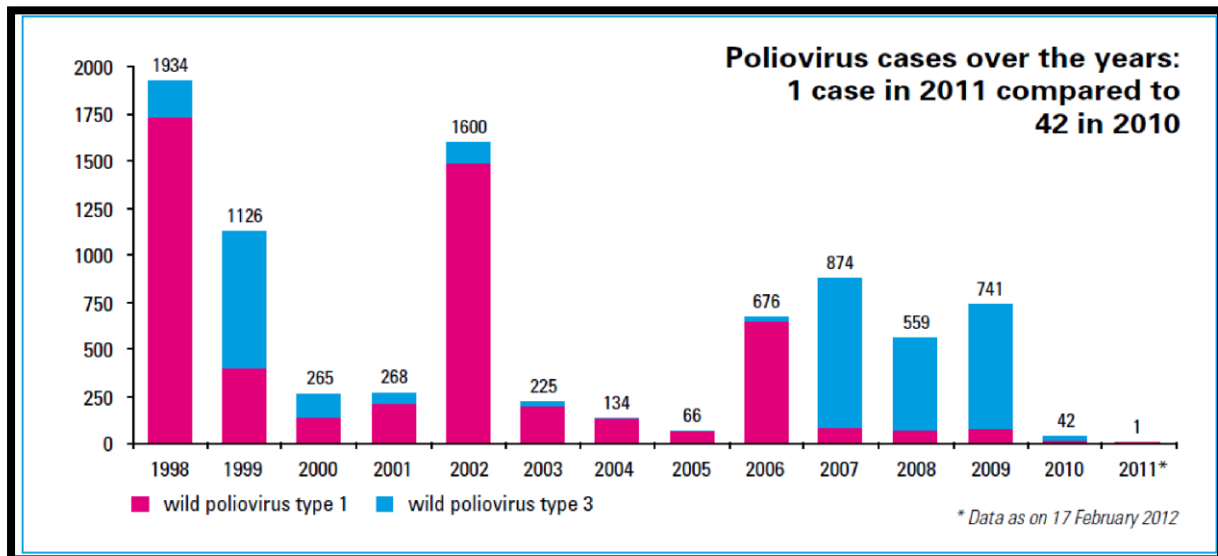
In 1994, the WHO Region of the Americas was certified polio-free, followed by the WHO Western Pacific Region in 2000 and the WHO European Region in June 2002. Of the three types of wild poliovirus (type 1, type 2 and type 3), type 2 wild poliovirus transmission has been successfully stopped (since 1999).

More than 10 million people are today walking, who would otherwise have been paralysed. An estimated more than 1.5 million childhood deaths have been prevented, through the systematic administration of Vitamin A during polio immunization activities.

Source - <http://www.who.int/mediacentre/factsheets/fs114/en/>

INDIA

Before the launch of the Global Polio Eradication Initiative, polio crippled an estimated 200,000 children in India each year. As recently as 2009, India reported almost half the world's cases – 741 out of a total 1604 cases worldwide. Most of the health experts predicted India would be the last country to eradicate polio.



Yet in 2012 India achieved a major milestone. By passing one full year without recording any cases, India is no longer considered a polio endemic country. India overcame huge challenges to stop poliovirus transmission, implementing strategies with constant unrelenting focus and rigour, continually evaluating the programme and introducing innovations to ensure vaccination campaigns reached all children – especially those at highest risk of getting polio. The programme did this by persuading the refusal families. There is no cure for polio, but there are safe, effective vaccines which, given multiple times, protect a child for life. If sufficient numbers are immunized against polio, the virus is unable to find susceptible children to infect, and dies out.

By immunizing the most vulnerable newborns and migrants – whether at brick kilns, in transit on trains or boats, or living in remote regions of the country such as the access-compromised Kosi River flood plains. By promoting strong community ownership to ensure parents continued to immunize their children every time polio drops were offered and by maintaining a rigorously monitored and highly accountable programme. For that they have done some innovations in surveillance and immunizations.

Innovations in Surveillance

- Polio case identification
- Reporting network expansion
- Increase in sensitivity of AFP Surveillance
- Change in laboratory testing methodology
- Supplementary surveillance for polio
- Genetic sequencing of wild poliovirus

Innovations Polio Immunization

- House-to-house vaccination
- Identification of missed children
- Categorization of X houses visited
- Back-up 'B' team
- Integration with NRHM
- Transit sites vaccination strategy
- Congregation site vaccination
- Newborn tracking
- Transit sites vaccination strategy
- Congregation site vaccination
- Newborn tracking
- Kosi River area intensification
- Vaccination of migratory populations
- 107 High-Risk Block Plan
- Special vaccination drive during festivals
- Immunization along the international borders
- Responding to polio as a public health emergency
- Emergency Preparedness and Response Plan

[http://www.unicef.org/india/Polio_Booklet-final_\(22-02-2012\)V3.pdf](http://www.unicef.org/india/Polio_Booklet-final_(22-02-2012)V3.pdf)

Objective

Objective of the study was to find any case of Residual Paralysis in patients of Acute Flaccid Paralysis of children below 15 yrs. of age.

Our Hypothesis was that there was a chance of many cases of Residual Polio Paralysis being present as there was no 60 day follow-up examination of the paralysis cases which is necessary in any Paralysis case of any child below 15 yrs. age. This was either done on purpose or being forgotten accidentally so that India can soon be declared Polio-free.

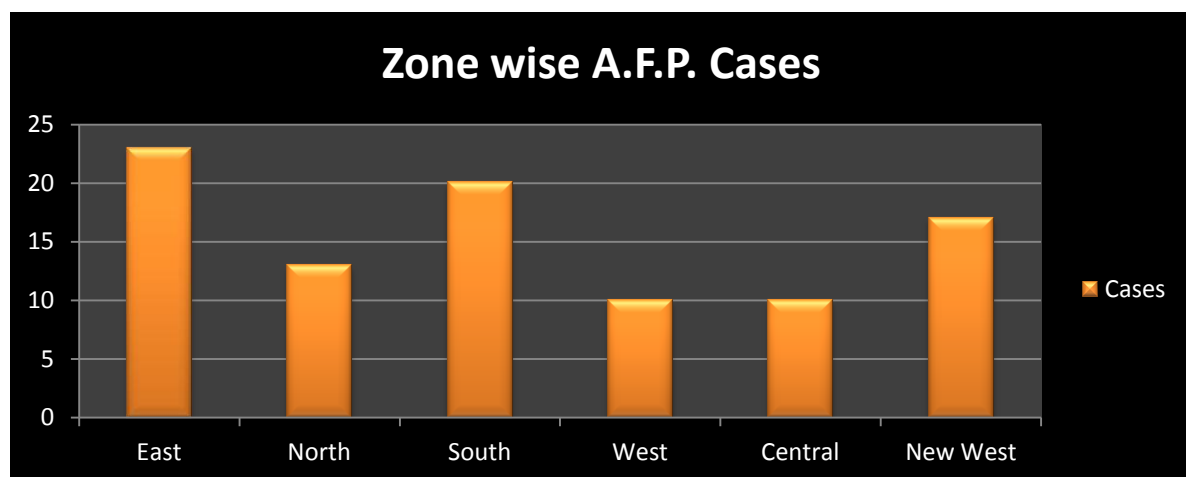
Research Methodology

- **Study Area**-All the five zones of Ahmedabad (East, West, South, Central and New West)
- **Sample Size**- 38 cases.
- **Sampling Technique**- Simple random sampling.
- **Study Design**- Observational
- **Tools Used**- Observation, physical examination, questionnaire, discussions with the patients and their families.
- **Study Duration**- 20 Days

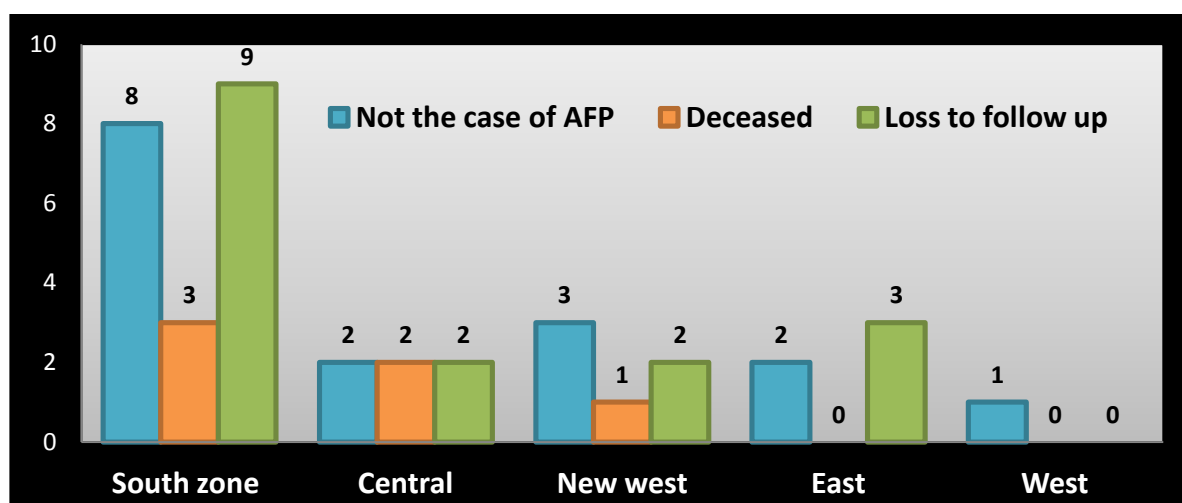
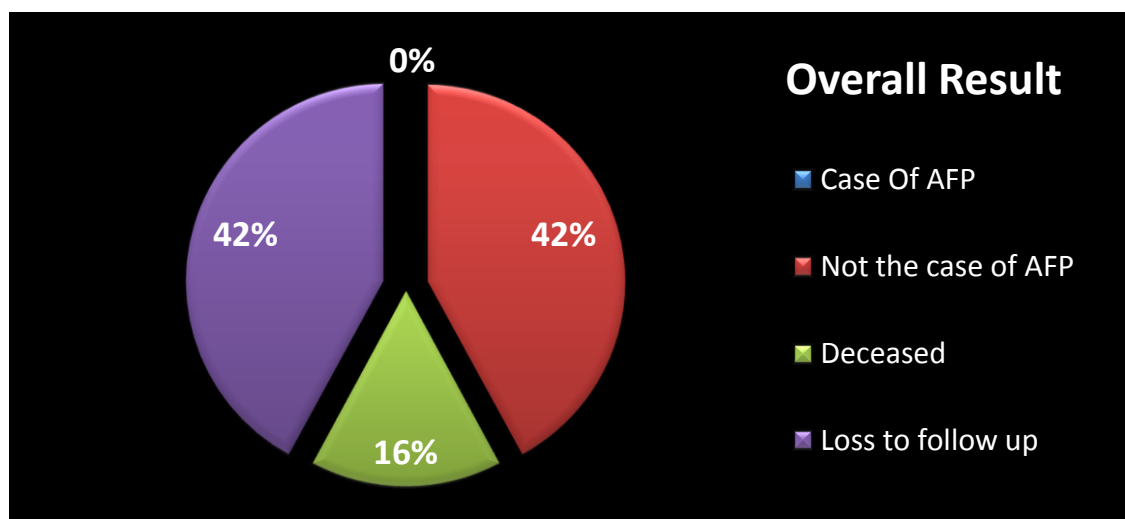
Micro planning

We acquired a list of cases of Acute Flaccid paralysis in children below 15 years of age from Ahmedabad Municipal Corporation for the follow up examination. This A.F.P. case line listing was from January to December 2012. All cases were from 6 Zones. The numbers of patients distributed in all zones are as follows:

23 cases from East zone, 13 cases from North zone, 20 cases from South zone, 10 cases from West zone, 10 cases from Central zone and 17 cases from New West zone.



We have started our study with the aim of covering at least 40% cases from the existing list. We selected South zone first as it required more attention and also contained the second –highest number of cases. Overall, we covered all the 20 cases from South zone, 6 cases from Central zone, 6 cases from New West zone, 1 from West zone and 5 from East zone. The overall findings from study zone wise are mentioned in below table.

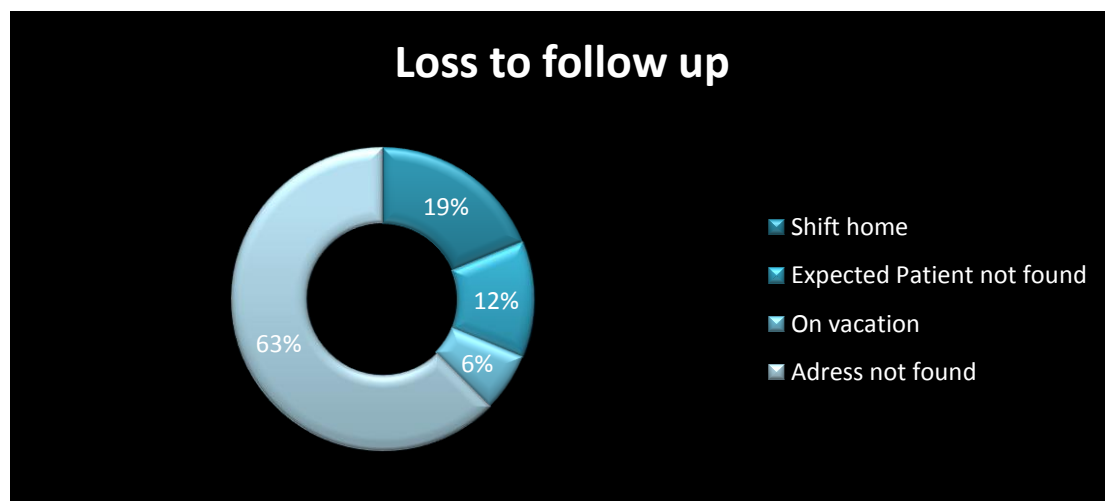


From all zones we found 'no polio virus detected or not the case of AFP' in '16' cases, deceased cases '6', '16' loss to follow up cases and '0' AFP cases. In loss to follow up we have considered the cases, of which we didn't find addresses as they were not incomplete and some have changed their residence.

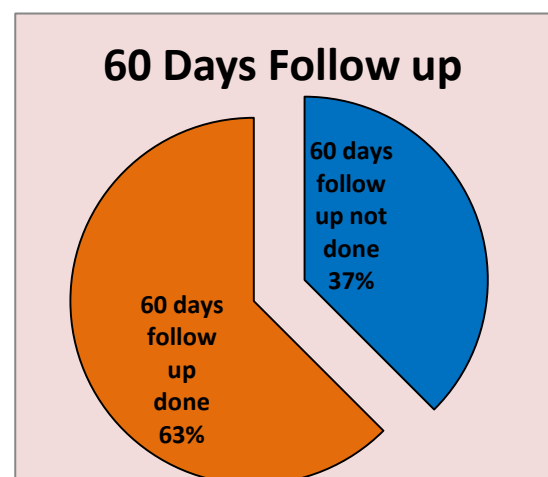
The major negative aspect we faced in this study was that of 'incomplete addresses'. Some addresses even didn't have house number, some didn't have society name and some were not having any nearby significant land mark. We got two cases where we were even able to trace address but couldn't find supposed person.

The second problem which we faced was the timing of the study. Summer vacation period was on, so it was difficult to find many cases at home. Because of these two issues, 42% of our cases visited are 'loss to follow up' cases.

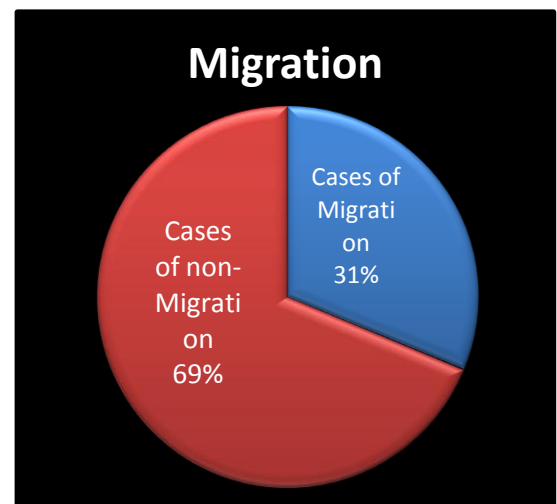
Locate residence of the child and inform the reporting SMO if address seems to be incomplete or incorrect are two major procedures as per the Actions by District Of Residence/ infection (where case has been cross notified) in Cross notification and tracking of cases mentioned in Red book. Here we found 63% incomplete or incorrect addresses which were not traceable. 19% had changed their residence, in 12% of cases-expected person/patient not found and we weren't able to visit 6% of cases as they had gone out on Vacation trips with their families.



Migration is also a concern as we got 5 cases of migration which is practically 31% and 11 cases of non-migration which is nearby 69% of the study sample.



The excretion of poliovirus diminishes rapidly after 14 days, but because a small proportion of cases can still excrete virus for several weeks following paralysis onset, stool specimens should be collected from late-reported cases for up to 60 days (2 months) after paralysis onset. DIO/SMO should ensure that all reporting sites initiate stool collection for AFP CASE



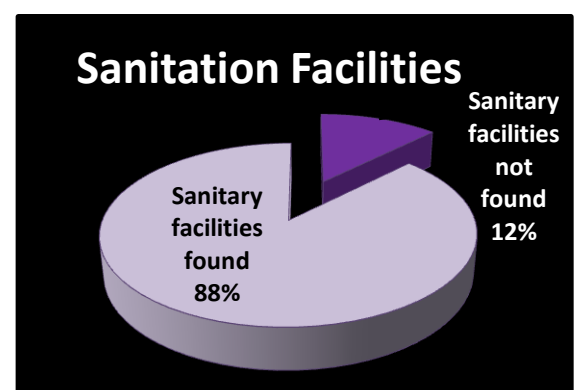
Every AFP case without waiting for the case to be examined by DIO/SMO. In 60 Days follow up we found that there is no 60 days follow up in 37% of cases and in 63% of cases the follow up data was reported.



After 60 days follow up on paper, communication of the result of the examination is essential. In this section we found that in 12-13% of cases no result were communicated after 60 days follow up. But 87-88% of cases

were communicated the result after follow up.

2 cases were not having sanitation facility from 16 cases. As Polio virus is transmitted by feco-oral route, we always have to check for availability of proper sanitation facilities. In our study, 12 % of cases did not have proper sanitation facilities in their homes.



Overview and Results-

From this study of a total of 38 cases out of 93 total cases of Acute Flaccid Paralysis, we can make the following conclusions-

- 1) From all zones we found 'no polio virus detected or not the case of AFP' in '16' cases, deceased cases '6', '16' loss to follow up cases and '0' AFP cases. In loss to follow up we have considered the cases, of which we didn't find addresses as they were not incomplete and some have changed their residence.
Thus, no cases of Acute Flaccid Paralysis were found in our study which could relate to our hypothesis.
- 2) 5 cases of migration which is practically 31% and 11 cases of non-migration which is nearby 69% of the study sample. This shows that many of the cases of Acute Flaccid Paralysis are actually those of migrants, people who migrate from different parts of India, which may also lead us to think that this type of cases occur in the migrant population living in slum conditions where hygiene and sanitation are not as good as necessary, or in some cases, absent.
- 3) In 60 Days follow up we found that there is no 60 days follow up in 37% of cases and in 63% of cases the follow up data was reported. 60 day follow up examination is mandatory in all cases of paralysis/ Acute Flaccid Paralysis
- 4) Communication of the result of 60 day follow up examination is also important for the patients as well as their families because any family in which a child has been struck by Acute Flaccid Paralysis is very tensed and there are many other factors also which is a cause of stress to the family. In our study, we found that in 12-13% of cases no results were communicated after 60 days follow up. But 87-88% of cases were communicated the result after follow up.
- 5) 2 cases were not having sanitation facility from 16 cases. As Polio virus is transmitted by feco-oral route, we always have to check for availability of proper sanitation facilities. In our study, 12 % of cases did not have proper sanitation facilities in their homes. Sanitation is very important for the prevention of spread of the virus.

Questionnaire

For Research

Purpose only

Acute Flaccid Paralysis

Case Investigation Form

1. Case Identification:
Patient's Name: _____
Date of birth: ____/____/____ Age (at onset): Years____months____
Father's Name: _____
Father's Occupation: _____
Mother's Name: _____
Grand father's Name: _____
Religion: Muslim / Hindu / Other Caste: _____
Address: _____
Landmark: _____
Village / Mohalla: _____ HRA: Y / N
Block /Urban area: _____ District: _____
State: _____
Tel. _____
Child belongs to migratory family/Community : Yes/ No/ Unknown
If yes, specify: Slum with migration/ Nomad/ Brick Kiln/ Construction site/ Others (specify): _____

2.Hospitalization: Yes / No	Date of Hospitalization:____/____/____
Name of Hospital: _____	
Diagnosis as per hospital records, if any: _____	

3.Immunization History:
a. OPV doses received through routine EPI (before onset): _____
b. OPV doses received through SIAs (before onset): _____
c. Total OPV doses (a+b): _____

4. Clinical Symptoms:	Date of Paralysis
Onset:____/____/____	
Number of days from onset to maximum paralysis:_____	
Acute paralysis: Yes / No / Unknown	
Flaccid paralysis (anytime during course of illness)Yes/ No/Unknown	

Any Injections during 30 days before paralysis onset: Yes / No / Unknown

If Yes, side and site of injection _____

Fever on day of paralysis onset: Yes / No / Unknown

Ascending paralysis: Yes / No / Unknown

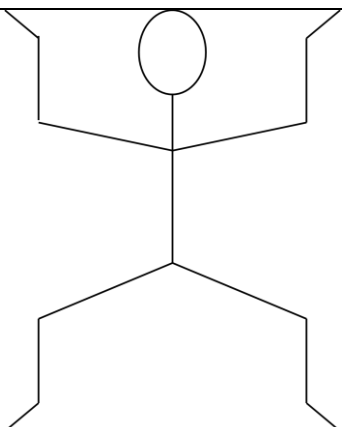
Descending paralysis: Yes / No / Unknown

5. Clinical History:

Respiratory involvement: Yes/ No Bulbar involvement: Yes/ No Bladder/bowel involvement: Yes/ No Joint pain/Swelling: Yes/ No Gait:	(write evolution and progression of illness)
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6. History of contacts with healthcare providers after the date of paralysis onset (including the notifying health facility):

Name & address of Hospital/ doctor/ quack:	1.	2.	3.	4.

7. Clinical examination:	Investigation Date: _____ Examined by: _____	
Tone: (normal/↓ /↑)	UL: Right: Left:	LL: Right: Left:
Power: (Grade 0 to 5)		
0 - No Contraction		
1 – Flicker of contraction		
2– Active movement with gravity		
3 – Active Movement against gravity but no resistance		
4 - Active Movement against resistance		
5 – Normal		
Reflexes:	N/↓ /↑ / absent/ uncooperative child	N/↓ /↑ / absent/ uncooperative child
Biceps:	Right	Left
Triceps:	Right	Left
Supinator:	Right	Left

Knee jerk:	Right	Left
Ankle jerk:	Right	Left
Plantar:	Right	Left
Circumference: Mid-arm:	Right: flexor / extensor/ Uncooperative child	Left: flexor / extensor/ Uncooperative child
Fore-arm:	Right	Left
Mid-thigh	Right	Left
Mid-calf:	Right	Left
Cranial nerves affected	Right	Left
Sensation loss: Yes / No / Unknown Asymmetrical paralysis: Yes / No / Unknown		
Site(s) of Paralysis: right arm / left arm / right leg / left leg / neck / bulbar / respiratory muscle / trunk / facial/ other_____		

8. Provisional diagnosis:
Guillain-Barre Syndrome / Transverse Myelitis / Traumatic Neuritis / Transient Paralysis / Facial Palsy / other / Unknown

9. Stool Specimen Collection:
Number of samples collected_____
Was the result communicated? Yes/No/Don't know
If Yes, What was the result?_____
If No, Why not?_____

10. 60 day follow up examination: Yes/No
If Yes, Who did it? Till how much time weakness was felt? RA: _____ RL: _____ LA: _____ LL: _____ NECK: _____ BULBAR: _____ RESPIRATORY MUSCLES: _____ TRUNK: _____ FACIAL: _____

11. Water source: Well/Bore well/Hand pump/Tank/Stream/Tap water/any other
Drinking water: Boiling/Filtering/RO/Any other

12. Using sanitation facility: Yes/No

If Yes: service type/P.R.A.I./R.C.A./Septic tank/Others

Toddler's defecation: courtyard/ indiscriminate/ lanes in front/ others.

Mode of disposal: Indiscriminate/others

13. Patient's Return to normalcy

- Able to perform his routine?
- What about studies?
- What about physical activities and sports?
- Attentive?
- Stress on family
- Expenses