



Susan Eik Filstead Stroke & Epilepsy Foundation, Inc.

A cure is at the ♥ of our mission

To Those Seeking a Cure,

I am pleased to enclose a conference report entitled: SEEKING ANSWERS: THE FIND A CURE FOR EPILEPSY CONFERENCE.

The ideas, issues, and suggestions raised by this prominent group of neuroscientists should serve as a roadmap for devising a multifaceted research agenda that can ultimately result in a cure for epilepsy.

A collaborative, integrated effort across disciplines and institutions that stresses both short-term and long-range results is an essential requirement for success.

It is my hope that this report can be a source of hope to patients and families who confront epilepsy on a daily basis and also a challenge to our government and private philanthropy to provide the financial resources necessary to make the goal of curing epilepsy a reality.

Your [contribution](#) to support the research activities funded by the foundation is greatly appreciated.

Sincerely,

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SEEKING ANSWERS:
THE FIND A CURE FOR EPILEPSY CONFERENCE
WILLIAM J. FILSTEAD Ph.D

**SEEKING ANSWERS:
THE FIND A CURE FOR EPILEPSY CONFERENCE
MAY 18 – 20, 2003
LAKE BLUFF, ILLINOIS
WILLIAM J. FILSTEAD Ph.D**

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The idea for the Find A Cure for Epilepsy conference emerged from the simple fact that, depending on the reference (Chang and Lowenstein, 2003; Kotagal and Lüders, 1999; Hauser and Hesdoffer, 1990; and Porter and Chadwick, 1997) anywhere from 30% to 60% of those who have epilepsy do not have any relief from it. That is, the epilepsy is intractable; not effectively treated.

Patients in whom epilepsy has been “successfully” treated either through surgery and/or anti-epileptic drugs (AEDs) typically experience significant side effects from these treatments. Fatigue, drowsiness, confusion, cognitive impairments, etc. are commonly occurring side effects of AEDs.

Thus, intractable epilepsy and significant cognitive/quality of life side effects from AEDs, leaves one option available – finding a cure. Anything less is unacceptable.

In the late summer-early fall of 2002 an email was sent to approximately 35 individuals in various academic and governmental organizations, all recognized names in the field of epilepsy and/or prominent neuroscientists. The email stated: “If a conference was to be held that had as its focus finding a cure for epilepsy who do you think should be invited – yourself included?”

This email generated a list of names and rationales for why the suggested individual(s) would be a valued participant. We also expressed the point that scientists who did not focus their work on epilepsy, per se, but had a significant point of view that would enhance our discussions during the conference were also welcomed.

A total of 58 suggestions were made spanning 32 individuals. From this list the invited participants were selected (see table 1).

The conference was convened for 2 days (May 18-20, 2003) at a meeting facility in the Chicagoland area. All invited participants knew who were attending and an information booklet containing biographical contact information along with an article that best represented the participant’s work were distributed to all attendees prior to the meeting.

The format of the meeting employed the Nominal Group Technique (NGT), which is a structured process that streamlines the presentation and discussion of ideas (Delbecq, VandeVen, and Gustafson, 1975; Delbecq and VandeVen, 1971). A focal question is asked: What do we need to know in order to cure epilepsy? In round-robin fashion each participant provides a response without any elaboration or general discussion. The goal is to obtain very specific and discrete responses (answers) to the question that is being asked.

Once all the responses have been given (each is written on a large piece of paper and taped on the meeting room walls) a period of discussion, clarification, elaboration, even merging of items occurs. This distilling state is aimed at developing the “cleanest and clearest” list of responses (answers) to the focal question.

The final step in the NGT is the voting, ranking and prioritizing of responses. There are numerous ways the voting can occur. In fact, there are typically multiple votes, which provide a sense of the varying importance specific responses have. For example, voting instructions such as “pick the number one area where we should begin “vs.” select the top 5 issues and rank order your responses”, yield very different sets of rankings. Each vote provides a different view on the relative value/importance of these responses. Such votes provide a framework for judging the value of these responses.

Ultimately a vote is taken and a sense of the relative importance of each response is identified.

Having arrived at some sense of the importance/value that each item has, the group is asked to focus on the top three items and brainstorm what types of actions can be taken to accomplish the intent of the responses.

Thus, the question yields responses that become ranked and yield their own suggested steps for achieving/accomplishing the intent of the item.

What follows is a presentation of the responses to the question: What do we need to know in order to cure epilepsy?

RESPONSES

As a point of clarification the issue of what is meant by “cure epilepsy” was raised. Is it aimed at preventing epilepsy (epileptogenesis) in those at risk? Or does it refer to ending the symptoms/seizures of those who presently have epilepsy? Based on the discussion it was decided that either definition of cure could be used in arriving at an answer to the focal question.

The group stressed the importance of the hyper-excitability of the brain as the most common indication of epilepsy. At the same time it was noted that there are numerous types of epilepsies from multiple causes (some known and others unknown), but that the hyper-excitability and/or seizures per se could be seen as common threads across the many epilepsies.

The participants provided a total of thirty-eight (38) responses. The specific responses are listed in table 2.

The nature of the discussion, the exchange of viewpoints, and the issues raised in the debate among the participants can provide a context within which to view the 38 responses.

1. Risk: What factors, genetic and/or environmental, place people at risk for developing epilepsy? What are the critical changes in the brain that result in a seizure? Are we talking about a circuit problem (molecular level) or is it a synaptic problem (strength of the connection)? Why do seizures start – and once started, why do seizures stop?

Discussion centered on the role of genetics vis a vis epilepsy. Understanding the various ion channels and how drug compounds impact the activity of these channels is extremely important.

Equally important is the need to understand brain pathology, especially a brain with epilepsy vs. a normal brain and/or a brain with some other condition. We know that epilepsy can occur spontaneously and that spontaneous remission occurs. Why is that? Such understanding might identify new targets for intervention.

2. Models: There are numerous animal models that need to be evaluated vis a vis various drug and/or other intervention strategies. Most importantly, these animal models have to demonstrate how they do or could translate to humans. This link in the research between animal and human studies is critical. Unless this connection between these research platforms can be established, the relative value and utility of animal models will be suspect.
3. Funding/Collaboration: The universal call for more research funding, both private and federal was made. Data does support the under funding of epilepsy research vis a vis

other neurological conditions, ex. Parkinson Disease. That is, a media persona like Michael J. Fox has not emerged to serve as a highly visible advocate for the cause of epilepsy funding.

Also, within the federal structure, epilepsy is under funded. NINDS and other federal agencies within NIH do not seem to request and appropriate a level of funding consistent with the broad impact and consequences of epilepsy. For example, the National Institute on Aging and The National Institute on Child Health and Human Development do not seem to support epilepsy research commensurate with the impact epilepsy has on older adults and children.

The extent to which the respective epilepsy organizations can coordinate or orchestrate a unified message was discussed. A public awareness campaign/message, which addresses the misperceptions society has about epilepsy and the different types of epilepsies are key themes to stress. The central component of the message is the need for a cure, or at worse, treatment that results in “no seizures/no side effect”. An aggressive public awareness campaign was suggested.

It is estimated the 2.5 million individuals in the USA have epilepsy (some think this number is significantly understated). The proportions of individuals with intractable epilepsy and/or significant cognitive side effects from AEDs have been estimated to be as high as 80% of this population.

Continuing to have seizures is like continuing to have an irregular heart rate. We know an irregular heart rate is not acceptable. Continuing to have seizures is unacceptable to the individuals with epilepsy and their families. These points can be made in this public awareness campaign.

The concerns over funding also apply to the private sector. The matter of investigator collaboration, within a university and across disciplines, as well as across institutions, is a major challenge to the way the business of research is presently conducted. The matter of overhead, academic promotion, stature within one’s profession, etc., all currently work against the collaborative research model.

Presently, the grant structure fosters competition rather than collaboration and cooperation. Politics and money prevent meaningful changes in the research process from occurring; however, if the monies are re-arranged so as to reward a collaboration model, then a major paradigm shift in how research is conducted has a chance. Requiring multiple institutions and multiple departments/disciplines within institutions to bond together in order to be considered for large scale center grants would have a profound impact on the structure, organization, scope, and ultimately the results of such research.

FOCUSING THE RESPONSES

The identified 38 items in table 2 formed the core of the discussions. Given similarities, overlapping concepts, and the elaborations of ideas that were contained in these items, it was decided to merge items into three distinct domains.

1. Animal Models: The rodent model can be used to identify differentially regulated gene products in epilepsy. One could begin by taking the gene products most likely to be involved in epileptogenesis and test a large battery of compounds against these products. After identifying a few hundred compounds with activity

against these gene products, such compounds could be tested on the rodent epilepsy model in hopes of finding a drug(s) that is (are) effective in treating epilepsy.

Clearly, this approach is complicated since current animal models involve destructive lesions to the brain either by chemical and/or trauma. How adequate this “animal epileptic brain” is in comparison with human epilepsy is unknown. Furthermore, it is unclear how predictive animal models could be of human epilepsy.

A major suggestion was made to convene a meeting of researchers who are identified with an epilepsy animal model – as well as other successful animal model researchers - to compare and contrast their work in the hopes of developing a sense of where the future direction of this animal model research is or should be headed.

A suggestion was made that the mouse models that could make an immediate impact are a multi-drug resistant model, a model that focuses on intractable epilepsy, and a mouse model that looked at the acquisition of epilepsy (i.e. post-trauma, post surgical, etc.).

2. Seizure Prediction: There was extensive discussion on the importance of understanding the “circuitry of a seizure” in real time. What happens pre-ictal, ictal, and post-ictal?

Can the chain of events in this process be identified? Better imaging techniques would be key to this objective. Why do seizures start and why do they stop? What are the similarities and differences across the various epilepsies?

Comparisons could be made across human samples that contain different seizure-epilepsy types. Such a database (containing labs, films, tissues, etc) would allow both longitudinal and comparative studies to be done.

In essence this topic stresses the importance of understanding epileptogenesis.

3. Proteomics: This is an extremely important and evolving field that concentrates on advancing the insights from sequencing of the human genome to the level of protein expression and modification. Genes express themselves through a myriad of proteins that provide their biological and physiological functions. The focus of this field is on understanding the connection between proteins and genes.

Such research may aid in developing molecular biomarkers for epilepsy.

This research could result in designing molecules with specific biological or functional properties for understanding drug transport and developing novel approaches to drug deliveries.

This area of investigation is closely linked with gene research. For example, are there underlying genetic pathways such as common transcriptional events that occur during the development of epilepsy. Some would have us believe that “it’s all in the genes”. How much understanding and cure of epilepsy is genetic is to be determined. But it is an extremely promising area to explore.

GENERAL DISCUSSION: MOVING AHEAD

A wide-ranging discussion occurred at the end of the conference, which focused on: 1) things to do, 2) the next step(s), and 3) remarks in general. A flavor of these discussions follows:

A point was made that seizures appear to be common to most, if not all, epilepsies. Seizures can be characterized as two-dimensional: 1) there can be a focus and 2) there can be a spread. While the focus varies greatly, the spread, one cell to another – one neuron to another, is common to all types of seizures. What factor contributes, causes, and participates in, the propagation of a seizure?

Politically, it is imperative that the nature and level of federal funding for epilepsy be understood. How much is being spent on what projects by which agencies?

Many diseases and conditions cut across NIH Institutes. Funding and collaborative efforts should reflect this collection of individuals with the condition as well as the complexity of the various manifestations of epilepsy. For example, epilepsy has implications for the National Institute on Aging, the National Institute on Child Health and Human Development, and NINDS to name but three of the obvious Institutes.

The focus of funding needs to be the condition(s) and not the vested territorial interest of specific Institutes or Centers.

In the NIH appropriation request before the 106th Congress there was specific language from both the House and the Senate that directed NIH to establish an Interagency Epilepsy Coordinating Committee to coordinate efforts among Institutes other than NINDS. This language also stressed the importance of setting aside funds for epilepsy research from Institute budgets other than NINDS (emphasis in text done by author).

From Senate NIH OD language:

“Epilepsy - The Committee recognizes that while the NINDS is the primary Institute for addressing epilepsy, several other Institutes are also involved in related research. As 75 percent of epilepsy cases begin in childhood, the NICHD has an important role to play in studying this disease. So, too, does the NHGRI, which is urged to assist the NINDS in the search for a genetic fingerprint diagnostic test aimed at improving drug therapy for epilepsy, and the NIMH, which is urged to explore the link between epilepsy and mood disorders, both of which are often treated with anticonvulsant medications. Finally, the NIA is encouraged to examine epilepsy in patients over age 65. The Committee urges the Director to coordinate research efforts among all these Institutes through an Interagency Epilepsy Coordinating Committee that includes agency scientists and industry and patient representatives.”

Furthermore, NINDS was specifically directed by language from both the House and Senate to establish a coordination mechanism for opening communications among the Institute vis a vis Epilepsy.

From Senate NINDS language:

“Epilepsy - The Committee believes that NIH should make finding a cure and effective treatments for epilepsy a priority. The Committee is encouraged by the

establishment of 13 epilepsy research benchmarks resulting from the NINDS March 2000 conference "Curing Epilepsy: Focus on the Future." The Committee encourages NIH to develop a plan to implement the research benchmarks, as the Director deems appropriate, including the funding projections needed to carry out the plan. The Committee directs that the plan be submitted to Congress by April 1, 2002. Further, the Committee encourages the establishment of an Interagency Epilepsy Coordinating Committee comprised of agency scientists, industry, and patient representatives."

From House NINDS language:

"Epilepsy -- The Committee is encouraged by the development of 13 benchmarks for epilepsy research resulting from the Institute sponsored conference held in March 2000 on "Curing Epilepsy: Focus on the Future"... The Committee urges NINDS to enhance research efforts in the prevention, treatment and eventual cure of this disease through all available mechanisms, as appropriate, including the development of a plan to implement the research benchmarks and establishment of an Interagency Epilepsy Coordinating Committee. The Committee also urges the Institute to enhance efforts to address research issues related to the impact of seizures on young children, women, the elderly and those with intractable or uncontrolled epilepsy. NINDS is also encouraged to develop research plans and goals for the anti-epileptic drug development program. The Director should be prepared to testify on its efforts to advance these areas of research at the fiscal year 2003 appropriations hearing."

This renewed focus on epilepsy was included within Institute directives:

From Senate NHGRI language:

"Epilepsy - The Committee encourages the Institute to intensify its efforts to identify epilepsy genes for the more than 40 different types of epilepsy, and to assist the NINDS in the search for a genetic fingerprint diagnostic test aimed at improving drug therapy for epilepsy. The Committee suggests that the Institute coordinate efforts with the NINDS to create a national consortium to identify new epilepsy susceptibility genes through a large-scale genotype:phenotype screen. The Committee urges the Institute to make research in epilepsy a priority and to coordinate research efforts with other Institutes through the Interagency Epilepsy Coordinating Committee comprised of agency scientists and industry and patient representatives."

Clearly, expanding the research funds available for epilepsy by including other relevant Institutes (besides NINDS) is an excellent idea, but the funding of such an undertaking needs to be monitored. Each of the aforementioned Institutes (NIMH, NIA, NHGRI, etc) should set aside a percentage of their budget in line with the scope and impact of epilepsy.

Such an allocation of resources beyond those of NINDS would significantly increase the "pot of money" available to focus on epilepsy.

To date, neither the proposed committees nor any earmarked epilepsy funds outside of NINDS has occurred. Furthermore, why is it difficult to obtain an actual dollar amount that relates to specific problems? For popular issues such as AIDS the dollar figure is readily known – what about other less public illnesses such as epilepsy.

It is vitally important to know what funds are being spent on what specific research activities in order to have a clear sense of the resources behind what specific research tracks.

At the university level issues of overhead and tenure act as barriers to large-scale cooperative consortiums. Basic science needs to be seen in the same light as multi-center clinical trials. Basic science, which is the underpinning of much that was discussed, should have access to these multi-site collaborative research center grants.

The public does not seem to know epilepsy . There is a no “face” that can be attached to this disease like Michael J. Fox can be linked to Parkinson Disease or Christopher Reeve to spinal cord injuries. Epilepsy still is stigmatized which results in misconceptions and misunderstandings as to what it is.

Various efforts to create a gene data bank or a tissue bank focused on epilepsy would enhance research efforts. Blood samples of patients with and without epilepsy could be stored in this data bank of analysis. If susceptibility genes could be identified, then mouse models could be used to test compounds.

How many individuals have epilepsy? What types with what consequences? Perhaps an extensive epidemiological project could begin which aims to provide up to date data on the incident and prevalence of this problem. What is the ratio of children to adults? What proportion of cases have a known vs. unknown etiology? What is the relative effectiveness of which medication(s) for whom? How is the quality of life affected? What can be done to promote and enhance research funding for epilepsy? These are basic fundamental questions that need current information.

No seizures. No side effects. These are the current objectives of many who study, treat, and live with epilepsy. Short term it is vital to understand how seizures occur (predicting seizures onset is a goal) and why a seizure stops.

A longer view focus upon mouse models for testing compounds and understanding the role genes play in epilepsy.

Ultimately the goal is a cure – for those living with epilepsy and intervention strategies to prevent epilepsy from occurring.

This day will come and we trust this conference can speed this process along.

Table 1.
 Conference Attendees

PARTICIPANTS	AFFILIATIONS
<p>Nihal C. deLanerolle D.Phil D.Sc Marc Dichter MD Ph.D Raymond Dingledine Ph.D Jerome Engel Jr. MD Ph.D</p> <p>Gregory Holmes MD Leon D. Iasemidis Ph.D James O. MacNamara MD David McKinnon Ph.D Istvan Mody Ph.D Louis J. Ptacek MD Michael A. Rogawski MD Ph.D Paula Schauwecker Ph.D</p>	<p>Yale University School of Medicine University of Pennsylvania Medical School Emory University School of Medicine Reed Neurological Research Center UCLA School of Medicine Dartmouth Medical School Arizona State University Duke University School of Medicine SUNY Stony Brook UCLA School of Medicine University of Utah School of Medicine Epilepsy Research Section/NINDS Keck School of Medicine at USC</p>
CONFERENCE FACILITATORS	
<p>Jorge J. Asconapé MD William J. Filstead Ph.D</p>	
CONFERENCE RECORDERS	
<p>Amy Filstead MS Riley Snook MD</p>	
GUESTS	
<p>Susan Axelrod Susan Eik Filstead Susan Klingenstein</p>	

Table 2.
Specific Responses to the Focal Question

1.	Surrogate markers of epileptogenesis need to be developed. i. What factors put people at risk for epilepsy?
2.	A better understanding of the natural mechanisms that prevent epileptic changes/epileptiform activity needs to be developed. o An example is <i>epilepsia partialis continua</i> : intractable focal epilepsy, which doesn't spread - why doesn't it spread?
3.	What methods can be developed to aid in predicting epilepsy onset? o An example is using EEG or other methods to predict who will develop epilepsy and possibly initiate treatment before seizures begin.
4.	Bring geneticists and physiologists together to address issues central to epilepsy. o Also try to engage pharmaceutical companies to fund and facilitate drug development
5.	Look for new ways to design drugs and methods to deliver drugs to the brain. o Design drugs specifically for the epileptic brain (not normal brain).
6.	Need to understand the critical elements in brain changes after damage, which lead to epilepsy.
7.	Understand and explore novel targets for epilepsy (i.e. ion channels).
8.	Understand the changes that take place between preictal, interictal, and postictal states. o What are the changes in the state of the brain that occur to produce seizures?
9.	Discover new epilepsy genes. o By discovering epilepsy genes and their products, new treatment approaches can be developed.
10.	Update our understanding of the pathologic changes in epilepsy by applying new techniques (i.e. molecular profiling) in hopes of identifying new targets for therapy.
11.	Improve collaboration between basic science research and clinical trials by the formation of consortiums.
12.	Discover commonalities that underlie genetic pathways such as common transcriptional events that take place during the development of epilepsy.
13.	Validate an animal model of epileptogenesis o Construct mouse models of epilepsy which can be validated and stored in a central location so they can share between researchers.
14.	Attempt to understand the primary dysfunction underlying epilepsy. o Is epilepsy a circuit problem (a change in connections in the brain)? o Is epilepsy a synaptic problem (a change in strength of connections in the brain)? o Is epilepsy an intrinsic neuronal problem (a structural problem with the neurons)?
15.	Define a molecular basis for epilepsy.
16.	Understand the mechanism for spontaneous remission in epilepsy.

	<ul style="list-style-type: none"> ○ Develop experimental models (i.e. mouse models) to understand why epilepsy spontaneously remits in some patients.
17.	<p>Understand what changes take place in the brain when an epileptic seizure stops.</p> <ul style="list-style-type: none"> ○ Develop a mouse model to understand the mechanism(s) that "turn off" a seizure.
18.	<p>Understand the differences in mouse genetics that produce different phenotypes in the animals despite being bred from the same genetic strain.</p> <ul style="list-style-type: none"> ○ Why do pure-strain inbred mice express differences in respect of seizure parameters (threshold, remissions, etc.)? ○ Extrapolate differences in mice genotype/phenotype expression to human research.
19.	<p>Develop better methods for identifying localizing seizure foci in epileptic patients.</p> <ul style="list-style-type: none"> ○ Improvements in non-invasive and invasive localization methods could lead to more accurate (less morbid) surgical treatments.
20.	<p>Understand the limitations of using animal models of epilepsy to predict human epilepsy.</p> <ul style="list-style-type: none"> ○ Discover what parameters of animal epilepsy models can be applied to human epilepsies, and conversely, what factors are not applicable.
21.	<p>Improve resolution of imaging and recording techniques in epilepsy.</p>
22.	<p>Understand the "direction of information flow" in epilepsy.</p> <ul style="list-style-type: none"> ○ Solidify our understanding of the sequence and processes involved in seizures down to the molecular level.
23.	<p>Develop a 3-dimensional "whole brain" understanding of brain structure and function vis a vis a seizure.</p> <ul style="list-style-type: none"> ○ Explore the concept of "networks" of signaling and structural interaction.
24.	<p>Develop animal models of drug-resistant epilepsy.</p>
25.	<p>Develop a better classification system for epileptic seizures and syndromes.</p>
26.	<p>Understand how a seizure may impact the development of future seizures.</p> <ul style="list-style-type: none"> ○ Discover the mechanism by which "seizures beget seizures".
27.	<p>Develop genetic animal models that can be used to produce pharmacogenetic profiles.</p> <ul style="list-style-type: none"> ○ If several animal models can be developed and validated, they can be tested against current and investigational compounds. ○ This method could be used to develop an understanding of the best treatment for different types of epilepsy and lead to the discovery of new drug treatments.
28.	<p>Form a consortium of non-epilepsy researchers to collaborate on the problem of curing epilepsy.</p> <ul style="list-style-type: none"> ○ Bring together basic-science researchers who deal with ion channels, other physiologic processes to focus on processes involved in epilepsy.
29.	<p>Develop techniques (stem cells, neuronal transplantation, etc.) to replace damaged neurons with healthy neurons.</p>

30.	Work with politicians and lobbyists to decrease the bureaucracy and streamline the access for funding of epilepsy research.
31.	Focus on understanding some of the unique epilepsies of childhood (i.e. Lennox-Gastaut, infantile spasms) and extrapolate this knowledge to adult epilepsies.
32.	Apply the emerging field of proteomics to epilepsy research.
33.	Expand pathologic understanding by application of new techniques (molecular profiling). <ul style="list-style-type: none"> ○ Compare common pathologic substrates between different epilepsies to improve the general understanding of epilepsy.
34.	Explore reallocation of financial resources in drug research for epilepsy and search for ways to decrease pharmicoeconomic barriers to expanding the pool of drugs available to treat epilepsy. <ul style="list-style-type: none"> ○ Improve support for orphan drugs.
35.	Pool resources to try to cure a specific, known type of epilepsy (i.e. Juvenile Myoclonic Epilepsy). <ul style="list-style-type: none"> ○ After curing an epilepsy like JME the knowledge gained can be used to focus resources on other types of epilepsy.
36.	Understand how seizures propagate (what changes take place in the brain just before and just after a seizure starts). <ul style="list-style-type: none"> ○ Use this knowledge to try to prevent seizures before they start.
37.	Focus on improving epilepsy surgery in regards to localization, timing, etc.
38.	Understand the concept of hyper synchrony in seizure propagation.

References

1. Kotagal, P. and Lüders, H.
The Epilepsies: Etiology and Prevention
Academic Press, San Diego, Calif. 1998
2. Hauser, W. and Hesdorffer, D.
Epilepsy: Frequency, Causes and Consequences
Demos Publications, New York 1990
3. Porter, R. and Chadwick, D.
The Epilepsies 2
Butterworth-Heinemann Publishers, Boston, 1997
4. Delbecq, A., VandeVen, A. and Gustafson, D.
Group Techniques for Program Planners
Scott Foresman & Company, Glenview, IL 1975
5. Delbecq, A. and VandeVen, A.
“A Group Process Model for Problem Identification and Program Planning.”
Journal of Applied Behavioral Science, Vol. 7: pp 466-491, 1971
6. Chang, B. and Lowenstein, D. “Epilepsy” NEJM, Vol. 349, #13, pp 1257-1266, 2003